

**Donald F. Klein, Max Fink, Edward Shorter and Thomas A. Ban: Comment
exchange on Flagrant Catatonic Behavior**

August 16, 2017 at 6:00 PM – August 23, 2017 at 11:17 AM

From Donald F. Klein to Max Fink

August 16, 2017 at 6:00 PM

Max

Wanted to make sure you got this. Believe you think Catatonia often subtly present, detectable scale wise and benzodiazepine treatment (BZD Rx) validated.

However do we agree flamboyant postures that led to across the street diagnosis (Dx) are gone? Any ideas? My-guess, Neurotropic virus that killed off patients and themselves, survivors had mutated to less lethal or deteriorative species?? Like 1919 virulent flu epidemic often led to Parkinsonism and disappeared. Or something else??

Best

Don

From Max Fink to Don Klein

Aug 17, 2017 at 9:00 PM

Dear Don,

In the 1950s many catatonics were poorly treated and we saw the classic cases. Now, even if catatonia is not recognized, all sorts of meds are given to change brain chemistry -- poison further and the expression changes.

We are increasingly more successful in educating and early diagnosis. My most recent visitors were 2 docs from South Africa who are seeing case after case. Same true for doc in Buenos Aires.

And even at SBU, now that a resident read some literature, he is finding cases that didn't exist in his first 2 years.

Best regards.

Max

From Donald Klein to Max Fink

August 18, 2017 at 4:16 PM

To follow up our discussion, what I said was in early observations of acute psychosis obvious catatonic behavior was common. Two years later this obvious behavior seem to have disappeared. The question is, "How come?"

It's true back in 1953, I was seeing Pre-antipsychotic, Pre-BZD, acute patients with postural and locomotor akinesia (many locomotor responsive to IV Amytal but then delusions expressed), mutism, psychomotor retardation etc. obvious signs. Catatonia frequent and obvious. Some delusions had what seems to be an explanatory intent. "Every time I close my jaws 30,000 Chinamen die". Often had abrupt, quick, motor and cognitive remissions to ECT- (Doc, did I really believe that?) Dx—Schizophrenia, Catatonic Type was standard.

I should explain being out of touch with psychotic patients for almost 2 years. In 1954 joined USPHS (expecting to take care of tubercular Eskimos for 2 years--better than Korea). Instead

assigned to 1000 bed Lexington, KY. USPHS Narcotic Hospital, about 700 Federal Prisoners Opiate Addicts about 300 Volunteer Status. The Federal crime was mostly mail theft.

Since had 1 year Psych Residency. Was given one day of exquisitely boring lectures, Then, immediately started running the 72 bed methadone withdrawal unit-about 20-30 admissions/week-responsible for daily rounds, medical and psychiatric workup. Supervised 1 junior staff MD -- zero residency-medically inept--Was my successor when reassigned. Excellent experienced nurses kept my mistakes to a minimum. Also, supervision of 200 patient Woman's Unit mostly to defang occasional mild scuffles, Women's Sick Call-Daily, ----So for 18 months had a lot of addict contact-- fit none of my pre-Lexington, preconceptions, (pre-psychotic, deep oral fixations, required opioids to stave off impending anxiety or depression or psychosis etc.) Saw no psychosis, no anxiety states, no lasting severe depression, lot of women. (All ex-prostitute) abdominal -vaginal soreness, tenderness, verging on pain, physical negative, no localizing signs, no fever, no WBC increase: Dx: Chronic PID (acceptable to patient) or Malingering (resisted by patient). Complaint (and Patient) went away quickly when would not relieve from work assignment (e.g., mopping floors).

Patients met criteria for Opioid Dependence and often Antisocial Personality with early onset, lying, stealing, disregard for the rights and feelings of others etc. Aggressiveness was rare. Emphasis on chronicity and treatment failure. I observed that complaints about manipulation, deceit and being conned psychopathic behavioral features were at a minimum in this. reasonably comfortable jail, where there was little to exploit. A famous quote of Oscar Wilde is that he can stand anything except temptation. It is interesting that there was so little manifest psychopathology or antisocial behavior once settled into jail, despite a clear history of deviant personality development and a very poor prognosis.

I was deflected from Catatonia by reminiscence.

After 18 months was assigned to run Kolb Hall Psychotic Unit. About 100 WW One!!! veterans. Pre-VA, chronically psychotic since between 1918-1925, formerly in St Elizabeth's Wash. DC, all drug virgins except for paraldehyde. To keep busy did case reviews, neurological exams and

psychiatric interviews on all. Had been given excellent, attentive, nursing care, opportunity for several occupational trainings where one could become proficient. Every attempt was deceit avoid the demoralizing effects of institutional living. This had failed. They had substantial ECT without benefit. The patients sat around all day, some staring at television, some staring at nothing. However, when it was time to eat, easily got up, formed a line sat in their usual chair, ate with dispatch, with no socialization. The obvious pathognomonic catatonic signs were missing. A few talked incessantly. They were manifestly paranoid, delusional, being tortured by the enemy etc. You could start a reasonable neutral conversation but shortly they would go off on a psychotic tangent about what they were doing to him.

Max hypothesizes that the disappearance of flagrant catatonic behavior is due to various treatments that are now available in the community. But this chronic group had not received such treatments. This observation would be more problematic if this sample had a history of such obvious catatonic behavior. However, I couldn't find any description of their early psychosis. Any reports of follow-up of catatonic cohort. Kahlbaum?

Sending a copy to Tom Ban and Barney (Carroll).

Hope it prompts them and you to comment.

Best

Don Klein

From Max Fink to Donald F. Klein
August 19, 2017 at 1:09 AM
cc. Bernard Carroll, Edward Shorter
From Max Fink to Thomas Ban
August 19, 2017 at 1:13 AM

Dear Don,

Two factors influenced your experience with catatonia -- the populations that you serviced (Creedmoor, Hillside, KY Drug dependence, Columbia outpatient clinics) and the glasses that you wore with a template for "catatonia". In each population, the subjects that came to your attention were filtered. The Creedmoor sample was of chronic mentally ill, with Kahlbaum catatonics galore. You remember the "Kraepelin schiz". But in KY these were prefiltered and admitted elsewhere. At Hillside, the Kahlbaum cases were treated before admission although some were on the Adolescent unit. The adult cases were labeled "SCZ" not CAT. The same filtering affected your samples at the Columbia clinics -- the CAT cases were at Neurologic Institute or treated in the PI ER.

The same population selection was true for me. I recall CAT cases at Bellevue Psych on 30 St during med school and neuro residency, then the same at Hillside. I saw such cases when I was in St Louis at the State Hospital but had no interest. At Metropolitan Hospital I treated the same drug addicts that you treated at Lexington; Abrams and Taylor saw the CAT patients on the hospital's medical wards. My introduction to CAT came in 1980 at SBU as an Attending on the In-Pt service and my service as ECT head and service in Psych ER. Everything changed when Greg Fricchione, fresh from MGH and his essays with lorazepam for CAT, saw cases, treated them with LRZ and then shipped those that failed to me for ECT. Suddenly, 7% of UH admissions met criteria for Kahlbaum/Fricchione CAT.

The second factor was our Dx eyeglasses. CAT = SCZ. By mid-1980 we learned that CAT + CPZ/HLD = NMS. After the Kahlbaum image replaced the Kraepelin, recall that you saw a report of CAT that was a missed Dx and we wrote a Letter indicating that the patient was catatonic. (Fink M, Klein DF. "An ethical dilemma" *Psychiatric Bulletin*. 1995; 19:650-1.)

By 1995 we had Kahlbaum/Fricchione/Morrison/Abrams/Taylor CAT eyeglasses. The SBU 1996 ACTA reports offered new glasses for the profession and these have been picked up widely. Ned and my history of CAT tells this story dramatically; we are putting finishing touches before sending it off to OUP.

All the best. Remember me to Rachel. We should get together for wine and cheese before it is too late.

Max

From Donald Klein to Thomas Ban and Max Fink

August 21, 2017 at 10:51 AM

Has anyone ever published diagnostic reliability assessments on W-K-L? The DSM five assessments were generally felt to be a disaster, although defended by Helena Kraemer.

Best

Don

From Max Fink to Donald Klein

August 21, 2017 at 12:12 PM

Dear Don,

I have not followed the WKL literature and cannot answer the question. The history of catatonia makes clear, however, that each writer, especially the grand book writers, conceived of the syndrome with their own eyes. Their populations differed (acute, chronic, psychotic, neurological and infectious complications, etc). Each is an individual painter, quite distinct in his style. They are to be appreciated as historical moments, deserve approbation as art works in museums, but you and I must see our clinic patients as unique for us. We have tools and interventions that they could not imagine. The remarkable feature about catatonia and its history is that our post 1970s explorations have given us not just another painting but another medium. By applying the Hunterian medical diagnostic model to our present experiences we have identified a unique system, like the switch from painting to You-Tube or Twitter or Facebook, and not just another Picasso or Rembrandt or Gilbert Stuart.

Admire the WKL folk and their products, but do not adopt or copy them. They are best stored in museums.

All the best.

Max

From Thomas Ban to Max Fink

August 21, 2017 at 12:32 AM

Dear Max,

I don't know of any follow up studies on patients with the syndrome Don describes. In the 1980s in our study we could still identify all the different forms and sub-forms of catatonia Leonhard described.

I think the late Christian Astrup published on follow up studies which included catatonic cohorts.'

When I hear the term "catatonia" I associate the name Kahlbaum with it but more strongly my first two patients diagnosed as catatonic, in 1955: one a "negativistic" and the other a "speech inactive". I also remember that they did not respond to ECT but to CPZ (in a low 150 mg daily dose: 50 mg tid). I also remember of being taught (at bedside) how to differentiate depressive retardation from catatonic inhibition. In the 1980s I had some interest in the periodic form but most patients I consulted for "catatonic excitement" or "catatonic inhibition" did not qualify for our criteria (on Leonhard's line) of "periodic catatonia," but qualified for hyperkinetic-akinetic motility psychosis.

We will be addressing issues relevant to "catatonia" in INHN's educational series (Monday postings).

Warm regards,

Tom



From Edward Shorter to Max Fink

August 21, 2017 at 1:41 PM

The most interesting confirmation of the Wernicke-Kleist-Leonhard diagnoses is as follows (from our Catatonia ms):

And this enterprise, however quixotic it might appear to American readers, did have one signal success. In 1962, Fish and Christian Astrup at the Gaustad Hospital re-diagnosed 285 "chronic schizophrenic" inpatients on the basis of the Leonhard criteria. The results were surprising. On the basis of responsiveness to phenothiazines, there were two clearly delineated groups of catatonia; The "non-systematic schizophrenias" were highly responsive to the phenothiazine "tranquilizers," especially levomepromazine and chlorpromazine. Of 43 patients with "affect-laden paraphrenia," 84.4 percent had an excellent degree of improvement; only 1 patient failed to improve. "Cataphasia" followed close behind: 78.5 percent had an excellent response, no response in 3; periodic catatonia had 60.0 percent of the patients responding, only 2 unresponsive. By contrast, patients diagnosed with "systematic schizophrenia" did poorly on antipsychotics: best was "systematic paraphrenia" (40.3 percent an excellent response); worst was "systematic catatonia": none responded well, and, of 107 patients with that diagnosis, 38 didn't respond at all. This study stands as a single sentinel to the fact that there is something there in these clunky Leonhardian diagnoses, at least as regards catatonia. Ref: Frank J. Fish, "The Influence of the Tranquillisers on the Leonhard Schizophrenic Syndromes," *L'Encéphale*, 53 (1964), 245-248.

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From Thomas A. Ban to Donald F. Klein

August 21, 2017 at 3:28 PM

I don't know whether anyone published findings on reliability studies with the WKL system. In so far as I know there are only three instruments which provide algorithms to derive diagnoses in Leonhard's classification (not counting three CODEs). We did not publish findings on reliability studies with the DCR (see review of DCR in the attachment) but Les Morey published findings in his reliability study with CODE-DD, an instrument which incorporates the relevant component of the DCR. The Hungarians might have done and published in Hungarian findings of their reliability study of the KDK Budapest, the instrument that served as a starting point for developing the DCR and the Germans might have published reliability findings with Fritz and Lanczik's Schedule for Operationalized Diagnosis according to the Leonhard Classification (Psychopathology 1990; 23/24). We will address questions like yours in a proper context in postings on INHN's website and I hope you will participate in the exchange that will follow postings.

Best,

Tom

From Thomas Ban to Edward Shorter

August 21, 2017 at 19:13 PM

Thank you Ned.

Tom

**From Donald Klein to Bernard Carrol, Max Fink, Edward Shorter, David Healy, Tom
Bolwig, Tom Ban**

August 22, 2017 at 4:45 AM

Colleagues

If something is there its not much. Outside of lack of hypotheses about how nosological distinctions get translated into therapeutic distinctions, there are data driven statistical problems e.g. choice of contrasts, randomization of medication, effects of covariates, age, sex chronicity, history of asociality, past treatment responses, lack of attempts to deal with multiplicity of contrasts and more --it seems that even their worst prognostic groups did surprisingly well, How was responsiveness defined? any discharge data? Did Astrup and Fish diagnose independently? Any reliability indices re Dx & re improvement? Is original article available in English? At best this is exploratory, far from definitive.

Will await replication.

Best,

Don

Thomas A. Ban to Donald F. Klein

August 23, 2017 at 2:40 AM

Don

Most of the writings of Christian Astrup and Frank Fish are in English.

Both were dead by the time we started to prepare tapes for testing inter-rater reliability. But, if you are interested in the quality of their work may I suggest to read Christian Astrup's Schizophrenia Conditional Reflex Studies (Springfield: Thomas; 1962) and Frank Fish's Schizophrenia (Bristol: Wright; 1962 first edition). (The 2nd edition of the Fish book, revised by Max Hamilton after Fish passed away, was published in 1976).

Astrup classified his acute schizophrenic patients on the basis of Carl Schneider's criteria and the chronic ones on the basis of Leonhard's criteria. His study was done in the US at Johns Hopkins (although it might have ben in New York, as he was brought over by Joe Wortis).

Astrup's sample included 101 patients with different forms and sub-forms of catatonia.

Tom

Donald F. Klein to Thomas A. Ban

August 23, 2017 at 11:17 AM

Tom,

Thanks. My reading list gets longer and longer just to Try To answer my own questions.

Best

Don Klein

