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Catatonia from Kahlbaum to DSM-5

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Background

I regularly have to write fraudulent prescriptions for patients who have a condition that few doctors or pharmacists recognize.

One of these patients is Kate.¹ You would be hard put to guess – as she confidently juggles a busy job, four children, and social commitments – that hidden behind the image is a problem that made her death once seem almost inevitable. A crisis in university 20 years previously led to an overdose and a string of therapists who grappled with the fact that every so often, out of the blue, she would lose her way, become perplexed and suicidal, and finally come to a full stop. Friends or family had to put her to bed. Nothing any therapist ever suggested prevented whatever it was, or stopped it once it started.

She could feel antidepressants like fluoxetine make her emotionally numb and see why some people find that helpful, but it was not for her and it did nothing to stop her episodes. Over the years psychiatrists moved with the fashion from depression to bipolar disorder, and put her on mood stabilisers.

Did these help? *'No. Nothing useful that I could see. The only thing that changed was my weight – I put on over 30 kg.'*

Two things made a difference. *'My friends think I run compulsively but it helps control something.'*

'The only medications that ever helped were benzodiazepines but when I told my doctor this he stopped them.'

The average doctor would have a problem with the fact that Kate seems to wake up on 40–50 mg diazepam.

This is the kind of dose used to manage delirium tremens. On this dose she becomes functional and can get on with her life until the episodes wear off a few days or weeks later.

What happens when you stop? Any withdrawal? *'Nope.'*

None of this fits with depression or anxiety. But few doctors could contemplate giving diazepam in these doses. It is a far darker drug than fluoxetine – so dark that even the brand name Valium[®] has been expunged.

Double-binds

My journey to finding out what was wrong with Kate had started 16 years earlier with Cora. Cora was 18 and beautiful. She had just finished school, where she had been head girl. She had no idea what to do next. Her parents did not approve of her boyfriend, but she was more worried he might leave her. She had a lot of difficult choices (see the Rewriting the 1960s box). At a rock festival, she had a fall. Several days later, looking what was best described as perplexed, she was brought to the unit where I worked.

Had she been traumatized? Had she had a bad drug trip? Had her boyfriend left her? She was close to mute – we could get no answers from her. She was not psychotic, depressed, elated, or anxious. But she was volatile. At times, apparently improved, I gave her leave with her parents, but she was typically brought back confused – sometimes only minutes later. At other times, she lay in bed almost completely unresponsive. I put her on nothing. We just watched over her.

Finally, after 6 weeks she went on leave with her parents, held her own, and did not come back. I filed her case as diagnosis unknown. I heard she went to college, was doing well, and dating the same boyfriend.

A year later she was readmitted – eight and a half months pregnant. Where she had been almost paralyzed and mute before, now she was overactive, but her actions did not seem under her control. Again the best word to describe her was perplexed. After the birth, she went to a hospital with a mother and baby facility. A new team diagnosed schizophrenia. She was put on antipsychotics, but made little progress and the baby was taken from her. Some months later, on weekend leave, having told her parents she was going out for a walk, she laid her neck on the track in the face of an oncoming express train (see the Anna Karenina box).

Looking back, Cora's perplexity and switches between immobility and overactivity make her a textbook case of catatonia. But few doctors know what catatonia is. Mental health professionals know about it, but even we can be guaranteed to miss all but gross cases.

Cora died when a few days' treatment with a benzodiazepine would have cured her. The problem is benzodiazepines are no longer on patent. Drug companies have no incentive to

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help doctors spot catatonia. Instead, efforts go into marketing diseases for on-patent drugs, even if this means conjuring conditions like fibromyalgia out of thin air to market pregabalin, or restless leg syndrome to market ropinirole.

Where have all the catatonics gone?

For 100 years from the opening of the asylums, patients with catatonia lay or stood motionless in odd, sometimes apparently physically impossible postures for days, defecating and micturating on the spot, inaccessible to human contact (see Figure 1). [AQ: 2] They sometimes had to be force-fed through clenched teeth. It was the most lethal disorder in the asylum, as behind the immobility, heart rates, blood pressures, blood sugars, and temperature span out of control (Healy et al., 2012a, 2012b).

For most of this time, catatonia was thought of as a malignant form of schizophrenia. Then, in the 1960s, academic articles began to ask: where have all the catatonics gone? Many offered the view that early treatment with new antipsychotics like chlorpromazine from the 1950s must have nipped the problem in the bud. But antipsychotics make laboratory animals catatonic. It seems unlikely that a drug that makes animals catatonic would cure it in humans (Healy, 2002).

There are no descriptions of schizophrenia before 1809 and no recognition of manic depressive illness before 1854. But in 180 AD, Galen drew a vivid portrait of a catatonic student who just before his exams appeared almost comatose to colleagues, but when he recovered was able to tell them the exact details of the conversation they had around him. Could a condition that has been around for 2000 years suddenly disappear?

One possibility was that modern patients were healthier. It seemed reasonable to me in 1998 to assume that the physical health of patients admitted to mental health units in India

Figure 1. [AQ: 7]



would be worse than that of their British counterparts. So Padmaja Chalasani recorded 100 consecutive admissions to an Indian and then a Welsh unit. She found that one in 10 of the Indian patients had signs of catatonia. So far, so good. Except that Welsh admissions had exactly the same rate. But you had to look explicitly for catatonic signs to spot them. Increasingly trained to ask rather than look, we miss these signs (Chalasani et al., 2005).

What is going on? Sitting back in the historical record, there is an oddity. In 1930, William Bleckwenn announced a cure for catatonia using the barbiturate, amobarbital sodium (Bleckwenn, 1930). The standard histories of psychiatry now say catatonic patients indeed woke up when given amobarbital, but sank back into their stupor when the drug wore off. Electroconvulsive therapy (ECT) in contrast supposedly cured these patients when it came along.

But this does not explain why there were no catatonic patients around from the mid-1960s to be given ECT.

Back to the future

In May this year, the fifth edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-5) will be launched. Even before publication, it has been excoriated as the document that might derail psychiatry. It is difficult to find anyone with a good word to say about it. But DSM-5 does resurrect catatonia. The resurrection is down to the work of one man – Max Fink.

Fink was born in Vienna, Austria in 1923 and emigrated to the USA in 1923. My first hints as to what was wrong with Cora and what might have saved her came when I interviewed him in 1998 (Fink, 2000). Fink is now identified as the saviour of ECT, but in fact had little to do with it until it was about to be swept away by antipsychiatry in the 1960s, when he was the person who stepped into the breach to defend it.

ECT led him to catatonia. 'Convulsive therapy was discovered in the 1930s when given to catatonics. It produced a get up off their bed and walk response that just wasn't seen in other cases of schizophrenia.' [AQ: 3]

Malhi, 2013

'This intrigued me', Fink explained, 'and led me back to 1874 before schizophrenia had been created.' [AQ: 4] In that year, Karl Ludwig Kahlbaum outlined a new condition he called catatonia (Kahlbaum, 1973). Kahlbaum is a mysterious figure. He was not part of the German establishment (Braunig and Krueger, 1999). He rarely gave lectures, perhaps because his first effort to outline how we should classify mental diseases was ridiculed by his contemporaries, although we can now see he had laid out the basis for all modern classification systems (Kahlbaum, 1996).

Kahlbaum described underactive, overactive, and periodic catatonias that could be triggered by shocks – the forced choice or fork-in-the-road type of shock produced when left jilted at the altar – or by physical illnesses, or drug treatments, or other mental disorders. He characterized it as a state of tension that could last days, or weeks, or over a year, after which it usually cleared spontaneously. In the writings of others, a new word took centre stage – perplexity.

Twenty years later, this astonishing disorder was incorporated in schizophrenia where it lay entombed for 100 years until Fink began a campaign to exhume it (Fink and Taylor, 2003).

Catatonia led Fink to Bleckwenn, whose paper makes clear that when patients in the early stages of catatonia were given amobarbital sodium, over two-thirds were cured. Today, we accidentally prevent many cases of catatonia by putting people admitted to a hospital bed on a benzodiazepine hypnotic or tranquilizer.

Catatonia is in DSM-I, -II, -III, and -IV as a footnote to schizophrenia. Fink lobbied to get DSM-5 to make it an independent disorder. He had argued for 30 years that leaving it within schizophrenia or bipolar disorder was dangerous as this leads to treatment with antipsychotics (Fink, 2013).

While schizophrenia can produce catatonia, so too can a range of physical illnesses, drug treatments, and

emotional shocks. In just the way that a range of conditions can lead to cardiac arrests and faced with a cardiac arrest a doctor treats that first and then works out what has caused it, so also doctors need to treat catatonia and then pinpoint its trigger.

Mentally ill?

If 10% of patients going through a psychiatric unit have features of a condition, then this is not a rare disorder. But what does catatonia look like in the wider world?

Milder forms of catatonia almost invariably have anxiety or tension symptoms and most patients at present are likely diagnosed as anxious, depressed, or bipolar, but because of the horror of giving benzodiazepines they are given the wrong treatments.

Anxiety is well known to trigger fight-or-flight responses. But it can also trigger a paralyzing freeze. And this is at the heart of catatonia. This freeze is not just triggered by social stressors. Just as influenza [AQ: 5], the wrong drug or other physical illness that can make us tense, irritable, and anxious, can also cause us to freeze. Mice freeze in front of cats, but laboratory rats freeze in the same way after antipsychotics. Freezing is close to an end-state; before that, there are states of perplexity and anxiety.

Take Violetta, who became psychotic when given a stimulant as an appetite suppressant. She was prescribed olanzapine. When she recovered, she stopped it because of weight gain but became mute and unresponsive overnight and was hospitalized. The hospital staff had no idea what was wrong. Although unresponsive, she was not sleeping, so they gave her a sleeping pill and over a period of weeks she began to respond. They added an antipsychotic, *just in case*, which led to weight gain. Out of hospital this time, she tapered her antipsychotic gingerly and became aware that every taper caused marked

anxiety – but that this could be managed with benzodiazepines.

Neuroleptic malignant syndrome is a version of catatonia triggered by low doses of antipsychotics or antipsychotic withdrawal. It used to be fatal in up to 50% of cases before, in the mid-1980s, it was found to respond to benzodiazepines (Fricchione et al., 1983). This response ran so counter to accepted wisdom that it took almost 20 years for benzodiazepines to be recommended as a treatment.

In Violetta's case, the key lies in her original mute state. Like Galen's student 2000 years ago, where everyone else thought she was inaccessible, when I asked her she said she was aware of every conversation going on around her. 'The problem was every word struck terror in me. I was paralysed by anxiety.'

Benzodiazepines can help many forms of the disorder, but some patients appear to find a way to manage problems through running, physical exercise, or gyms. This makes sense as exercise offers a way to keep action under voluntary control.

Coming out

This journey makes clear what is wrong with Kate; but as things stand, if I want to treat her I have to write a fraudulent script. I can get away with writing four tablets of diazepam a day for a month and telling Kate to pay no heed to what it says on the box, and to take up to eight tablets four times a day if need be. If I write a script for the dose I really want Kate to take or indicate that I want her to experiment with benzodiazepines to find what works for her, the pharmacist would refuse to fill the script and might lodge a complaint.

DSM-5 may change this. The new premium it will put on recognizing catatonia could lead to rapid change if some drug company sold catatonia as part of a marketing campaign. Selling diseases to sell a medicine leads to accusations of disease mongering, but promoting catatonia rather than

Box 1. Did catatonia kill Anna Karenina?

Cora's case echoes Anna Karenina. Catatonia can be triggered by the fork in the road that offers equally impossible futures. It features growing perplexity and heightening anxiety where suicide can appear an escape.

Faced with losing her son, or her lover Vronsky, and maybe both, Anna 'had utterly forgotten where and why she was going, and only by a great effort did she understand the question. ... She thought how miserably she loved and hated him, and how fearfully her heart was beating ... the conductor and other passengers did not notice her panic-stricken face beneath her veil ... a bell sounded and was followed by the sound of shouting and laughter that irritated her agonizingly ... when the train came to the station, Anna tried to think why she had come here and what she intended to do. Everything seemed so difficult to consider, especially in this noisy crowd of hideous people. ... All at once she thought of the man crushed by the train the day she had first met Vronsky, and knew what she had to do.'

Box 2. Rewriting the 1960s

As it vanished in the 1960s, catatonia left a Cheshire cat grin behind it. RD Laing and the antipsychiatry movement were among the most significant cultural forces shaping the 1960s and the revolutions of 1968. The common theme to antipsychiatry and 1968 was that society and family make you mad. This came straight from Gregory Bateson's *double-bind* hypothesis. Nurse Ratchet in *One Flew Over the Cuckoo's Nest* chillingly deployed double-binds. You were double-bound by your mother when she rigidly approached you to kiss you with a body language that screamed the opposite, and even more entrapped if you shrank back and she pressed forward wondering why you were denying your feelings. Double-binds, according to Laing, caused schizophrenia. In the *Divided Self: An Existential Study in Sanity and Madness* and in *The Self and Others*, he made the case by highlighting catatonic behaviour to make his point. Reverse the double-bind and you could cure schizophrenia. Prevent double-binding and you could prevent psychosis.

Cora's case brings out the double-binds that can affect late adolescence. Kahlbaum extraordinarily pinpointed the role of such triggers a century before Bateson or Laing. It is likely that many today who talk about having recovered from schizophrenia or psychosis – especially before 1980 – had in fact catatonia and may have been cured by coincidental benzodiazepines or by time.

It may be time to start rethinking an important part of the cultural landscape of the 20th century.

fibromyalgia or restless leg syndrome would save lives. Perhaps Nike® or Reebok® could be persuaded to get involved – as running almost certainly helps to prevent *freezing*.

A recent best-seller, *Brain on Fire: My Month of Madness* (Cahalan, 2012), may also make a difference. This has sold on the back of an appealing message – if you have been diagnosed with a mental illness, get them to look again as you may have a physical problem.

Cahalan, a New York journalist, abruptly descended into a catatonic state lasting a month that was triggered by N-Methyl-D-aspartate receptor-antibody encephalitis. She pieced together a gripping story from her medical records and video clips. In her version, she was only saved by a lucky encounter with a doctor who

recognized she had a physical rather than a mental problem. After a month and a million dollars of high-powered investigations and immunoglobulin infusions, she recovered. Her recovery and rehabilitation would likely have been with a benzodiazepine only, rather than the cocktail of medications she was put on. But her case serves to remind everyone making a diagnosis of catatonia of the need to investigate what has triggered the condition.

Declaration of interest

The author reports no conflicts of interest. The author alone is responsible for the content and writing of the paper. **[AQ: 6]**

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[AQ: 6]

Notes

1. Kate is a composite patient. Cora and Violetta are pseudonyms for real patients. All patients or their surviving relatives have given informed consent.

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