Thomas A. Ban: Towards Personalized Treatment of Depression
(Phenomenological Psychopathology)
Collated by Olaf Fjetland

This collated document includes Thomas A. Ban’s Preface to his monograph, Toward Personalized Treatment of Depression, in preparation, a comment on this Preface after posted by Martin M. Katz and Ban’s reply to Katz’s comment.

This project was terminated and the collated document is now open to all INHN members for final comment.

Thomas A. Ban June 27, 2013 Preface
Martin M. Katz July 25, 2013 Comment
Thomas A. Ban May 19, 2016 Reply to Katz’s comment

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Thomas A. Ban: Preface

I entered the field of psychiatry in 1954 in Hungary, my native country, about three years before the antidepressant effects of imipramine and iproniazid were discovered (Kuhn 1957; Loomers, Saunders and Kline 1957). The presence of serotonin (5-HT) and norepinephrine (NE) in the brain, the monoamine neurotransmitters that were to become the centre of interest in depression for several decades, was already demonstrated (Twarog and Page 1953; Vogt 1954). The spectrophotofluorometer, the first instrument with the resolution power to study the action of drugs on monoamine turnover rate in the brain, was to be introduced within a year (Bowman, Caulfield and Udenfriend 1955).

I started my apprenticeship in psychiatry at the National Institute of Nervous and Mental Diseases in Budapest. It was at the tail end of the period when spinal tap was routinely carried out in newly admitted patients for ruling out cerebral syphilis, and when insulin coma was the prevailing treatment modality for the schizophrenias. Although electroconvulsive therapy (ECT) was already available for some time, opiate tincture was still the predominant treatment of “depression.” An “opium cure,” was a
three-week procedure during which the dose of the substance was raised by daily increments from 3 to 25 minims, then decreased gradually until discontinued (Ban 2001).

Management and treatment of “depression” at that time were based almost exclusively on information about the psychopathology of patients. The psychopathological symptom profile of patients allowed assignment to “prototype-based” or “nosology-derived” diagnoses which provided reasonably accurate predictions about prospective course and outcome of illness and responsiveness to treatment. Psychopathology in the mid-20th century provided the knowledge base that guided education, clinical practice and research in European psychiatry (Ban 2004).

Psychopathology

The term psychopathology was introduced by Ernst Feuchtersleben (1845) in the mid-19th century and used as a synonym for “psychiatry” for more than 50 years. In the early 20th century the use of the term became restricted to the “science” of psychiatry by Karl Jaspers (1910) with a methodology suitable for the study of pathological mental phenomena encountered in the “practice” of psychiatry. The methodology was suitable also for defining the boundaries of psychiatry by separating psychiatric “disease process” from “anomalous personality development.” Within Jaspers’ frame of reference, the signal difference between the two is that “personality development” grows on a predisposition (“Anlage”) in a continuous sequence, with changes brought about by respective age periods, whereas the psychiatric “disease process” follows its own course that interrupts and becomes superimposed on these built-in sequences. It was also suggested that “personality development” could be understood through “meaningful connections” by empathy and introspection, whereas "disease process" could not and had to be explained by a causal factor.

The roots of “Jasperian psychopathology” are in Aristotle’s distinction between “form” and “content” and in the postulation that the “content” of psychopathological symptoms is based on patients' life experiences, whereas the “form” in which the content is experienced is determined by patients’ illness. The recognition that patients with different mental illnesses “process” their sensory-perceptual experiences and ideas differently in their brains, as expressed in the “form” of their psychopathological symptoms, has led to the birth of “phenomenological psychopathology” (“phenomenology”), a discipline that provides a methodology for the detection and
monitoring of mental pathology in patients. For the “phenomenological psychopathologist” (“phenomenologist”), it is not the subject matter that the patient talks about, but the form, how the patient talks; and it is not the content of a symptom, such as a “somatic complaint,” but whether the “somatic complaint” is experienced in the form of “bodily hallucinations,” “obsessive ideas,” or “hypochondriacal delusions,” that is relevant to patient’s mental illness and psychiatric diagnosis (Fish, 1967; Hamilton, 1985; Taylor, 1981).

Phenomenology

The term "phenomenology" was adopted by Jaspers from Edmund Husserl (1859-1938), a German philosopher and mathematician. However, Jaspers’ "phenomenological psychopathology" has nothing in common with Husserl’s "phenomenology,” a philosophy based on the assumption that the "study of subjective experience of psychological events” is the science of sciences: the science that has preceded, and governs, all other sciences. The same applies to Jaspers’ “phenomenological analysis. It has nothing in common with Binswanger’s (1852-1929) “phenomenological analysis,” in which "morbid psychic experiences” are reformulated in terms of Heidegger's (1889 – 1976) "existentialist philosophy" with the basic presumption that this would render "pathologic realities" understandable.

During the years from 1918 to 1933, a group of psychiatrists that included Hans Gruhle and Wilhelm Mayer-Gross in Kurt Wilmanns’ Department of Psychiatry at the University Clinic of Heidelberg spearheaded a “phenomenological analysis” of psychopathological symptoms. Their efforts resulted in a vocabulary that includes distinct terms in reference to pathologies of mental processing, embracing the entire field, i.e., to processing within and across mental structures from the pathologies of “symbolization,” such as “condensation” (combining diverse ideas into one concept) and “onematopoeisis” (building new phrases in which the usual language conventions are not observed), to the pathologies of “psychomotility,” such as “ambitendency” (the presence of opposite tendencies to action) and “parakinesis” (qualitatively abnormal movements). In “phenomenological psychopathology,” “dysphoria,” the negative pole of “vital emotions,” is distinguished from “dysthymia,” the negative pole of mood; “psychomotor retardation,” the experience of a spontaneous slowing down of motor activity, is distinguished from “psychomotor inhibition,” the experience of slowed down or “obstructed” motor activity, etc. (Fish 1967; Jaspers 1913; Nyíro 1962; Taylor 1981).
Furthermore, by linking the different abnormal forms of experience ("psychopathological symptoms") to psychiatric diagnoses, the Heidelberg School created a self-contained language of psychiatry that reflected the pathology in mental processing in the brain. In this language, “tangential thinking,” characterized by talking past or around the point (Vorbeireden), was linked with the schizophrenias; “circumstantial thinking,” characterized by overbearing elaboration on insignificant details, was linked with the dementias; “rumination,” characterized by endless pondering of unpleasant thoughts, was linked with depressions; etc. (Hamilton 1985).

With the employment of "phenomenology" in a period of less than two decades, the Heidelberg group turned psychiatry into an experimental discipline with the potential to separate one mental illness from another. They also separated “abnormal behaviour,” the subject matter of “abnormal psychology” in which behavioural anomalies are perceived as deviation from instrumental means which are accepted as normal for a subject with a particular social background, from “psychopathology, the subject matter of “psychiatry” in which abnormal mental phenomena are perceived within the frame of reference of mental illness (Juhász and Pethö 1983; Schneider 1950).

The "golden years" in the development of "phenomenology" came to an end in 1933 with the removal of Wilmanns from his position by the Nazi regime, partly because of his reference, in his lectures, to the “hysterical blindness” of Adolf Schicklgruber (alias Adolf Hitler) towards the end of the first World War. He was replaced by Carl Schneider, who was to authorize the murder and sterilization of many mental patients. Hans Walter Gruhle, who was the intellectual leader of the group, left the university to take a position at a provincial psychiatric hospital; Wilhelm Mayer-Gross moved to England and, before long, the team that was instrumental in developing “phenomenological psychopathology” as a foundation of psychiatric research at the Heidelberg psychiatric clinic disintegrated (Shorter 2005).

Although the "golden years" had ended, the tradition of Heidelberg continued, even during the tenure of Carl Schneider (1942), who contributed to the dismantling of the diagnostic concept of schizophrenia by describing three distinct symptom complexes in acute schizophrenia he referred to as the “thought withdrawal syndrome,” the “desultory syndrome” and the “drivelling syndrome,” and by maintaining that they are selectively affected by the schizophrenic process. When Kurt Schneider took the
helm in 1946 at the Heidelberg University Psychiatric Clinic, all the different areas of research Jaspers subsumed under “general psychopathology” had evolved into a discipline of its own. Thus, by the mid-1950s, “general psychopathology” included, in addition to “phenomenological psychopathology,” “performance psychology” that deals with “objective,” observable or measurable performances of mental life, such as perception and memory; “somatopsychology” that is focused on somatic symptoms accompanying mental pathology; “understanding psychology” that is concerned with meaningful connections and comprehensible relations, i.e., how one mental event emerges from another; “explanatory psychology” that studies cause–effect relationships in mental life; and “nosology” that provides the methodology for synthesizing psychiatric disease from “psychopathological symptoms” and classifying the diseases synthesized. Kurt Schneider (1957) himself suggested that disturbance of ego(self)-integrity was central to the psychopathology of the schizophrenias and referred to the symptoms which reflect this pathology, such as “thought broadcasting,” “thought withdrawal,” etc., as “first rank symptoms” of schizophrenia. With the application of Jaspers’ (1910) distinction between “personality development” and “psychiatric disease process,” Schneider was first, in 1920, to separate “vital depression,” an illness, from “depressive psychopathy” and “reactive depression.” During Kurt Schneider’s nine-year tenure, “phenomenological psychopathology” flourished once again at the Heidelberg clinic. His retirement in 1955, followed shortly by the introduction of several new “psychotropic drugs,” ended an era in psychiatry with the last attempt to render mental pathology accessible to research directly, in statu nascendi, as the pathology in mental processing emerges in the brain before it becomes translated into social behaviour.

Kurt Schneider’s (1920) “vital depression” with “vital feelings,” (experienced by patients as if their whole body was affected by their illness), “corporization,” (experienced as if they were more ill than in any physical illness they ever had), somatic symptoms and disturbance of vital balance (experienced by feeling tired in the morning), were frequently encountered forms of depression we saw was in the mid-1950s. But clearly, they were only some of the many forms of disease we saw that were diagnosed as “depression.” It was different from the “depression” described by Emil Kraepelin (1899) and characterized by retarded thinking and decreased motor activity (that in some cases was so severe that “stupor” ensued) that made it difficult to distinguish from “catatonic inhibition,” although it differed from it by responsiveness to ECT. It was also different from the many other prototypes of depression: one dominated
by “anhedonia,” another by “depressive evaluations,” a third by “micromania,” etc. (Nyirò 1962). Each of these forms differed distinctly from one another by “elementary” psychopathological symptoms that dominated the clinical picture (Krahl 2000; Wernicke 1892). They also differed in responsiveness to ECT, verbal interaction and life events (Shorter 2013) to the extent that in some forms of depressive illness, e.g., “stuporous depression,” ECT was so reliably effective that it could be used in the differentiation of depressive stupor from catatonic inhibition, whereas in others, e.g., “vital depression,” ECT had no effect, or even a negative effect.

By the time I left Hungary in 1956, I learned that even if almost all the different forms of depressive illness follow an episodic course, with full remission between episodes, and even if almost all patients with depressive illness, regardless of which prototype they fit, suffer severely during their episode, the responsiveness of each patient to treatment is dependent on the form the depressive illness that becomes manifest, i.e., the psychopathological symptom profile of the patient.

Since the time I left Hungary, more than half a century has passed and during these years I have participated in the clinical development of numerous drugs for the treatment of depression, from desipramine to reboxetine, with trazodone and viloxazine in between (Ban and Lehmann 1962; Ban et al 1974, 1998; Petrie et al 1982). Working with patients while studying these drugs, I became keenly aware that each of these drugs can give life if given to the right patient, but can add to the hell of a patient if given to the wrong one. I also learned that, as far as treatment with antidepressants is concerned, the chances are that by prescribing any one of these drugs without identifying the form of depression from which the patient suffers, one could potentially harm (by inducing side effects) at least as many patients as one could potentially help.

By the dawn of the 21st century, “phenomenological psychopathology” had become the forgotten language of psychiatry; neuropsychopharmacologists had become involved studying genetic and epigenetic mechanisms to develop treatment for depression; and psychiatrists were treating patients with a unitary diagnosis of “major depression,” using a wide variety of “antidepressant” drugs with limited success (American Psychiatric Association 1980). Considering that “major depression,” a consensus-based diagnostic concept, provides the only clinical end-point for both treatment and research in “depressive illness,” which is a consensus-based diagnostic concept, it is reasonable to assume that uncovering psychopathology-based diagnoses
covered up by “major depression” is a prerequisite for developing personalized treatment for depression. Verifying psychopathology-based diagnoses should also open the path for the study of the biology and genetics of the different forms of depression.

References:


Fish F. Clinical Psychopathology. Bristol: Wright; 1967.


Jaspers K. Allgemeine Psychopathologie. Berlin: Springer; 1913


June 27, 2013

**Martin M. Katz’s comment**

In a relatively brief, inviting Preface, Tom Ban recounts the history of research in European psychopathology during the 20th century. He details the contributions of many of its leading figures and covers ground unfamiliar to many American psychiatrists. These early workers arrive at different formulations of depression, different diagnostic systems and different treatments. Of specific interest is the development of “phenomenologic psychopathology” referencing the roles of Karl Jaspers and Kurt Schneider, noting that they reopened the science in a more enlightened context. The new antidepressants have clearly shaken the approaches to treatment. Such earlier theoretical concepts have been set aside as clinicians adopt a more practical trial
and/or approach with the new drugs and show less concern for lessons in this historical sphere. Ban is more at home in that context because the approach which relies less on ideas about etiology, provides the foundation for the methodology he will use in the book to “deconstruct major depression, (to) open the path in the study of the biology and genetics of the different depressive subtypes” In so doing he hopes to achieve a “personalized medicine” capable of individualizing the treatment approach for each depressed patient. Ban’s approach will attempt to provide psychiatrists with a new context within which to work. One can look forward to a more complete blueprint for this strategy in the text that follows.

July 25, 2013

**Thomas A. Ban’s reply**

Thank you for your encouraging comments on the “Preface” of “Toward Personalized Treatment of Depression,” a monograph still in preparation, in which the need for deconstructing the consensus-based diagnostic concept of “major depression” is addressed. As you rightly noted, my “Preface” is focused on “phenomenological psychopathology,” a research method of psychiatry developed in Europe in the second and third decades of the 20th century that separates “abnormal psychology” from “mental pathology” and distinguishes between diagnostic concepts (and sub-forms within these diagnostic concepts), on basis of “abnormal forms of experience” instead of behavioral measures and social performance. Employment of “phenomenological psychopathology” should yield diagnostic concepts closer to “prototype-based” diagnoses of “depression” than current “consensus-based” diagnoses. It might also yield pharmacologically sufficiently homogeneous populations for neuropsychopharmacological research in depression. But, even if that would not be the case, it would facilitate a more discriminate use of “antidepressants” by restricting the depressive population to those with “abnormal forms of experience,” and by separating within “major depression” subpopulations with different “abnormal forms of experience.”

August 22, 2013
Collated by Olaf Fjetland (November 16, 2017)