What historians and clinicians can learn from the history of medicine: the example of fatal catatonia

Edward Shorter

Summary. The utility of medical history to present-day clinicians is not always apparent. One can safely practice cardiology or nephrology without knowing the history of these disciplines. Yet in psychiatry, with its long history of shifting – and often forgotten – diagnoses, engaging in what historians call disease biography and clinicians term historical epidemiology may be helpful in the identification and treatment of uncommon medical conditions.

In this paper we consider the historical origins of malignant catatonia. This acute and often fatal syndrome was identified in 2003 by Max Fink and Michael Alan Taylor, yet remains an unfamiliar diagnosis to many psychiatrists. Understanding this illness, with its various forms of frenzy and stupor, is important today because police and security officers must frequently deal with its victims, and the disorder often ends fatally although today it is readily treatable with benzodiazepines and electroconvulsive therapy.

Keywords. delirious mania; historical epidemiology; malignant catatonia; retrospective diagnosis

The usefulness of medical history to clinicians today is not always apparent. One can effectively and safely practice cardiology or nephrology without any knowledge of the history of these disciplines. How about psychiatry?

Consider the following case. In October 2007 a new immigrant arrived at the Vancouver international airport from Poland. Unaccustomed to travel and unable to speak English, he anticipated that his mother would meet him at the luggage carousel. Unfortunately, neither of them understood that the luggage area was behind customs control, and that he would have to collect his bags – heavy with geography books, his favourite interest – before meeting his mother. Bewildered and apparently incapable of asking for help, he spent the next six hours pacing around the international arrivals area of the airport while his mother, increasingly frantic, entreated airport employees “in broken English” to find him. An eyewitness video recording shows that

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the man, “is clearly agitated, yelling in Polish, and appears to be sweating. He can be seen taking office chairs and putting them in front of the security doors”; he later “picks up a computer and throws it to the ground”. Police were called to the scene and tasered the man, apparently up to four times; he fell to the ground screaming and convulsing, and minutes later was dead².

Of course it is impossible to know exactly what psychological processes the victim experienced, but his behaviour and symptoms are not incompatible with the diagnosis of delirious mania³. Delirious mania, in turn, is the agitated version of what psychiatrists Max Fink and Michael Alan Taylor called in 2003 malignant catatonia, “an acute onset fulminating psychotic and delirious illness, often with hyperthermia that results in death in about half the patients”⁴. Catatonia was until then thought to be a subtype of schizophrenia. Clearly this young man did not develop schizophrenia in his six-hour ordeal at the airport. The diagnosis delirious mania, a type of malignant catatonia, was new to many psychiatrists.

Catatonia is a movement, or motor, syndrome that occurs in the context of various psychiatric and medical illnesses. Fatal catatonia, as the term suggests, includes those cases that may well have a malignant outcome, and the single symptom that most signals danger is a rise in bodily temperature.

Let’s consider a historical example. During the earthquake that struck Rome in 1703, according to physician Georgii Baglivi writing in 1715, few houses were destroyed and no one directly killed, but the great terror everyone experienced meant that some died “with fever” and that those who lay in bed got worse (qui tunc aegroti in lecto jacebant)⁵. This should raise the historian’s suspicion index: People become terrified, febrile, and then die? And they were all in bed? Although, again, we do not know exactly what happened, this is not incompatible with the stuporous form of malignant catatonia, and the term ‘fever’ is a big red flag.

Malignant catatonia, with its several forms of frenzy and stupor, is clearly an important illness because the police must constantly deal with frenzied individuals; stupor may be confused with drunkenness; the disorder often ends fatally; and today it is readily treatable with benzodiazepines and elec-

troconvulsive therapy. Yet for much of medicine’s long history, malignant catatonia has been concealed from view.

The present paper is intended as a contribution to what historians of medicine call disease biography and what clinicians might be inclined to think of as historical epidemiology. An increasing number of medical historians have taken on the challenge of trying to describe how diseases themselves (as opposed to what was thought of them or how they were treated) have changed over the years. Some medical historians, such as Charles Rosenberg, remind us that the identification of diseases as concrete entities, as apparently real as mumps, is fraught with peril, and that diseases are always ‘framed’ by the social and cultural contexts in which they are ascertained. Other historians believe that retrospective diagnosis tout court is so hazardous as to be impermissible, and that the only medical diagnoses possible are those made by living clinicians in living patients. This strikes me as reductio ad absurdum, yet there are those who argue for it.

There have been several notable efforts to write the biographies of various diseases, such as George Rosen’s efforts on ‘nostalgia’ (see below) and Victoria Harden’s account of Rocky Mountain spotted fever. Psychiatrist Conrad Swartz and medical historian Edward Shorter have also hazarded a history of psychotic depression. So though we are wading here in potentially murky waters, this may be where an interesting catch is made: The literature reviewed here about acute delirium and fatal catatonia has not, to my knowledge, been previously evaluated by a medical historian.

**Acute delirium**

Among the older medical and psychiatric diagnoses, the diagnosis of acute delirium probably came closest to fatal catatonia. To be sure, there are even older terms: ‘Frenzy’ was known to the ancients, and in these accounts of frenzy with fatally-ending fevers accompanied by mental confusion and

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disorientation there was doubtless some malignant catatonia. Yet there were many infectious fevers too.

It was Alexandre Briere de Boismont who, in his private psychiatric hospital in Paris, first separated systematically in 1845 the cases of acute delirium (*délire aigu*) from the great mass of infectious conditions, gross brain injuries and cases of *stupidité*, or stupor. Acute delirium, he said, was always accompanied by fever, was of brief duration (of sixteen cases, twelve lasted less than fifteen days), and was often fatal, especially when the patients refused to drink: of nine with ‘hydrophobia’, seven died. “The patients are agitated, frenzied (*furieux*); they have to be contained with a straitjacket. Their entire mind is affected. They cry, gesticulate, speak with extreme volubility, do not respond at all to questions; they refuse to drink.” Briere described patients who clenched their jaws with great force and contorted their entire faces at the approach of a cup of water. “Their face is red, their pulse febrile”. He described mainly agitated cases (it is likely that such patients were more likely to be admitted to an institution than stuporous ones). “What is *délire aigu*?” he asked. “It is a nervous disorder that doubtless corresponds to a modification in the brain”\(^\text{10}\).

Thus acute delirium began its victory march. In 1859, Louis Calmeil, chief physician at the government asylum in Charenton, a Paris suburb, gave a careful account of acute delirium, which he refused to call *délire aigu* and instead called “insidious periencephalitis”. (“*Délire aigu*” was, however, the editor’s running head at the top of the page!) Calmeil described various series of patients at Charenton, all of whom came to autopsy, with this diagnosis. Series one, for example, had a sudden explosion of fury, followed by febrile symptoms and a rapid death. Briere had failed to differentiate his acute delirium patients from those in the early stages of neurosyphilis, yet Calmeil did so and provided a much tighter picture of a distinct disease\(^\text{11}\).

*Other early clinical reports*

Cases that sound like fatal catatonia were reported with many different diagnoses, and while there is no certainty that these corresponded to the fatal subtype of catatonia, they at least raise our suspicion index. Fatal

\(^{10}\text{Alexandre Briere de Boismont, }Du \text{ délire aigu observé dans les établissements d’aliénés, Paris, Baillière, 1845, pp. 104-105.}\)

\(^{11}\text{Louis Calmeil, }Traité \text{ des maladies inflammatoires du cerveau, Paris, Baillière, 1859, vol. I, pp. 153 ff.}\)
Fatal catatonia

cases of ‘hysteria’ in otherwise healthy young women offer an excellent example. Shortly after the death in 1853 of a beloved sister, Auguste H, age eighteen, of Berlin developed symptoms of furious mania. Admitted first to a district hospital, then to the psychiatric division of the Charité hospital, she developed convulsions, together with tonic rigidity of the jaw and neck muscles. A fever was treated with ice packs. She began grinding her teeth loudly, simultaneously boring her head back into the pillow and continually hurling it right and left. Her breathing became laboured, her skin cyanotic, and two days after admission she died. What is going on here? This sounds like fatal catatonia, but even today there is no biological test for catatonia. To be sure, among those today who respond to a test called intravenous dosing, ninety percent do well on the benzodiazepine agent lorazepam. But for these historic patients we have no way of determining what they really had.

Henry Maudsley, a psychiatrist at the West London Hospital, was however certain of his ‘patients’ disorder: “When the temperature rises notably in cases of insanity”, he wrote in 1867, “we may then justly suspect [...] a tendency to fatal exhaustion”. His furor transitorius was the equivalent of what others described as furious mania. “When recovery does not take place, the disease passes into chronic insanity, or into dementia, or ends fatally.”

Another diagnostic equivalent of fatal catatonia seems to have been much of what was called ‘nostalgia’, or Heimweh in German. Young men and women who had never previously been out of sight of the village steeple would go off far away on military service, as servants in the big city, or as members of religious orders, become melancholic, develop fevers, and die. “I have seen nostalgia terminate by incurability and by death”, wrote Bénédict-Augustin Morel, chief psychiatrist at the Saint-Yon asylum near Rouen, in 1860. This is interesting: we have sudden deaths in a population of otherwise healthy twenty- to thirty-year-olds. Melancholia is not typically a fatal illness, though inanition and suicide might number among causes of death. Some clinical reports say the death was preceded by a fever or other catatonic symptoms. A portion of these Heimweh fatal-

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13 M. Fink and M.A. Taylor, Catatonia, cit., p. 159.
ities were almost certainly owing to fatal catatonia, though our uncertainty index is high\textsuperscript{16}.

At a time when the boundaries of melancholia were much larger than today, ‘pernicious’ melancholy, meaning with a fatal outcome, also raises the catatonia red flag. In 1908 Berlin psychiatrist Alfred Döblin (who also authored in 1929 the novel \textit{Berlin Alexanderplatz}) reported two such cases, one with mimicry and mutism; neither had an organic condition that might explain the sudden death; both reminded him of ‘senile catatonia’\textsuperscript{17}.

Medical diagnostics before 1900 abounds with various furores, frenzies, and other agitated confusional states. They doubtless had many causes. Yet catatonia as a cause of those ending fatally, or just barely snatched from death, has not been widely considered.

\textit{The discovery and disappearance of catatonia}

Meanwhile in Germany, the science of psychopathology – the careful description of individual symptoms and their assemblage into syndromes and diseases – was making progress. In 1874 Karl Kahlbaum, owner and chief physician of a private psychiatric hospital in Görlitz in the eastern part of Germany, described catatonia as a distinct illness. The individual symptoms had long been known, but under other labels such as catalepsy, stupor, and of course acute delirium. Yet such was his prestige as a research psychiatrist that the label catatonia caught on instantly in Germany and abroad. Kahlbaum conceded the possibility of fatal outcomes: “Catatonia is probably a directly lethal psychosis, just as progressive paralysis [neurosyphilis] is, in contrast to most of the other psychiatric diseases”. He said that in other psychiatric illnesses the brain is not involved when the cause of death is exhaustion, food refusal, or some somatic complication. “But in

\textsuperscript{16} See George Rosen, \textit{Nostalgia: a ‘forgotten’ psychological disorder}, “Psychological Medicine”, V, 1975, pp. 340-354. Rosen, citing the French surgeon Dominique-Jean Larrey, says that on Napoleon’s retreat from Moscow, a number of young troops developed “Cerebral excitement [… ] accompanied by certain physical symptoms. The temperature of the head is elevated, the pulse accelerated, the conjunctivae reddened, and the patient exhibits unusual movements of the body as well as rapid and incoherent utterances.” All symptoms worsen and the cases end fatally (p. 348). This is a description not incompatible with fatal catatonia. See also Stanley W. Jackson, \textit{Melancholia and depression from Hippocratic times to modern times}, New Haven, Yale University Press, 1996, pp. 373-380.

catatonia the end of life is the final stage [of the disease] and in some sense the most extreme developmental form of stupor (Attonität), comes from the disease process itself.” Kahlbaum also attached a great deal of importance to the stupor found in melancholia, “for it appears to some extent as a kind of vita minima, like syncope and lethargy, and after a long period of chronicity […] appears to go over into a complete stoppage of vital functions”18. Thus stupor in either catatonia or melancholia was a potentially fatal symptom.

Catatonia’s period of independence in German nosology was brief. Yet the point is that as long as catatonia remained an independent diagnosis, authorities admitted the routine possibility of a lethal outcome. It was said by Heinrich Schüle, chief physician at the prestigious Illenau asylum in 1878, that “Given the weakened nervous and circulatory systems - a small, quick pulse, cool skin, rapid emaciation –, a quick lethal ending is in the offing”19. After catatonia became a subtype of schizophrenia, a lethal outcome was no longer admitted as part of the standard course. As long as there was no effective treatment for catatonia or melancholia, the failure to correctly diagnose stupor or manic fury did not matter so much. Yet catatonia became treatable in 1930 (with barbiturates)20 and with chemical convulsive therapy in 193521; melancholia was treated successfully in 1938 (with electroconvulsive therapy)22. Thereafter, the correct diagnosis did matter, because schizophrenia responded to none of these treatments.

It was Emil Kraepelin, the great German nosologist, first in Heidelberg, then after 1903 in Munich, who in 1899 put an end to catatonia’s independence, in the sixth edition of his famous textbook. As late as 1896, in the fifth edition, he considered Kahlbaum’s catatonia as an independent illness entity that normally ended in dementia (‘Blödsinn’, not ‘Demenz’, was the term he used). In the fifth edition, he delivered the kind of careful psychopathological description for which the Germans were known – in contrast to the French with their speculative interest in neuropathology. Yet he showed little interest in lethal outcomes: “In a few cases it does happen that catatonic perishes from the illness amidst extreme agitation bordering on

18 Karl Kahlbaum, Die Katatonie oder das Spannungsirresein, Berlin, Hirschwald, 1874, p. 97.
exhaustion”. Kraepelin claimed that deaths from tuberculosis were far more common. In the next edition in 1899 he said catatonia was a subtype of dementia praecox, with a grim prognosis: only thirteen percent recovered, the others ending in terminal dementia. He repeated the glancing comment of the previous edition that “a few cases” end fatally. In sum, catatonia was very much like paranoia and hebephrenia: a relentlessly downhill course and ugly termination, but not really a fatal disease.

The sixth edition of Kraepelin’s textbook in 1899 thus marked the end for much of world psychiatry of catatonia as an independent disease, and an end to the discussion of fatal catatonia as well.

Stauder’s catatonia

In 1934 catatonia re-emerged dramatically, and this time in its lethal form. Karl Heinz Stauder, a junior clinician at the University Psychiatric Hospital in Munich, called lethality not just a possible outcome but a subtype of catatonia. He reported twenty-seven cases of catatonia that had ended fatally in the Munich clinic. A student of Oswald Bumke, who was not a big fan of Kraepelin’s dementia praecox, Stauder did not see catatonia as a subtype of schizophrenia:

The catatonia cases presented here as a unitary group had the following features in common: an acute onset of the psychosis, a short, acute course, fatal outcome and common symptoms in the clinical picture. They also had some somatic features in common and, finally in common, lack of unitary autopsy results.

Patients in the Munich clinic did not offer the same picture of excited agitation described by others. As the illness approaches the end,

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Now the patients are mainly lying in bed, in spasmodic positions, or are tossing themselves about hither and yon, with jaws cramped shut; every muscle is tensed to the utmost; the patients attempt again and again to pitch themselves from bed or relentlessly beat their own bodies with their fists. Now they appear as animals that are being hunted to death. […] The agitation continues until their last energies have been exhausted.

Death is now hours away. Among the characteristic somatic symptoms in addition to fever were skin haematomas, a possible consequence of the fury with which the patients beat themselves or hurled themselves against fixtures (anticipating what would later be called catatonic self-injurious behaviour); and cyanosis, in particular of the hands and feet. Stauder left open the question whether fatal catatonia represented a distinctive clinical syndrome, as opposed to being a “unitary group”. Stauder’s careful clinical and pathological description has never since been matched and even today fatal catatonia is referred to among psychopathologists as “Stauder’s catatonia”.

**Fatal catatonia after Stauder**

In German-speaking Europe, fatal catatonia became a significant diagnosis after the war. O.H. Arnold, who had case records on 142 patients at Vienna’s Steinhof mental hospital for the years 1935-47, in addition to fifteen cases of his own, said that the clinical profile was unforgettable: “The picture of such a truly frenzied (tobsüchtigen) patient with his heedless and meaningless explosion of energy belongs to the most dramatic in psychiatry”. (Arnold is remembered for treating fatal catatonia successfully with intensive electroconvulsive therapy, or Schockblocks). Fatal catatonia thus entered the German tradition at just the moment when German diagnoses started to matter less internationally.

Stauder’s fatal catatonia never really caught on in the big world of psychiatry outside of Germany. Perhaps the time was not right: German scientific attainments after the Nazi seizure of power in 1933 tended to be neglected abroad. One can understand why! The Germans themselves had caused much of the fatal catatonia of the war, and the French used ‘délire

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aigu’ rather than ‘Stauder’s catatonia’ to describe it. The following scene of horror was not uncommon: As the terrified civilian population of northern France fled the advancing German armies in 1940 (and Stuka airplanes bombed the crowded roads from above), there was massive fear. Some of the worst afflicted found their way to the psychiatric hospital at Auxerre, where there were seventeen cases of délire aigu, almost all with a fever and ending fatally. In 1984 Pierre Scherrer described some of these dramatic cases on the basis of hospital archives. It was a kind of epidemic of fatal catatonia, but one caused by the events themselves rather than by contagion. Scherrer concluded, “The national catastrophe embodied in this immense exodus of unfortunates fleeing the enemy armies, pursued and slaughtered by aerial bombs, triggered many acute mental disorders: mania, melancholia, la bouffée délirante [short-duration psychosis], mental confusion, and, their ultimate complication, délire aigu”.

Evident catatonia vanished into these vague but non-German diagnoses.

Fatal catatonia fared no better elsewhere. Juan José López Ibor, professor of psychiatry in Madrid, though heavily influenced by German professors such as Kurt Schneider with whom he had studied, made little use of the catatonia concept in his classical 1950 study of psychotic anxiety. Yet he dilated at length upon “anxiety and death”. He invoked the German philosopher Martin Heidegger to make sense of the data. Fatal catatonia might have offered a more economical explanation.

In 1956 Toulouse psychiatrist Paul Guiraud, who, together with Maurice Dide, wrote a highly successful guide to clinical psychiatry that first appeared in 1922, noted of délire aigu that it was called abroad ‘catatonie grave’ or ‘catatonie mortelle’, using Stauder’s term without his name. Today, said Guiraud, death from catatonia was becoming increasingly rare. An additional consideration: the diagnosis of schizophrenia was so powerful that the detachment of one of its subtypes into an independent illness seemed reckless.

As late as 1967 Henri Ey, at the Bonneval Psychiatric Hospital near Chartres, along with his collaborators, offered a crisp account of acute delirium, a syndrome that was “generally fatal”. It was characterized mentally by “profound confusion or an intense oneiric [dream-like] psychosis with

30 Juan José López Ibor, La angustia vital (patología general psicosomática), Madrid, Montalvo, 1950, pp. 128-134.
very violent agitation”. Physically, there were “hyperthermia, dehydration and hyperazotemia [high serum nitrogen, usually urea]. As the temperature approaches forty or forty-one degrees, motor agitation is intense and disorganized. The countenance bears expressions of fright and terror. The patient violently resists all those who approach him”. The pulse is elevated, blood pressure fluctuates, respiration is rapid and there is “generalized sudation”.

It is bewildering that these authoritative clinical accounts from Germany, under fatal catatonia, and from France and elsewhere, under délire aigu, had so little impact internationally. The major American textbook of the day, dominated by psychoanalysts, emphasized catatonia as a result of “abnormal communication”.

The end of délire aigu in francophone Europe

In the background of these specific nosological events moved great currents of medicine and society. The end of the Second World War marked the beginning of American medical ascendancy, particularly in the vast sums for research allocated by the National Institutes of Health. Within psychiatry, psychoanalysis consummated its victory march. And the rest of the world started to fall in line behind American medicine.

Between Brière in 1845 and DSM-III in 1980, it was France that dominated world literature on acute delirium. The French recognized catatonia after Kahlbaum’s work, but rarely have French investigators associated acute confusional states with catatonia, which was for them and most others a subclass of schizophrenia.

French diagnostic independence from the United States ended with the publication of the third edition of the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders in 1980. A French translation was soon available. And because the Americans did not recognize acute delirium at all, and saw catatonia only in the context of schizophrenia, the French began to do so as well: délire aigu started to seep out of French psychiatric diagnostics thereafter.

As well, in 1975 the international disease classification of the World Health Organization (ICD-9), based in Geneva, split the discussion of delirium and

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34 For an overview of these events, see Edward Shorter, The health century, New York, Doubleday, 1987.
stupor entirely from that of catatonia. For the WHO, catatonia was a subtype of schizophrenia; the delirium-stupor group was part of “transient organic psychotic conditions”. The ICD-9 did, however, include acute delirium. Neither the schizophrenia nor the delirium-stupor sections mentioned a subtype with a lethal outcome35. The tenth edition of the manual in 1992 dropped acute delirium and mentioned merely stupors of various kinds, including the cata-tonic stupor of schizophrenia36. Fatal catatonia had truly been extinguished!

As a postscript it might be noted that the diagnosis of fatal catatonia has begun to experience a revival, beginning with the work of Stephan C. Mann and his collaborators in 198637. In 1999 Max Fink described delirious mania, which is really the core diagnosis of malignant catatonia, calling it “a syndrome of excitement, delirium, and psychosis, of acute onset”38. In 2001, Fink and Taylor proposed expanding malignant catatonia to include neuroleptic malignant syndrome (NMS), a febrile, potentially fatal side effect of neuroleptic treatment; and toxic serotonin syndrome (TSS), a similar condition following the use of Prozac-style agents that inhibit the reuptake of the neurotransmitter serotonin at the neural synapse39.

A cross-disciplinary thought

For historians and clinicians alike, there are advantages and disadvantages in this kind of exercise.

For historians, one advantage is the deployment of their skills in a useful and productive way, not just as knowledge for its own sake but in the service of healing. If the ascertainment and treatment of these agitated and delirious motor conditions is improved through insight into their historical shape, one can only be thankful.

38 M. Fink, Delirious mania, cit., p. 54.
It is also useful for historians of psychiatry to realize that psychiatric illnesses may have a biological as well as a social dimension. In past historiography we have heard a great deal about the social side: women and hysteria, ‘confinement’ in the asylum and the rise of capitalism, and so forth. But catatonia is a biological brain state and not affected by capitalism: This insight, too, is welcome.

For historians, the disadvantage is the flip side of the above point: darting about time and space in this manner discourages the contextualizing of these medical conditions. The poor Polish immigrant at the Vancouver airport: How did the experience of growing up in Poland serve him ill in this new and unfamiliar environment? Would a Canadian at the Warsaw airport have experienced events differently? Were Brière de Boismont’s female patients more vulnerable by virtue of their gender, and the time and place in which they had grown up? Would Italian women today, their nervous systems perhaps more hardened by the rough and tumble of Rome’s traffic, have been less vulnerable to pitching over the cusp of catatonia?

For clinicians, the advantage of historical epidemiology is its ability to aggregate large numbers of patients over vast reaches of time and space. Fatal catatonia is not a common condition: One will never see enough in one’s clinical practice to form an independent impression of its presentation and course. But understanding fatal catatonia historically places some of the acute encephalitides seen by neurologists today in perspective: These brain inflammations may stem from the malignant catatonia syndrome, and respond to the standard treatments for catatonia in a way that acute encephalitis in general does not.

The disadvantage for clinicians is uncertainty about the diagnosis: Not being able to validate the diagnosis of catatonia with a trial of benzodiazepines, nor verify it with a course of electroconvulsive therapy, means that at the end of the day, we cannot really be sure what some of these patients had. For other patients, the nature of the illness virtually flies in our faces with its obviousness. But there remains for many patients in the past a zone of uncertainty, and it is unwise to pontificate too loudly about the advantages of retrospective diagnosis, because clinical diagnosis is an art of its own.

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41 One autopsy-based study of acute catatonia deaths in psychiatric inpatients excluded the possibility that the psychosis could have had a “direct influence” on most of the deaths. Rosemarie Locher, Über den plötzlichen Tod bei Geisteskranken und die akuten, katatoniformen Zustandsbilder mit tödlichem Ausgang, “Monatsschrift für Psychiatrie und Neurologie”, CIII, 1940, pp. 278-307.