DOWNLOAD PDF EVOLUTION OF APHASIC SYNDROMES ANDREW KERTESZ

Chapter 1 : Adult language disorders (Book,) [blog.quintoapp.com]

Andrew Kertesz has expertise in Linguistics and Psychology. The pattern of evolution of aphasic syndromes and an overall prognostic guide are presented. Show more. Source.

Age of onset was earlier in tau negative cases, but the duration of illness and gender distribution were about the same in all histological variants. Although the tau negative and positive histologies are predicted to some extent by the clinical onset, the extent of the overlap and the convergence of the syndromes in the course of the disease argue in favour of maintaining the clinical and pathological varieties under a single umbrella. An alternative conceptualization considers the pathology related, even though certain distinctions are maintained. Arnold Pick described the clinical features: It was only later that the round, silver staining inclusions were considered characteristic Onari and Spatz, , and even later essential for the eponymic designation of the disease Brun, Subsequently, both the clinical and pathological entities were incorporated into the term frontotemporal dementia FTD Lund and Manchester Groups, Primary progressive aphasia PPA was considered a distinct entity Mesulam, An integrative approach based on clinicopathologic correlation suggests that not only do FTD and PPA overlap clinically and pathologically, but the extrapyramidal component, commonly described as corticobasal degeneration syndrome CBDS should be considered part of the overall entity named Pick complex Kertesz et al. Corticodentatonigral degeneration described by Rebeiz et al. Progressive supranuclear palsy PSP Steele et al. The concept was reinforced by the discovery that mutations on the Tau gene could produce many of the pathological and clinical varieties identified in sporadic cases. Currently, clinicopathologic studies recognize that ubiquitinated, tau and synuclein negative inclusions, or motor neuron disease type inclusions MNDI Jackson et al. All types include lobar atrophy, neuronal loss, gliosis, superficial spongiosis, and often ballooned neurons, and some glial abnormality. A range of histochemical abnormalities is found not only across the clinical phenotypes, but also among families with the same mutation, and even within a brain. Conflicting information about the duration of illness and sex differences between the pathological and clinical varieties have been published and additional demographic data are needed from autopsy specified cohorts. Recently the combined clinicopathologic experience from two centres, Cambridge and Sydney, provided the first large overview of the features of an autopsy-based population Hodges et al. Most patients were followed yearly and substantial clinical, behavioural, cognitive and neuroimaging information has been collected. FTD-bv presented a progressive deterioration of behaviour and personality Neary et al. We also included four patients, who were autopsied before and did not have the clinical diagnosis of FTD-bv, but had enough clinical information to fulfil the criteria. Semantic dementia Snowden et al. In this series this was only seen in patients as a second syndrome after onset with a behavioural disturbance. We do have patients with primary semantic dementia but as yet they have not come to autopsy. PPA Mesulam, was diagnosed as probable when aphasia was the first syndrome. This group included anomic, logopenic and non-fluent patients as described in detail previously Kertesz et al. In these patients the extrapyramidal symptoms developed first and were followed by cognitive change. Cases with cognitive syndrome first, who developed CBDS or PSP later, were included under the primary cognitive onset, and the motor disorder was documented under secondary and tertiary syndromes. PSP showed vertical gaze palsy, falling, axial rigidity and pseudobulbar palsy. Patients were followed annually with neurological, neuropsychological and behavioural testing. Telephone follow-up was obtained when patients were unable to attend the cognitive neurology clinic. Onset was determined by history, most often from informants, and the clinical features confirmed at the first clinic presentation. Onset of second and third syndromes in the interval between clinic visits was also determined by history and confirmed by examination. Occasionally signs such as unilateral rigidity or verbal apraxia would highlight a new syndrome, but with questioning symptoms were also usually apparent and served as the marker for duration. The time between the first and second and third syndromes is indicated in years mean and standard deviation in Figs 3 and 4. The diagnosis was based on

history and neurological examination primarily, with behavioural inventory, language and other cognitive tests and neuroimaging as supportive evidence. All caregivers were approached for provisional autopsy consent either at the first or on subsequent visits. Some patients had yearly neuroimaging with formal neuropsychological assessment. Frontotemporal atrophy, often asymmetrical, supported the diagnosis, but patients were not selected on the basis of neuroimaging. Neuropathology Neuropathological examination was carried out by one of the authors D. Histological methods included Bielschowsky and Gallyas silver stains, and immunostains for tau, Tau-2, Sigma-Aldrich, AT8, Innogenetics alpha-synuclein gift of Dr Masliah and ubiquitin U, Sigma with a few exceptions as indicated and prion protein immunostains where appropriate. Criteria for histological classification of FTD have been detailed elsewhere Munoz, ; Munoz et al. CBD was characterized by ballooned achromatic neurons Pick cells , tau immunoreactive, intracytoplasmic neuronal inclusions of variable morphology, stained by both Bielschowsky and Gallyas, pervasive grey and white matter threads, and abundant tau positive glial pathology, both coils in oligodendrocytes, and glial plaques in astrocytes. PSP differed from CBD by the predominantly subcortical distribution of the neuronal pathology, and the presence of tufted astrocytes instead of glial plaques. DLDH was diagnosed when only superficial linear spongiosis, neuronal loss and gliosis could be identified. Three cases in which ubiquitin immunostains could not be obtained because the blocks were not available to us were included by default in this group. Statistical methods Demographic information that included age of onset, duration and education were analysed among the groups using one-way ANOVA. The log rank test was used to analyse for differences in survival. Results Pathology A total of 60 autopsied patients with the clinical diagnosis of Pick complex as defined in the methods were included. So far no linkage or tau mutation has been found in that family. The remaining five patients form a heterogeneous group. Finally, a patient with florid behavioural symptoms died a few days after an accident sustained while riding a motorcycle without a license. The autopsy showed numerous contusions, haematomata, and axonal swellings, but a normal brain weight and neither evidence of cortical atrophy nor evidence of deposition of tau or ubiquitin. The pathological varieties, their demographic details as well as the number of clinical syndromes and the intervals between them are summarized in Table 1. The prognosis of the tau positive, tau negative and other pathology groups was the same according to the survival curves Fig. The log rank test found no significant difference between the pathology groups. Table 1 Subject demographics, syndrome frequencies and intervals according to pathology subtypes.

Chapter 2 : The evolution and pathology of frontotemporal dementia | David MuÃ±oz - blog.quintoapp.com

The syndromes of aphasia: similarities and differences in neurolinguistic features. Goodglass, Harold Goodglass, Harold Less.

Age of onset was earlier in tau negative cases, but the duration of illness and gender distribution were about the same in all histological variants. Although the tau negative and positive histologies are predicted to some extent by the clinical onset, the extent of the overlap and the convergence of the syndromes in the course of the disease argue in favour of maintaining the clinical and pathological varieties under a single umbrella. Revised June 16, Accepted June 17, An alternative conceptualization considers Brun, Arnold Pick described the clinical fea- degeneration of the non-Alzheimer type Gustafson, ; tures: It was of the frontal lobe type Neary et al. Subsequently, The Author For Permissions, please email: Most patients were followed Manchester Groups, Primary progressive aphasia yearly and substantial clinical, behavioural, cognitive and PPA was considered a distinct entity Mesulam, An neuroimaging information has been collected. Corticodentatonigral degeneration described by Rebeiz et al. Furthermore, cases presenting with progres- cognitive neurology clinic or in the University of Western Ontario sive aphasia and the behavioural variant of FTD FTD-bv dementia study in London, Ontario, during the years â€" and met the clinical criteria described below and came to autopsy. Progress- ive supranuclear palsy PSP Steele et al. The concept was reinforced by the discovery that study diagnosed as atypical dementia, but both had documented mutations on the Tau gene could produce many of the patho- onset with behavioural symptoms, followed by aphasia. Semantic dementia Snowden et al. We do have patients with primary semantic dementia but as yet they have not come to autopsy. Munoz , Munoz et al. This group included anomic, logopenic and applied when tau or ubiquitin positive inclusions are lacking non-fluent patients as described in detail previously Kertesz et al. All types include lobar atrophy, neur- We use Jackson et al. A range of histochemical abnormalities is syndrome. In these patients among families with the same mutation, and even within a the extrapyramidal symptoms developed first and were followed brain. Conflicting information about the duration of illness by cognitive change. Cases with cognitive syndrome first, who and sex differences between the pathological and clinical vari- developed CBDS or PSP later, were included under the primary eties have been published and additional demographic data cognitive onset, and the motor disorder was documented under are needed from autopsy specified cohorts. Recently the secondary and tertiary syndromes. Since the clinical and pathological overlap with the features of an autopsy-based population Hodges et al. Telephone follow-up was obtained plex patients, who were prospectively followed to autopsy. This when patients were unable to attend the cognitive neurology clinic. Onset was determined by history, most often from informants, and here usually accompanied by ubiquitinated dystrophic neurites. Onset DLDH was diagnosed when only superficial linear spongiosis, of second and third syndromes in the interval between clinic visits neuronal loss and gliosis could be identified. Three cases in which was also determined by history and confirmed by examination. All caregivers were ships between clinical syndromes and pathology. The log rank test approached for provisional autopsy consent either at the first or on was used to analyse for differences in survival. All statistical analyses subsequent visits. Some patients had yearly neuroimaging with formal neuropsychological assessment. Fronto- temporal atrophy, often asymmetrical, supported the diagnosis, Results but patients were not selected on the basis of neuroimaging. Neuro- Pathology psychological testing included screening with the mini mental state A total of 60 autopsied patients with the clinical diagnosis of examination MMSE , Mattis Dementia Rating Scale DRS , clock Pick complex as defined in the methods were included. So far no linkage or tau mutation has been found in Neuropathology that family. Pick body dementia authors D. DLDH was were reviewed. Criteria for histo- nine of which had the clinical diagnosis of possible PPA, logical classification of FTD have been detailed elsewhere Munoz, ; Munoz et al. The remaining five patients ized by round or oval, compact intracytoplasmic neuronal inclusions form a heterogeneous group. One with a combination of stained by

Bielschowsky but not by Gallyas, tau immunoreactive and cortical Lewy body and Alzheimer pathology manifested located in dentate fascia, hippocampus and cerebral cortex. Finally, a patient instead of glial plaques. ITSNU, Munoz, rested on the presence of these structures in The autopsy showed numerous contusions, haematomata, the cytoplasm of neurons in the fascia dentata and cerebral cortex, and axonal swellings, but a normal brain weight and neither The evolution and pathology of frontotemporal dementia Brain , , â€" Table 1 Subject demographics, syndrome frequencies and intervals according to pathology subtypes N Gender Age of onset, mean Years to second Years to third No. F standard syndrome, mean syndrome, mean mean standard standard deviation standard deviation standard deviation deviation deviation Tau negative 24 The pathological varieties, their demographic details as well as the number of clinical syndromes and the intervals between them are summarized in Table 1. For statistical purposes, 0. Tau negative patients were younger Other 0. The prognosis of the tau positive, tau negative and other pathology groups was the same according to the survival curves Fig. PA was tau-negative pathologies. The log rank test found no significant difference between the pathology groups. PSP type pathology was toes, hyperreflexia and dysphagia. Among those of frontal control of swallowing. One of these developed a movement disorder. The six cases of DLDH each presented pathology developed a movement disorder followed by pro- at onset with FTD-bv and only one developed MND as the gressive aphasia while the other four showed no additional Brain , , â€" A. The average intervals and SDs between syndromes are expressed in years. Notably the six patients with these pathologies. Typical An analysis Fig. FTD-bv onset was by far Table 2 Demographics among pathologic groups the commonest in tau negative pathology, while PPA was according to clinical onset more predictive of tau positive pathology, most of which N Gender Age of onset, Duration, in this series were CBD. MND occurred only in the tau neg- M: F mean standard mean standard ative group, while CBDS was infrequent three cases only deviation deviation with this pathology. No significant differences were found in gender distribution, duration or education between basic demographic comparisons between tau positive and the clinical groupings. The number of the tau underlying pathology, but the main division appears to be negative group was inflated by including four members of a along tau negative and tau positive histochemistry, in addi- Mendelian dominant pattern family. As a group the familial tion to the differences in the cortical and subcortical distri- cases were significantly younger than the others mean age at bution of the lesions. Although there is a trend to expand the diagnosis of semantic dementia, our conservative definition includes patients only if their comprehension deficit is prominent and they question the meaning of words heard in conversa- 0. In fact we found three instances when such a FTD movement disorder was associated with tau negative patho- logy, albeit in some only appearing late in the course of their 0. Such cases have been reported sporadically in the literature Grimes et al. There were three cases where even 0. These findings suggest that 0. The Log rank test found no significant differences between the clinical groups. Another smaller patho- logical series from a brain bank have suggested also that the cord of ALS patients Okamoto et al. The high frequency of progressive This prospective study of a clinical cohort, provides aphasia onsets in CBD confirms previous data Kertesz important confirmation of the initial impression of relatively et al. Their most common histopathology both. Similarly pathologists may report transitional positive pathology outnumbered the tau negative cases. The cases with both diagnoses. Our clinical FTD cohort, from which the behavioural onset, which may include features of semantic this study is derived, has about equal numbers of aphasic dementia. We have identified two cases of prominent and behavioural onsets, although our interest in aphasia semantic dementia after the onset of behavioural abnormal- may influence referrals. This includes a significant number ities, and these were included with the FTD-bv onset group. These patients also tend to be older, but this Brain , , â€" A. The nosological position of MRI. These few exceptions rather than of Pick complex. Similar cases were described sporadically indicating heterogeneity of pathology, underline the fallibility Karbe et al. The younger age of onset seems an important consistency and the quality and length of the follow up. There clinical feature especially in FTD-bv and less so in PPA and is an ongoing debate about the definition of both the clinical CBDS who tend to be older when the disease strikes them, and and the pathological

phenotypes. However, we made an especially if they have an underlying tau positive pathology. We feel that the et al. There were no gender study confirms the concept of clinically identifiable syn- or education differences between the tau negative, tau positive dromes that overlap in time to a considerable extent. The and overall groups, but CBD occurred in twice as many males relationship of the pathological varieties remains controver- and DLDH had five males to one female. These differences are sial. Some consider it a heterogeneous collection, others a probably related to the smaller sample size. We did not find related spectrum. Overall, several observations can be made significant gender differences between the clinical syndromes about the underlying pathology according to the onset and either, contrary to other reports in the literature Hodges et al. While tau negative is the most The lack of gender difference in the overall disease common pathology among those with FTD-bv onset, the sub- complex is similar to a large survey study in The Netherlands sequent development of a movement disorder increases the published recently Rosso et al. The duration of illness likelihood of tau positive pathology 3:

Chapter 3 : Neurobiological aspects of recovery from aphasia in stroke.

remission and evolution of aphasic syndromes has contributed to our knowledge of language recovery and the nature of aphasic impairments (e.g. Kertesz and McCabe , Lomas and Kertesz).

Clustering based on the objective and stan- dardized language scores of the WAB yielded significant differences for the acute and chronic groups. The overlap between objective clustering and clini- cal typology, also based on test scores, was sufficient to allow us to interpret the data in clinical terms. Recovering patients are often reclassified as anomics; this changes the anomic clusters, loading one with patients who fall into the recov- ered category. Chronic clusters appeared more distinct with less overlap. The trends in both data sets were investigated by principal components analysis. This showed that all language scores contributed to the first component in both populations fairly evenly. Therefore, the main contribution to the first com- ponent was severity the combination of scores. Comprehension and fluency were the major contributors to the second root in both populations, indicating the diagnostic significance of these parameters. Repeti t ion featured more prom- inently in the second root of the acute, than the chronic population. Introduct ion The classification of aphasic phenomena remains a fundamental problem for the clini- cian and researcher. Intuitive clinical classifications have been in use since Broca and Wernicke described the main varieties of aphasic impairment. None of the classifications Offprint requests to: Phipps has gained universal acceptance although they made a similar distinction between the same types of aphasias. The types often acknowledged are: Global total aphasia with poor fluency and poor comprehension. Anomic amnesic aphasia with word- finding difficulty but good comprehension and repetition. Conduction central apha- sia with poor repetition, many phonemic paraphasias, but relatively fluent speech and good comprehension. Transcortical echolalic aphasias, all with preserved repetition, the motor adynamic variety with poor spontaneous speech but good comprehension, the sensory asemantic variety with good ouput and poor comprehension. A minority of authors place all aphasics along a continuous dimension of unitary impairment, and regard classifications as invalid, even harmful. The majority of clinicians and scholars of language and behavior, however, agree about the need for an accurate and workable classification of common clinical entities. Various theoretical concepts of language impairments have resulted in other labels for these entities. Sometimes, differing populations such as predominantly neurosurgi- cal cases, will produce different entities from cerebrovascular patients. The differences between the methods of examining patients is another source of disagreement. When the sources of variability are recognized and the methods and populations are defined, classifications or taxonomies become more meaningful. In this study, an important source of variability in the taxonomy of aphasias will be examined: Recovery from cerebrovascular accident is the underlying physio- logical and psychological process. We undertook the objective numerical taxonomy of aphasias based on the scores of a comprehensive, clinically relevant, standardized apha- sia test, the Western Aphasia Battery WAB Kertesz and Poole, , utilizing mod- ern clustering algorithms. Numerical taxonomy has been used mainly in the biological sciences Sneath and Sokal, , but has found its way into psychology CatteU and Coulter, Clustering in psycholinguistics Zurif and Caramazza, ; Hecaen and Kremin, ; Ammon and Goedehardt, and aphasiology Kertesz and Phipps, ; Crockett, ; and Kertesz, is promising to provide a new objec- tive method, in addition to traditional classifications. Fluency of spontaneous speech scored 0 - 1 0 according to these criteria: Paraphasias may be prominent. Normal rhythmic patterning may be Taxonomy of Aphasis present within phrases. Marked word finding difficulty. Some word finding difficulty. Information content of replies to standardized conversational questions scored 0 - Comprehension of 20 yes-no questions. Auditory word discrimination pointing to objects, pictures, colours, numbers, let- ters, shapes, body parts, and fingers. Repetition of words, numbers, composite words, sentences of increasing length and complexity, high and low probability. Naming of 30 objects on visual confrontation. Word fluericy naming of animals in one minute. The scores from these parameters form the attributes or characters for the cluster- ing. The use of a clinically valid, comprehensive,

yet practical, easily scorable, and well standardized aphasia test is crucial to this study. The WAB has been extensively stan- dardized, showing high construct validity, internal consitency, test-retest reliability, in- tra- and inter-judge reliability. The rationale and standardization of the test have been described in detail elsewhere Kertesz, Numerical Taxonomic Methods A minimum variance clustering algorithm, called the sum of squares agglomeration on the Euclidean distance matrix of dissimilarities, was used Orloci The results are displayed on dendrograms Fig. The patients who are most similar to each other are adjacent in each group. The abscissa of the drawing is not a parametric axis. The distinc- tiveness of the clusters was measured by determining the intercentroid distances, and the dispersion of each pair of clusters towards each other, which also permitted a nearest neighbor network analysis Fig. Ordination was carried out by principal components analysis Cooley and Lohnes, of the correlation matrix. Both Q and R strategies were employed Orloci, b , the former being used to depict the individuals in the attribute space Fig. The P o p u l a t i o n Two hundred and ninety-two patients suffering from aphasia due to cerebrovascular ac- cident were admit ted to the s tudy from a larger populat ion of more than patients examined for impariment of higher cerebral function. This is a populat ion referred to an aphasia and neuropsychology laboratory in a neurology unit in London, Ontario. It is representative of the acute and chronic aphasic populations of this medium-sized Ca- nadian city. Only right-handed and native En- glish speakers were included. The patients were unselected for type, severity, age, sex, education, or location. The mean ages were: The sex ratio in the acute group was M: Fifty-eight patients were considered to belong to both groups since they had tests in the acute stage of their illness and also one year later. The evolution of their aphasia was tabulated separately. The ordinate represents the level of variance and the individual aphasics are at the terminal branches of the inverted tree. They are distributed along the abscissa mainly according to severity. The order of clinical diagnoses in the groups from left to right roughly corresponds to the following: The clusters were then subjected to at t r ibute analysis and the resulting cluster profiles are displayed in Table 1. Twen- ty-six of these patients are classified as global, and two as isolation aphasics according to our clinical classification, based on the scores shown on a synoptic table Table 2. It is separated from Cluster VII mainly by better repetition. Cluster IX, n -- 19, has better scores in all at tr ibutes than the previous clusters except in word fluency. This group has 17 anomic, 2 transcortical sensory, and 1 conduction aphasic patients. Twenty-eight anomic and 1 conduction aphasic belong to this group, which has the best overall scores and it is the least affected of the acute aphasics. Nearest neighbour network analysis was performed to determine the intercentroid distances and the standard deviation of each cluster. The results for acute aphasics forming 10 groups are displayed on the first linkage diagram Fig. The results indi- cate the closest linkage between groups IX and X which in fact were anomic groups, clini- cally indistinguishable. Although the intercentroid distances are larger, clusters III and IV are less well separated if one observes the overlap between the arrows and smaller numbers representing the standard deviation from the centroid. Linkage diagram of the acute clusters. The clusters are indicated by roman numerals. The large arabic numbers are intercentroid distances, The small numbers with the arrows are standard deviations of the dispersion A. The categories are exclusive. Each individual aphasic is identifiable and coded with the clinical diag- nosis. The diagram illustrates the rate of dispersion and the interlocking of some of the groups. The individuals are distributed in A attribute space, which is multi-dimensional even though the scatter diagram depicts the clusters only in two dimensions. For the interpretation of the horizontal and vertical dispersion, see the discussion. The R-type Principal Components Analysis determines the percentage of total variance contributed by each root and the contribution of each attribute to these roots Table 3. The contributions of the test scores characters to each root are set out in percentages. This determines the extent to which each character language parameter accounts for each root factor in dispersion. The visual display correspond- ing to this factor analysis of roots is the scatter diagram or Q-PCA. Root 1, for instance, accounts for the greatest extent of dispersion, corresponding mainly to the horizontal axis of the Q-PCA. The other roots are responsible for dispersion in other dimensions vertical axis and depth of attribute space. The ordinate represents the level of the variance, and the individual patients are distributed along the abscissa at the

terminal branches of the dendrogram like the acute aphasics. The groups are simi- lar in number but differ in size and dispersion. The clusters were subjected to attribute analysis and cluster profiles similar to those of the acute aphasics were obtained Table 4. Dendrogram clustering of chronic aphasics. Each square represents a patient, The clusters groups are marked by roman numerals. The ordinate is the percentage of variance. In fact, 12 of these patients have recovered according to our criterion Kertesz, and 10 are still classified as anomic. These patients are clinically anomic except two who reached the criterion of recovery. It consists of anomic patients only see discussion for further comments. The nearest neighbour network analysis was based on the intercentroid distances and the dispersion of individuals of each cluster in the direction of the adjacent clusters. The linkage diagram for chronic aphasics Fig. All the groups are distinct, without obvious overlap in dispersion.

Chapter 4 : The numerical taxonomy of acute and chronic aphasic syndromes - [PDF Document]

Kertesz and McCabe () were the first to provide data on the relative incidence of the classical syndromes and patterns of evolution in a large group (N = 93) of patients. Reclassification of type based on Western Aphasia Battery (WAB) test scores was noted in 42% of their sample.

Chapter 5 : evolution and pathology of frontotemporal dementia | Brain | Oxford Academic

The rate, duration and the predictability of natural recovery, the effect of therapy, and major contributory factors are reviewed, with special emphasis on aphasia. The most likely mechanism of second-stage recovery is physiological and functional substitution by connected areas in the brain.

Chapter 6 : Andrew Kertesz, Schizophasia is distinct but not aphasic - PhilPapers

The clinical syndromes of Broca's, global, Wernicke, conduction, anomic and transcortical aphasias are detailed with definition, localization, and prognosis. Modality specific disorders associated with aphasic syndromes are discussed.

Chapter 7 : Biological Perspectives on Language | MIT CogNet

Extrapyramidal Syndromes in Frontotemporal Degeneration Andrew Kertesz & Paul McMonagle & Sarah Jesso aphasic, behavioral or both.