THE MADNESS OF FEAR

A History of Catatonia

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In memory of Martha Gross Fink

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PREFACE

Sometimes my body becomes demented.
Male, 19, at West Park Hospital in Surrey, 1939.[1]

What are the real disease entities in psychiatry? This question has bedeviled psychological medicine for a hundred years or more. Now, you'd think that this would be high on the research agenda of psychiatry. But it is low, and such basic science fields as neuroimaging, neurochemistry, and genetics carry the day instead. Not that there is anything wrong with laboratory science. But before you can study the role of brain circuits or cerebral chemistry, you have to be able to specify how the various diseases present clinically, meaning what the patients actually look like, and then identify homogeneous cohorts for study.

This brings us to catatonia. Unlike schizophrenia, we know what symptoms count as catatonic. Some are serious, with patients dying as their temperatures accelerate; they become dehydrated because they refuse to drink, and they risk inanition because they refuse to eat or move. Autistic children with catatonia may repeatedly hit themselves in the head. We don't really know what catatonia is or its causes, in the sense that we know what pneumonia is. But we can identify it and recognize its many forms, with the added benefit that catatonia is eminently treatable. We can make these patients better on a reliable basis. The same can be said of few other disease entities in psychiatry.

But why has there been so little psychiatric interest in catatonia? Why is it simply not on the radar of most clinicians? Actually, catatonia occurs in many medical illnesses, but it certainly doesn't leap to mind among internists or emergency room specialists. Why this ignorance? That's why we're writing this book. It is a remarkable story about how medicine flounders and then finds its way. And it will help doctors, their patients, and the public to recognize catatonia as a core illness in psychiatry and in medicine in general.

Psychologically, catatonia seems associated with fear. It is not clear if fear causes it or if fearful images surge from the inner mind during stupor and sustain it. Patients' faces are often filled with fear, and afterward they say they thought a catastrophe had occurred. In a catatonic stupor, they are usually motionless, their muscles rigid, as animals are in a fearful response to a predator. Maybe, in the long haul of evolution, Nature has built these fearful responses into us—and we still have them.

ACKNOWLEDGMENTS

Interest in catatonia came full forward with my assumption of clinical responsibility for the ECT Service and the treatment of acutely ill patients admitted to the Psychiatric Services of the University Hospital at Stony Brook University in 1980. Gregory Fricchione, then in charge of consultations, brought the wealth of case material to my attention and encouraged effective treatment protocols. Collaboration with Michael Alan Taylor, who challenged the Kraepelin delineation of catatonia as schizophrenia in the 1970s, led to our review of the place of catatonia in the classifications of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) and to the publication of our texts on catatonia in 2003 and on melancholia in 2006. Collaborations with Andrew Francis, Georgios Petrides, and George Bush, among other Stony Brook staff members, led to the catatonia rating scale, the sedation test, and the optimized treatment protocols with benzodiazepines and induced seizures. David Healy's tests of the incidence of catatonia in Wales and India reinforced our studies. My acquaintance with Edward Shorter began with his history of the shock therapies, written with David Healy; our clarifications of the types of catatonia; and our collaboration on the history of melancholia and now catatonia. I am also indebted to Krysten Nyitray, archivist of the Stony Brook University Special Library collections for her support of the Max Fink Archives.

—Max Fink

It was Max who interested me in catatonia, and I wrote the historical part of this book with a real sense of, "here, we are reviving psychiatry's ancient wisdom for the benefit of a contemporary world that has largely forgotten it." Catatonia is a disease concept that is simply unfamiliar to many, and we hope that *The Madness of Fear: A History of Catatonia* will increase its recognition among clinicians and the general public. Susan Bélanger, the administrator of our history of medicine program in Toronto, helped immeasurably with this task, as did Esther Atkinson and Hannah Johnston. Our agent, Beverly Slopen, was a kind and immensely well-informed ally. We are grateful to Andrea Knobloch at Oxford University Press for her assistance.

—Edward Shorter

1

INTRODUCTION

Yes, this rigidity of his, his fits of perspiration, that periodic twitching of the eyes, he observes us intently, but won't talk or eat, all that looks like catatonic trouble.[1]
—A description of Franz Biberkopf, in Alfred Döblin, *Berlin Alexanderplatz* (1929)

"Our son is an athlete, a college recruited athlete," said one mother of a lad of 21 who lapsed into catatonic mutism and staring. "At the hospital, the nurses would seek to draw out speech by asking him about his sports. Always, no response."

Then one day, a nurse handed him a "ball" that she had made from the Play Doh at the craft center.

"He reached for the ball, got out of bed, went into the hall, and started pitching." The mother went to catch his throws. He would respond to her commands.

"I'd say: 'full wind-up.' He'd pitch full wind-up.

"I'd say, 'now, out of the stretch.'" He'd switch to stretch position.

"I'd say, 'runner's going.' " He'd do his pivot and throw to 'first base' to stop the steal."

Finally, the nurse said they couldn't throw balls on the unit, and he went back to bed, and was silent again.[2]

THIS IS CATATONIA

This is catatonia. It is quite common. About 10 percent of the patients in a psychiatric intensive care facility are found to have it. Here also is good news: three-quarters have a full remission within 2–4 days when recognized and properly treated.[3] Yet, for many physicians, to say nothing of the patients and their families, catatonia has been a mystery. "The structure of catatonia remains to be discovered," said Jo Ellen Wilson and Stephan Heckers at Vanderbilt University in 2015.[4] We have the skills to find it and to treat it successfully (better than almost all other disorders labeled in the *Diagnostic and Statistical Manual of Mental Disorders* [DSM-5]). But first it must be diagnosed.

Catatonia is a behavior syndrome of movement and mood, classically marked by stupor, mutism, posturing, rigidity, and repetitive speech and acts. Usually acute in onset, its signs are recognizable, and, when recognized, it can be successfully treated.

What is it? Not an infection, nor a specific tissue pathology, nor a posttraumatic consequence. Like schizophrenia and personality disorder and melancholia, we know it when we see it. But what is it in Nature?

We wonder whether catatonia has been put into a very weak part of medical neuroscience among pill-prescribing psychiatrists because it is more often seen on acute medical services. These clinicians are best enabled to treat the syndrome successfully.

"I assume," says Max Fink, "that I had been shown catatonic patients during my days in the 1940s in medical school and residency training. Indeed, I recall walking through hospital wards, dressed in the short white coat of the student, with two vials of the barbiturate Amytal sodium, each of 500 mg in one pocket, a metal box with sterile syringe and needles, tourniquet and bottled water in the other, to sedate the excited and the manic and to relax the negativistic and the mute.[5] But during the decades of clinical practice as a research physician in New York and St. Louis hospitals, I cannot recall recognizing catatonia as a distinct syndrome. In my research positions, I had little front-line responsibility to examine and treat the acutely ill.

"It was during a visit to the Bakirköy Hospital in Istanbul in 1965 that I saw nude women, standing in Christ-like postures, in hospital windows and rows of posturing men. Turan Itil, my research colleague, and I were visiting to supervise a study of a new neuroleptic, butaperazine. When we arrived we were welcomed by a patient band, dressed in nineteenth-century Turkish pantaloons and multicolored shirts, an image of the mental hospital before the psychopharmacology era.

"My interest in catatonia was aroused when I became responsible for the teaching of students and the care of patients on the in-patient unit at University Hospital at Stony Brook. My experience with a fully restrained delirious woman, sick with lupus, referred for ECT, and the resolution of her illness pointed me on the road taken."[6]

CATATONIA IS IDENTIFIED

Catatonia today is a syndrome that is identifiable, verifiable, and eminently treatable, a circumstance existing in the armamentarium of psychiatry only for melancholia and neurosyphilis. And like melancholia and neurosyphilis, catatonia is a disease of the brain. Karl Kahlbaum, who coined the term in 1874, said flatly, "Catatonia is a brain disease with a cyclically variable course."[7] There is a psychological component, yet the main symptoms of catatonia—stupor, agitation, "waxy" flexibility of the limbs, mutism, and negativism—seem brain-driven. The attacks often come suddenly.

Stupor is very common. Stupor does not mean a clouding of consciousness just a peg above coma. It means being shut off from communication with the outside world while remaining mentally awake. The patients' eyes dart about freely, and patients later remember most of what occurred around them during the stupor even though they were silent, motionless, and unresponsive to communication or pain. This book has one piece of news about what happens in stupor, and it is news that gives the book its title: these patients are frequently overwhelmed by fear, dread, and anxiety. They imagine that their house has burned down or that the hussars are coming. These are psychotic symptoms, out of touch with reality, and we therefore speak of "the madness of fear."[8] Catatonic stupor is a terrifying experience, not a gentle oblivion.

We recognize degrees of stupor going from the marble-like rigidity of apparent death to a transitory fixing of gaze on a spot on the wall. Catatonia is more than a movement disorder, although it differs from other psychoses in embedding movement in its core definition. It entails negativistic behavior and psychotic ideation as well as rigidity, immobility, posturing, muscle tension, stupor, agitation, tics, echolalia (repeating others' words), echopraxia (imitating others' movements), and mannerisms.

There are two forms. The catatonic symptoms that are present in other diseases might be termed "*catatonic syndrome,*" as they adhere in a recognizable syndrome

rather than just occurring as isolated "features." Then there is the full-fledged form of "*Kahlbaum's disease,*" which Kahlbaum described in 1874. Kahlbaum's catatonia and the catatonic syndrome are part of the same underlying condition, all parts of which are responsive to benzodiazepines and to electroconvulsive therapy (ECT). Parkinson's disease offer an interesting parallel, as we speak of Parkinson's disease, which is a disease *sui generis*, and a row of other diseases with parkinsonian symptoms.[9]

Kahlbaum's catatonia and the catatonic syndrome are related manifestations. Their basic unity is the commonality of their response to the anticatatonic treatments.[10] Among the guidelines in modern psychopharmacology is the belief that the common response of a population to a single treatment identifies their biological commonality. It is useful to insist on Kahlbaum's catatonia as a separate presentation to counteract the field's century-long tradition of diagnosing catatonia as "schizophrenia" when psychotic symptoms appear. This "schizophrenia" tradition in the interpretation of catatonic symptoms has been a barrier to effective care. Calling attention to Kahlbaum's catatonia is a start on breaking the stranglehold of "schizophrenia" on catatonia in its psychotic manifestations. The test of treatment responsiveness is also a metric of other diagnoses that have sprung up as supposedly independent clinical entities, such as neuroleptic malignant syndrome and pediatric "stereotypic movement disorder." These respond to ECT and to the benzodiazepine lorazepam, and current evidence suggests that these, too, are forms of catatonia.

Fed up with the DSM and its contrived categories, a group of investigators led by Max Fink has been trying to get back to the basic illness entities in psychiatry as they exist in Nature. Members of this group have written about three such entities—hebephrenia, catatonia, and melancholia—which we have called elsewhere "the Three Ugly Stepsisters." The group has produced monographs about two of the stepsisters—melancholia and catatonia.[11] Here, we take on catatonia again, but this time its history. The history of catatonia demonstrates clearly that the symptoms of catatonia are among the most fundamental clinical pictures in psychiatry and, as such, deserve wide attention.

While at the University of Michigan in the first decade of the twenty-first century, Michael Alan Taylor, formerly the chairperson of psychiatry and psychology at Chicago's Rosalind Franklin University, described patients in various intensive care units of the large academic hospital as "Lazarus patients." "With lorazepam and ECT we raised them from the dead and they walked out of the hospital back to baseline. Some literally had hospice papers on their bed stands."[12] With lorazepam and ECT, the Consultation and Liaison psychiatrists were able to give these patients—many of whom were in long-term catatonic stupors—new lives.

Today, the situation is quite different: patients are being more readily diagnosed with catatonia, treated effectively, and returned well to the community. Often they do not relapse, nor do they have residual symptoms. It is a kind of miracle. This book is about that miracle.

A HISTORY OF CONFUSION

Anyone who thinks that medicine is one brick of knowledge carefully laid upon the next will despair at reading the history of catatonia. In 1873, David Skae, professor of psychiatry in Edinburgh, described a typical case of "adolescent insanity" caused,

he thought, by masturbation: "The boy was sometimes preternaturally excited, but more often dull, and sullen—he wept, and said he had committed the unpardonable sin [masturbation], and tried to tear off his clothes and throw them into the fire. Sometimes he appeared to be in a cataleptic fit, falling down and appearing to be in a trance. He had a silly and stupid look, refused to take his food, and would not answer questions when spoken to. Sometimes he would suddenly throw himself on the floor, but whether voluntarily or involuntarily could not be made out. . . . He occasionally practiced some curious movements. He would stand for more than an hour at a time working his hands backwards and forwards. . . . On other occasions he would devote as long a period to twitching the corners of his mouth up and down, and jerking his body backwards and forwards. If any one went up to him and shook him gently he immediately stopped these movements." The boy recovered within "three or four months."[13]

The patient had an array of catatonic symptoms: alternation of stupor and agitation, posturing, negativism (food refusal, refusing to answer), and stereotypical movements. Skae's article was published in the very year that Kahlbaum's classic German work appeared, 1874, yet it is unlikely that Skae would have diagnosed the patient with catatonia because the term was slow to catch on in Britain[14]

The Paris of the celebrated neurologist Jean-Martin Charcot was full of catatonia in the 1870s and '80s, although it was called "hysteria" or "catalepsy." Charcot positively cultivated it at the vast women's hospice, the Salpêtrière, where he was the medical director. Under the term "hysteria," many of the young female patients went into stupors at a signal such as the clanging of a bell. Jules Claretie, a boulevardier who frequented the hospital, reported in 1881, "The ringing of a gong, an overly bright light, or staring fixedly at an object—suffices to set off catalepsy. During a religious holiday at the Salpêtrière, someone forgot to stand down the brasses and the cymbals in the military music that accompanied the parades through the courtyards of the hospital. The music played, the cymbals resounded, and—suddenly—a whole column of patients remained in place, in catalepsy, their eyes turned upward, their limbs stiff as a statue. This won't happen again. There'll be no more parades at the Salpêtrière."

Out on the streets of Paris as well, there was catalepsy, continued Claretie. "One fine day she'll give a sudden laugh, penetrating, nervous: and a fit happens! There's a tremor, and what one calls choreiform movements appear. The woman, seized suddenly, emits a prolonged scream, holds out her arms and falls backwards, almost gently. Then, her jaw locked, her neck tensed and swollen and the sounds of digestion in her gullet [stertor]: she's fixed there, her eyes wide open, pupils dilated, looking upwards, arms rigid, spread out like a cross, literally crucified—her legs extended, next to each other, stiff. . . . There are fits that can last up to five hours."[15]

US psychiatry, by contrast, had definitely not been accustomed to thinking about catatonia, and it was simply not on the radar of many clinicians. William T. McKinney Jr., later a distinguished figure in psychosis research, was a medical student at Vanderbilt in the early 1960s. "I'll never forget my first patient in my clinical psychiatry rotation," he said in a later interview. "I was out at Central State Hospital near Nashville. The patient didn't talk and was mute; the diagnosis was catatonic schizophrenia. Frank [Luton] and Bill Orr [McKinney's teachers] taught us to respect the patient's need for distance and predictability. You're respectful, not too demanding, but just there. They had their space and you don't encroach on it." McKinney would return to the patient week after week, to see if he was ready

to talk. But he wasn't, not for a long time.[16] The anecdote is heartwarming as an expression of humanism in medicine, yet rather chilling as an illustration of inadequate care. Whether the patient had "schizophrenia" is impossible for us to know now, but he certainly had catatonia, a serious and potentially fatal disease. In the early 1960s, catatonia was eminently treatable with barbiturates and ECT, as it had been since the 1930s. So, out of respect for his "space," the patient, in the grips of a major illness, was not treated.

Today, we are much occupied with psychiatric diagnosis. Commissions appointed by the American Psychiatric Association produce the fat volumes of the DSM. In the famous third edition in 1980, the commissioners invented such diagnoses as "major depression" or "posttraumatic stress disorder (PTSD)," more or less on the spot, either to solve a political problem within the Association (as with major depression—to get approval by the psychoanalysts) or in response to an outside pressure group (as with the Vietnam veterans, who foisted PTSD upon the disease-designers in a strong-arm political play). Diagnoses tended to be someone's bright idea that then became heavily politicized and acquired lobbies on their behalf.[17]

Things used to be different in psychiatry. Diagnoses would percolate for decades before crystallizing and taking a place in the manuals. There would be a vast input from individual clinicians in many lands that would finally turn into a river of certainty as one of the principal figures, such as Emil Kraepelin or Sigmund Freud, incorporated the diagnosis in a definitive system. Delirious mania percolated in this manner in mainline psychiatry and "neurosis" within psychoanalysis. Such broad streams were the opposite of the horse trading that goes on around a DSM table: "I'll give you your diagnosis if you give me mine." Catatonia came together in one of these broad-stream stories, beginning as a wide variety of different terms—"stupidity," "catalepsy," "*Starrsucht*"—then coming together in the work of a central figure, Karl Ludwig Kahlbaum, who owned a small, private mental hospital in Görlitz, Germany, and who, in 1874, coined the term "catatonia." (Not that it was immediately accepted, anything but.)

Which system is better, the slow convergence or the sudden inspiration from heaven? Hard to say. "Hysteria" was the product of the slow convergence of many minds, and it turned out to be a disaster, especially for women. "Major depression" was the bright idea of Robert Spitzer, head of the DSM Task Force, in 1980. The takeaway lesson from this book is that we learn valuable lessons about psychiatric diagnosis from the storied past, and this has been forgotten in a psychiatry that today is highly presentist.

2

CATATONIA BEFORE KAHLBAUM

A girl of five, having been quite shocked at mealtime that her sister had taken for herself some choice morsel that the girl desired, all of a sudden became stiff. The hand holding the spoon remained in the same position stretched out towards the platter; she looked at her sister sideways, with an indignant expression; although people called out loudly to her, and shook her firmly, she understood nothing; she moved neither her mouth nor her lips; she walked when she was pushed or taken by the hand; her arms, whether they were raised, lowered or placed transversally, remained in the same position; you would have thought you were looking at a statue of wax; after this fit, she remained stiff and cold, as though of marble; after an hour she started to warm up little by little, stretching out her limbs with deep sighs. . . . Finally, after a lot of sweating, she returned to normal.[1]
—Samuel-Auguste Tissot, physician in Lausanne, mid-eighteenth century

One often encounters the statement, "Catatonia was first identified by Karl Kahlbaum in 1874."[2] The statement is technically correct but historically wrong. For centuries, physicians have described the symptoms of catatonia, although it was indeed Kahlbaum who assembled them into a neat package bearing the label "catatonia." Before Kahlbaum catatonia was called different things, and the commonest term was "catalepsy."

CATALEPSY

Catalepsy meant stupor plus waxy rigidity, the limbs holding the new position in which they had been placed. The earliest descriptions are in Hippocrates' writings, with the first careful account appearing in the work of Galen in the second century AD. Thereafter, descriptions of stiff limbs and stupor appear sporadically.[3] One scholar tabulated 150 cases in the medical literature from Galen to Madame D in 1855.[4] In Toulouse, in 1415, a monk saying Mass, on the moment of raising the chalice, "became stiff and immobile, his eyes open and turned upwards. The Brother who was serving Mass, saw that the monk remained overlong in this state, approached him, and. having shaken him several times by the robe, found him in this same immobility." There was a murmur among the congregation. Perhaps a miracle? A physician examined the stricken monk and said there had been no miracle, but that it was "a very dangerous malady and difficult to cure." Thereupon, a second monk began the continuance of the Mass, raised the chalice, and was stricken by the same symptoms! There was now a great tumult in the city. But the opinion of the physicians was that

the first monk "had been surprised at that moment by a malady that they call *caroche* or *catalepsy*, and that this had an effect on the second monk of 'fear' together with his 'wounded imagination.' "[5]

In 1737, Madame AA left her home in Vesoul to be judged in a criminal affair in Besançon, the regional capital. But a medical problem developed. The physicians who rushed to her bedside found

> the lady seated on a chair, immobile, her eyes fixed upwards and glistening, her pupils wide open and motionless, her arms and hands raised together, as if she were in ecstasy. . . . Her limbs were supple, light and let themselves be positioned wherever one wanted without resistance, but—and this is what characterized her illness—they were too obedient; they didn't leave the position in which they were placed. . . . We raised one arm, then the other; they did not drop down again. . . . We raised them so high that even the strongest man would not have been able to keep them in that position; they remained up of their own.

Samuel-Auguste Tissot, the Lausanne physician who gave us this account in 1789, went on at length about how one could reposition Madame AA's limbs at will and see them retain their new positions.[6] It was as though one were molding a candle of soft wax, hence the term "waxy flexibility." That plus stupor were the essential features of catalepsy.

In the view of Paris surgeon Ambroise Paré in 1628, "catalepsie" eventuated when "putrid vapors" rose from the uterus to affect the brain. "The body becomes rigid and cold. . . . The eyes are open, without seeing, without hearing; lethargy, apoplexy follow, and frequently death."[7] Here, we have some key elements of catalepsy as seen in the Paris of Louis XIII—the stupor in which vision and hearing are lost, the body rigid—and, indeed, death may follow in a lethal version of catalepsy discussed later.

Catalepsy may not have been frequent, but it was not infrequent either. References to it dot the medical publications of the day. In his account of "periodic illnesses" in 1764, Mannheim physician Friedrich Casimir Medicus—who actually is better known as a botanist than a medical writer—described "periodic catalepsy" (*periodischer Starrsucht*) as a "repeated obtunding of the senses occurring at certain times, with a distinctive cramping [or tonic stiffening (*Krampf*)] of the body." Several authors, he said, had described the symptom in its recurrent form. "I myself previously knew a woman who was therewith afflicted."[8]

Catalepsy was called "*Starrsucht*" in German, the patients turned into "blocks of marble" yet displaying waxy rigidity. "Persons afflicted with it," said Leon Hirschel, a physician in Berlin, in 1769, "after a previous headache become . . . rigid and immobile, like a poster-pillar, lose their awareness, see nothing even with open eyes, neither hearing nor feeling anything, but they are able to get their breath, although weakly, and the pulse is natural. . . . The limbs may be moved, but remain in the position that is given them, so that, for example, if you flex or extend an arm or a finger, it remains flexed or extended until the attack is over."[9]

François-Xavier Mezler was a court physician to the prince of Hohenzollern-Sigmaringen. One of his young patients, a pregnant woman, was subject to loss of consciousness plus muscular rigidity. "When I visited her," he reported in 1794, "I

thought she was sleeping. . . . I wanted to awaken her, but she was rigid, like wood, when I wanted to move her hand. This lasted for several more minutes and then she came to herself." She remembered only that she thought "something had climbed in her throat" (*globus hystericus*). In other attacks, Dr. Mezler reported, "It took a great deal of slow and steady pressure to move her arm, e.g. to give it another position." (This would later be known as *gegenhalten*.) Dr. Mezler apparently became a frequent caller. "I once saw, as she kissed her husband, that she got an attack; [her husband] was thus obliged to hold her in his arms until she came to her senses again."[10]

Around 1800, catalepsy was sidetracked into a near-fatal byway, as "magnetizers" associated with Franz Friedrich Anton Mesmer, first of Vienna, then of Paris, latched on to it as evidence of the action of the body's "animal electricity." This was the beginning of hypnotism—but hypnotism more as a psychodrama of suggestion rather than a medical tool. An entire hypnotic circus flourished for around 20 years, in which physicians imagined that their patients' hearing, for example, had been transferred to their abdomen or to the tips of their fingers, and "cataleptic" trances became common for highly suggestible individuals. All this had nothing to do with catalepsy as a symptom of catatonia; one of us has discussed this elsewhere, and the subject will not be reviewed here.[11] But this is why Paris psychiatrist Claude-Etienne Bourdin wrote a big book on catalepsy in 1841: to save the concept from being abducted by "the magnetizers." (Bourdin, anticipating Kahlbaum, thought catalepsy a unique disease entity.[12])

The first modern description of catalepsy comes from Philippe Pinel, the founder of psychiatric nosology. In his *Nosographie Philosophique* in 1803, Pinel defined catalepsy as "the sudden privation of sensory function and muscular movement." There may be posturing, he said, and waxy flexibility of the limbs as well. He reproached his predecessors for attaching the term "catalepsy" to all manner of conditions (this would be "symptomatic catalepsy" meaning not primary but caused by other diseases), and he argued for the existence of a single cataleptic disease. (Kahlbaum would pick up this thread.) The core symptom, said Pinel, was muscular tonic contraction, which permitted waxy flexibility to take place. But stupor, the eyes generally open, was also part of the core picture, with the patients unresponsive to external stimulation. (There was also an ecstatic catalepsy, with the patients frozen in place in religious enthusiasm.)[13] Pinel's authority sufficed to center catalepsy in the nineteenth-century pantheon of distinct diseases.

The English have always been parsimonious with diagnoses, shunning many of the fads that drifted over from the Continent, such as "hysteria." Many of the patients described by English asylum alienists early in the nineteenth century as in the grips of "insanity" or "madness" exhibited catatonic symptoms that, in France or Germany, would have been deemed catalepsy. Here is James W, 29, admitted to the New Bethlem Hospital ("Bedlam") in 1821:

> When the paroxysm came on, however he happened to be situated, his whole frame from head to foot became stiff, as if all his joints and muscles were ossified. His eyes, though staring open, became fixed, and he foamed at the mouth. If sitting or walking when his fit came on, he would instantly fall to the ground, generally extended at full length on his back, with the same symptoms of rigid stiffness and insensibility: his eyes, open and inclined upward [oculogyric crisis], were insensible to the touch of

> a hand passed over them, which did not produce the slightest wink. No symptom of animation remained, with the exception of breathing, but this so faintly as to be scarcely perceptible. His condition in all other respects resembled death; and in this state he would sometimes continue for one, two, three, and even four days. . . . On being roused from his stupor, he recollected nothing of what had passed.

In a previous admission, he had been violent and seriously injured one of the attendants.[14]

By 1856, Henry Monro, chief psychiatrist ("physician") of the St. Luke's Hospital in London, reported that the British had become more accepting of the diagnosis of catalepsy. Monro had "half-a-dozen cases" of "cataleptoid" patients on his service, including some "who stand in apparently profound sopor; their eyes are glued down or else staring open in a fixed manner, so immovable that you do not observe the least twinkle of the eyelid. . . . You speak to them, they will not answer; you offer them food, they will not eat. They indeed are most unwilling to move from the spot which they have taken up," and if "you try to cross their will then you often find a most resolute resistance." Monro's patients were negativistic, mute, posturing, and agitated.

Were they "demented"? Not at all. Monro said, in a passage that today would leave the hospital ethics committee open-mouthed, "Sometimes when you lay hold suddenly of such a patient, you may shake him out of the stupor, and you find that his mind is by no means lost; that he has a clear perception of all that has been going on even during the trance; and he will argue about it as about an incubus which he could fully appreciate but could not control." Monro thus described the range of symptoms in catalepsy that anticipates Kahlbaum. "I am in the habit of pointing them out as specimens of the cataleptoid class, and the term has, I am happy to say, gained some approval."[15] Some might feel that Monro has the priority over Kahlbaum in that the "cataleptoid class" could easily qualify as catatonia. Yet Kahlbaum described the syndrome so much more comprehensively—with movement disorders, negativism, a sequence of stages, and so forth—that his priority remains intact.

Stupor, of course, was part of catalepsy, but English clinicians seemed more comfortable with it, often using stupor to describe incoherent symptoms. John B, 26, for example, fell ill on his honeymoon, "suffer[ing] pains in his head followed by mental depression." He was admitted to the Holloway Sanatorium outside of London in September 1894, discharged six weeks later. It was not quite clear what was wrong with him. His clinicians' diagnosis: "connubial stupor."[16]

Catalepsy appeared in the New World just as in the Old. In 1839, in Philadelphia, Isaac Parrish was called to the bedside of a lad of 15, whom he found "lying upon his back, motionless, and in a state of partial insensibility. . . . The eye balls were fixed in a wild stare." He had taken fright at a great fire at the Chestnut Street Wharf several months previously, and since then, "as soon as he closed his eyes, the scene of the fire would come before him, causing him to cry out with alarm." For several days prior to calling Dr. Parrish, the boy had been "unusually despondent and sat down during the greater part of the day with his head bent toward his chest; it appeared difficult for him to look up. Now at the bedside, Dr. Parrish elicited waxy flexibility: "I succeeded on several occasions in forcing his jaws open with the handle of a spoon; this appeared to cause him great pain; and so great was the tendency to remain open, that I was obliged to assist him in closing them."