Arnold Pick (1851-1924): a centenary appreciation

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Introduction

2024 marks the centenary of the death of Arnold Pick (1851-1924). Many clinicians are aware of the name and of "Pick's disease" although they may be unclear exactly who he was or what this terminology designates. This article seeks to give some brief biographical details, recap Pick's key findings on "Pick's disease", and relate the latter to current thinking about the classification of frontotemporal dementia, an undertaking which prompts the consideration as to whether the eponym should stand or be laid to rest as now obsolete and superseded.

Biography [1,2]

Arnold Pick (Figure 1) was born in Moravia, a province of the Hapsburg Empire, in 1851. He graduated from the Vienna Medical School in 1875; part of his training was with Theodore Meynert and he overlapped with Carl Wernicke. From the late 1870s he worked mostly in Prague, taking the chair of neuropsychiatry at the German University there in 1886 where he remained chairman until his retirement in 1921.

Pick's many publications covered a wide range of interests, including work on aphasia, wherein he introduced the concept of agrammatism. The influence of John Hughlings Jackson's (1835-1911) work on aphasia may be evidenced by Pick's dedication of his monograph of 1913 on agrammatism, *Die agrammatischen Sprachstörungen; Studien zur psychologischen Grundlegung der Aphasielehre*, to Hughlings Jackson, as "the deepest thinker in neuropathology of the past century" [3]. Kertesz reports that "Pick had Jackson's portrait on his desk" and that "Jackson wrote about Pick and popularised his work in England" (ref. 2, p.19). However, other than his mention of Pick in a footnote of his 1894 paper "The Factors of Insanities" ("I know of but a single study of re-evolution, a very valuable one by Professor Pick, of Prague" [4]), I am not currently aware of any other Jackson reference to Pick (he does not appear in Greenblatt's book on Jackson [5]). Luria [6] credited Pick with recognising the manifestations of afferent apraxia shortly after the original description by Liepmann (1905), citing his *Studien über motorische Aphasie* published in Vienna in 1905.

Pick's publications were by no means limited to behavioural neurology or dementia, nor to the Germanophone literature. He appeared several times in the pages of *Brain* [7-9], including a description of reduplicative paramnesia [8]. Indeed, his final paper, "On the pathology of echographia", appeared in *Brain* in 1924 with the by-line "By the late A. Pick. Professor in the German University, Prague" [9], indicating that he continued to write until shortly before his death.

Fig 1. Arnold Pick

Key papers on focal atrophy

Pick published several papers in the late 19th and early 20th centuries describing clinical deficits in association with focal brain atrophy, papers which have been critically discussed [10-12]. These deficits were either linguistic or behavioural in nature.

The first of these papers, dating to 1892, described a man of 71 ("August H.") with progressive aphasia who at post-mortem was found to have marked atrophy of the cortical gyri of the left temporal lobe [13]. Pick reported further cases of language disturbance in association with either frontotemporal atrophy (1901) [14], or left temporal lobe atrophy (1904) [15]. By contrast, a patient with behavioural disturbance (apathy, disinhibition, personal neglect) in association with bilateral frontal atrophy was reported in 1906 [16].

Pick was primarily interested in clinico-anatomical correlation and did not report microscopic pathological findings in any of these cases. Indeed, it was Alois Alzheimer (1864-1915), not Pick, who in 1911 described the histological findings in such cases (the name "Pick's disease" was not introduced until the 1920s). Alzheimer specifically described the argyrophilic intracytoplasmic inclusions ("Pick bodies") and the diffusely staining ballooned neurones ("Pick cells") which may be associ-

ated with some cases of focal lobar atrophy [17]. (Incidentally, I cannot immediately think of any other instance in which microscopic neuropathological abnormalities have acquired the eponym of someone who had no role in their initial description, but I stand open to correction on this point.) This nomenclature is perhaps all the more surprising in light of the reported rivalry between the laboratories of Alzheimer (in Kraepelin's department) and Pick, which may have been one reason for Kraepelin's promotion of "Alzheimer's disease" as of the 1910 edition (8th) of his textbook of psychiatry [18].

Judgment of posterity?

Perhaps only those dedicated to the study of the dementias in general and of the frontotemporal lobar degenerations in particular will keep abreast of the different classifications which have been proposed for these disorders. Previously lumped together as "Pick's disease", this latter terminology has steadily become more marginalised. If used at all now, "Pick's disease" denotes one subtype of frontotemporal lobar degeneration characterised by the neuropathological finding of Pick bodies and Pick cells. A necessary corollary of this formulation is that "Pick's disease" is not, and cannot be, an exclusively clinical diagnosis.

The heterogeneity of the frontotemporal lobar degenerations defined at clinical, pathological, and genetic levels [19] has been responsible for this marginalisation of Pick. An attempt to encompass all these conditions under the rubric of "Pick complex" [20] (i.e. as interrelated variants on the same spectrum, including frontal lobe dementia with or without motor neurone disease, corticobasal degeneration, and primary progressive aphasia) cannot be said to have prospered in the 25 years since its proposal. Current molecular classification of frontotemporal dementias categorises Pick's disease as 3R FTLD-tau, sometimes with coexistent TDP-43 pathology [21].

Accordingly, the term "Pick's disease" may now be regarded as effectively redundant, in fact obsolete, the moreso if one takes into account the fact that Pick did not describe the characteristic neuropathological findings of "his" disease. If so, it will nevertheless remain the case, as pointed out by John Hodges, that the relegation of Pick to a minor place in the terminology of frontotemporal dementia is sad in light of his "monumental contributions" [11]. In my clinical experience the terminology persisted only in non-specialist medical parlance (e.g. primary care referrals to the memory or cognitive clinic) and in some old age psychiatry clinics (wherein patients labelled as "Pick's disease" may nonetheless have received treatment with cholinesterase inhibitors!).

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