F. Seiden

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Dear Richard,

Your questions are most intriguing, and I hasten to argue my idiosyncratic views against the present drive to the study of anatomic structure as a basis for psychopathology. Since you limit the query to the past 25 years, I am precluded from arguing that the seminal studies of the century remain those of Wagner-Jauregg, Meduna, Weinstein and Kahn, Wikler and Martin. In the period requested, the most impressive studies have been those of Taylor and Abrams in identifying the syndrome of catatonia as associated as much with affective disorder as with schizophrenia (which effectively vitiated the logic of DSM-III); the genetic identical twin studies in London which found both schizophrenia and affective disorder among genetically identical siblings (which effectively discredited the present genetic studies); and which led to the unitary hypothesis of psychosis of Timothy Crow.

From psychopharmacology, I would credit Shagass with the best of modern patient classification devices, the sedation threshold; DeWied and others for discerning that brain peptides have behavioral effects; and the work of the unheralded chemists who developed naloxone, providing the basis for a life-saving technique in clinical medicine.

Your second query is even more intriguing. I can more easily answer the question about the two or three most damaging hypotheses underlying the biological understanding of psychopathology. For example, the belief held by many in neuroscience (and the ACNP and NIMH as well) that studies of rat brains will lead to an understanding of psychopathology is clearly wrong. Rats are not men; rat physiology and pharmacology is readily distinguishable from that of man. Further, normal man is not the same as sick man, since it is the differences induced by illness that are the basis for our interest. Similarly, it is silly to assume that studies of the normal rat bear any relation to mentally sick man.

In the same vein, the genetic and structural hypotheses of psychopathology are old theories, espoused in the 19th century, and probably as wrong now as they were then, albeit we have better ways to picture the structures of the brain.

If I were to argue for the hypotheses that are outside the mainstream, that may lead to an understanding of the bases of psychopathology in brain function, I would point to that model of psychopathology which argues (from the diabetes and neurosyphilis experiences) that the expression of psychopathology is the end product of a stressor (e.g., spirochaete or hypo-insulinemia) and its interaction in a psychological substrate (e.g., ego and superego; or character and personality; or memory and emotional patterns as modified by life experience).

This view is best stated as the unitary hypothesis of psychosis. The best example of this view is the case for catatonia (primary and secondary; malignant and NMS) as a general brain disorder not part of schizophrenia, and one that is highly responsive to ECT. That some patients with dementia praecox exhibit signs of catatonia is not unlikely, given the high probability that catatonia is an endocrine deficiency disorder.

Another support comes from observations that ECT is equally effective in treating mania and depression, with or without psychosis, with or without melancholia, with or without catatonia; with or without dementia ('pseudodementia'). This varied effectiveness is often cited as an example of the 'dirty' nature of ECT; I view it rather as an example of the specificity of ECT and the 'dirty' nature of our psychopathology. (Recall the varieties of neurosyphilis in this context.)

These views are outside the mainstream. That is why I continue to be intrigued by them, since I 'know' (from history), that the pack is usually wrong. And the present neuroscience pack is surely wrong since few, if any, have any experience in the clinic.

If you wish a concise view of catatonia, I urge you to read the recent review by Taylor in NNPBN.

My best regards to Charlie, whose work with the sedation threshold I have long admired, but still cannot comprehend.

Sincerely yours,

Max Fink, M.D.
Professor of Psychiatry