

Home

(Towards education)

October 29, 2015

Thomas A. Ban: Towards education in the history of neuropsychopharmacology.

Part 5. Fundamentals of the Wernicke-Kleist-Leonhard tradition.

### **Thomas A. Ban's reply to Ernst Franzek's comment**

I share your belief that only “close interaction between neuroscience and clinical psychiatry” and “not discussion about disease entities” can revive the major impact the introduction of psychotropic drugs had on psychiatry in the 1950s. It is pointless to quibble about disease entities in relationship to mental illness, as in psychiatry “diseases” are “open” diagnostic concepts, derived by different nosologic organizing principles for which closure will only be attained when fundamental issues of their etiology and pathogenesis are resolved (Ban 2013; Jablensky 2007; Meehl 1985).

I appreciate your concern that it should not be dismissed that Carl Wernicke (1900 a, b) conceptualized mental illness as *disturbance of brain function irrespective of its cause*, whereas Emil Kraepelin (1913, 1915) conceptualized mental illness as *a manifestation of endogenous toxic agents*. I can see that this difference in conceptualization led to debates in the early years of the 20<sup>th</sup> century. I can also see that Wernicke with his research contributions and with his conceptualization of mental pathology was instrumental for setting the foundation of a psychiatry that strives to become an integral part of the neurosciences. Yet, in a historical perspective the essential difference between Kraepelin's and Wernicke's tradition in psychiatry is that Kraepelin (1899) diagnosed patients with endogenous psychoses primarily on the basis of the “course” and “outcome” of their clinical manifestations and classified them with consideration of Thomas Sydenham's (1741) “disease model”, whereas Wernicke (1899) diagnosed patients on the basis

of their “elementary symptoms” (Ban 2015a; Krahl 2000; Wernicke 1893; Wernicke 1895a,b) and classified them with consideration of Wilhelm Griesinger’s (1843) “psychic reflex” model. The lack of recognition that these two approaches are complementing each other has had a negative impact on the development of psychiatry.

I think you are correct that it is virtually forgotten that Karl Kleist (1925, 1934) attributed different clinical pictures in psychiatry to “definite functional centers and regulatory loops” in the brain (Teichmann 1990). This might still be the most interesting way to look at psychiatric illnesses with respect to the results of modern neuroscience, as you suggest and some believe (Cuthbert and Insel 2019; Insel et al 2010). Yet, Kleist’s (1934) important findings in brain research found their place in neurology and they were. his contributions to the foundation of the “bipolar-unipolar dichotomy” (Kleist 1911, 1928; Neele 1949), that stimulated research in psychiatry. His diagnostic concept of “cycloid psychoses” (Kleist 1925, 1928) was also further elaborated (Leonhard 1957).

In so far as I know, we do not have conclusive evidence so far that would support your contention that Leonhard’s (1999) notion (“theoretical background”) that “each clinical picture has its own genetic background” is wrong (Rutten and Mill 2009). It might be correct to say that some researchers in neuropsychopharmacology and molecular genetics in psychiatry operate as if there would be no need for more discrete clinical end points. It is a moot question whether this is the case, but one should not ignore the possibility that in the impasse in the development of psychotropic drugs and molecular genetics of mental illness, the pharmacological heterogeneity of the populations studied, has played a role (Ban 1987 2002).

I think your discovery that the “cycloïd psychoses” are “a spectrum of stress induced psychoses based on special genetic and/or neurodevelopmental predisposition” (Franzek and Beckmann 1999; Franzek and Musalek 2011; Franzek et al 1996, 2004; Pfulhman et al 2004) implies only that Leonhard’s conceptualization of the place of “cycloid psychoses” between the “phasic psychoses” and “unsystematic schizophrenias” is wrong. As I see it, it does necessarily imply that the diagnostic concept of “cycloid psychosis” as a research and clinical end-point is wrong. Arguably, it was Leonhard’s (1957) separation of “cycloid psychoses” from the “phasic psychoses” and “unsystematic schizophrenias” that provided the clinical end-point to discover that this population is different from the other two populations in that it can be induced by stress. Pursuing the same argument further, perceiving this population as a “spectrum of psychoses”

(Akiskal 1983) in the tradition of Kretschmer (1921), will leave one hanging in air, whereas perceiving this population in the tradition of Wernicke (1899) and dividing this population on the basis of Griesinger's (1843) "psychic reflex" model into three diagnoses, i.e., "excited - inhibited confusion psychosis", "anxiety- happiness psychosis" and "hyperkinetic - akinetic motility psychosis", as Leonhard (1957) did, will provide one with clinical end-points for linking the different forms and sub-forms of "cycloid psychosis" to specific genetic and/or developmental predispositions.

I understand that in addition to the discovery that the "cycloid psychoses" are stress-induced psychoses, you observed that in clinical practice, patients who would have been diagnosed by Leonhard (1957) as one or another forms of "systematic catatonia" are mostly diagnosed as disorders of the "autistic spectrum" and patients who would have been diagnosed by Leonhard (1957) as one or another form of "systematic hebephrenia" are diagnosed as "severe personality disorders not otherwise specified". Moreover, you noted that many of those patients with a diagnosis of "severe personality disorder" could be found in addiction care units. (Franzek and Elsenaar 2008). Again, in my opinion, your observations don't indicate that those populations Leonhard described don't exist. For me, all they suggest is that in terms of psychiatric diagnoses, they are differently conceptualized in current consensus based classifications. Yet, even if some patients who would have been diagnosed as one or another forms of "systematic catatonia" or "hebephrenia" by Leonhard (1957), wind up with a different diagnosis based on current classifications, in a multinational survey on "chronic hospitalized schizophrenia" we conducted in the 1980s, we found patients with all the different forms and sub-forms of "systematic catatonia" and "hebephrenia" that Leonhard (1957) described, albeit with a different distribution than in prior samples (Ban, Guy and Wilson 1984a). We also found that patients diagnosed with different forms/subforms of schizophrenia are treated with different doses of neuroleptics (Ban, Guy and Wilson 1984b). Based on these findings and some further observation of patients taken off their treatment with neuroleptics, we questioned the need for maintenance neuroleptic treatment in "shallow hebephrenia" (Ban 1990; Kelwala and Ban 1981).

While your findings and observations challenge the place (conceptualization) of some of the psychiatric diagnoses derived in the Wernicke-Kleist-Leonhard (W-K-L) tradition among psychiatric disorders, a current report by Jules Angst and Christoffel Grobber (2015) is in favor of the validity of "unipolar mania", one of the contested diagnoses that has its roots in the W-K-

L tradition. The arguments against “unipolar mania” were sufficiently strong that the DSM-5 of the American Psychiatric Association (2014), doesn’t recognize “unipolar mania”, and classifies patients with recurrent mania without any depressive episode, in Kraepelin’s tradition, as “bipolar disorder”. Based on a review of literature and their own data, Angst and Grobber (2015) provided support for the diagnostic concept of “unipolar mania”. Of particular importance in this respect, are the epidemiological-genetic findings of Merikangas and associates (2014) and of Vandeleur and associates (2014) which indicate the genetic independence of mania from depression (Hickig 2014). Important also are findings in pharmacological treatment studies which indicate that patients with unipolar mania are different from patients with bipolar mania in that they require more antipsychotics (Grobber and his associates 2012) and are less responsive to lithium salts (Yazici and Cakir 2012).

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