The Lancet Psychiatry
Volume 8, August 2021, Pages 653–4

Catatonia: from pathology to brain imaging

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The separation of catatonia from psychotic and mood disorders in the forthcoming ICD-11 is sometimes viewed as a fair restoration of Ludwig Kahlbaum's original concept after neo-Kraepelinian decades. The recreation of this entity was mainly motivated by its specific responsivity to inhibition reinforcement (eg, diazepam or electroconvulsive therapy), whereas the prognostic argument remains as debatable today as it was during Kahlbaum and Kraepelin's controversy (1). We and others have described strong and specific functional correlates of ICD-catatonic episodes and Leonhard's periodic catatonia phenotype with hyperperfusion in the left premotor (including Broca) and supplementary motor areas (2)(3). These correlates were even reproducible at a single individual level, with good discriminative properties (4). What a surprise to realise that Kahlbaum already reported pathological changes in the same regions: "In younger cases we observed definite hyperplasia, slight swelling, increased blood content, tissue impregnation, in cases of medium duration a more normal condition in these respects (transitional stage), and in the oldest cases a general reduction of the brain tissue and a decrease of blood content... A further characteristic of catatonia is the [brain] location... The propensity of exudate to locate near the sylvian fossa and in the second and third frontal gyri... is most remarkable in the case of language manifestations (mutism and verbigeration)" (translated from German) (5). In 1874, when Kahlbaum published his monography, the left third frontal gyrus had been slowly accepted as the seat of the faculty of articulate language, after Paul Broca's 1860s heated debates with proponents of cerebral holism. Kahlbaum did not address the laterality of pathological findings, although 5 (50%) of 10 were left-sided (none were right-sided). He also reported medial brain anomalies, but their locations are not given with sufficient accuracy to establish whether they overlap with supplementary motor areas. The idea of a motor cortex was supported by findings on dogs by Gustav Fritsch and Edvard Hitzig only 4 years earlier, and its location in humans remained unknown; not to mention the supplementary motor areas, which were not described by Wilder Penfield and Keasley Welch until 8 decades later. Imaging findings are not a replication of Kahlbaum's post-mortem anatomical changes. However, the findings might be consistent with a functional correlate; a hypothesis that could be addressed by examining whether perfusion abnormalities progress with advancing illness, as do anatomical changes. If we add this corroboration of Kahlbaum's observations to the multiple rediscoveries of his muscular signs (eg, parakinesias and spontaneous dyskinesia), the history of psychiatry seems like an eternal beginning.

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