HAL
open science

Hysteria and catatonia as motor disorders in historical context

Edward Shorter

To cite this version:

Edward Shorter. Hysteria and catatonia as motor disorders in historical context. History of Psychiatry, 2006, 17 (4), pp.461-468. 10.1177/0957154X06073012 . hal-00570872

HAL Id: hal-00570872

https://hal.science/hal-00570872v1

Submitted on 1 Mar 2011

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

History of Psychiatry, 17(4): 461–468 Copyright © 2006 SAGE Publications (London, Thousand Oaks, CA and New Delhi) www.sagepublications.com
[200612] DOI: 10.1177/0957154X06073012

Hysteria and catatonia as motor disorders in historical context

EDWARD SHORTER*
University of Toronto

It is difficult to imagine motor symptoms in psychiatry as different as hysteria and catatonia. The mechanism of hysteria is presumed to be psychogenic, while catatonia has always been considered to be among the most organic syndromes in psychiatry. Yet hysteria and catatonia have historically been regarded as allied conditions, an observation borne out by recent developments in neuroscience as well as by a growing awareness that the presentation of both conditions has changed over the years. In hysteria, the main shift has been from motor symptoms to sensory complaints such as chronic fatigue; in catatonia, the major change has been the virtual disappearance of negativistic or oppositional behaviour. It is possible that catatonia as well as hysteria may be responsive to changing cultural norms.

Keywords: *catatonia; cultural influences; hysteria; motor symptoms; negativism*

It would be difficult to conceive of motor symptoms in psychiatry as different as hysteria and catatonia. In hysteria, the mechanism is presumed to be psychogenic. The mind, for reasons of its own, conceives a deficit; this is transformed into a physical incapacity such as a hysterical paralysis, which the patient perceives as organic in nature and for which the patient seeks medical help. This is a good working definition of hysterical illness, although it is hard to improve upon Joseph Babinski's (1901) formulation that hysteria is any illness that could be induced by suggestion and abolished by persuasion. Thus hysteria was really the work of the suggestible imagination.

Catatonia, by contrast, has always been seen as organic as measles. Karl Kahlbaum, who gave us the first modern description of the disease in 1874, believed catatonia to progress downhill into dementia, by definition as an organic process. And such recent observers as Max Fink and Michael Taylor,

* *Address for correspondence:* Faculty of Medicine, University of Toronto, 88 College Street, Room 207, Toronto, Canada M5G 1L4. Email: history.medicine@utoronto.ca

Edward Shorter 20/10/06 10:54 Page 2

in a monograph proposing the revival of catatonia as an independent illness entity, speak of it as 'motor system dysfunction' involving brain pathophysiology perhaps situated in the circuitry linking the anterior frontal lobe to the basal ganglia (Fink and Taylor, 2003: 33, 181).

Yet it is possible that catatonia and hysteria have more in common than has conventionally been assumed. Hysteria can result in profound physical deficits, and an epoch such as ours that seeks insight increasingly from the neurosciences finds it unlikely that such deficits are the product of a few idle thoughts alone, without accompanying underlying brain changes. The adage stands unimpaired that 'There is no twisted thought without a twisted molecule.'

Similarly, there must be some mental processing in catatonia. The mind inevitably intervenes between neurons and behaviour, and the editing of neural impulses surely informs neurological illness to some extent, though not perhaps to the same extent as psychiatric. Thus, even catatonic patients must pick and choose among which underlying neural impulses they decide to transform into behaviour.

This formulation will arouse the ire of some, I realize, for it does invoke an increasingly outdated mind-body distinction. Many psychiatrists argue that the notion of mind without brain is erroneous. As one critic states,

> There is no mind. It is a metaphor that is now dated. The 'mind' is just those brain functions of which we are aware. When people refer to their mind they mean feelings and emotion, internal language, memory, thinking. There is a neuroscience for all that. (Taylor, 2006)

Yes, and yes but. A neuroscience model of higher cognitive functioning must somehow deal with the phenomenon of suggestion. When we contemplate changes in the underlying brain processes that supposedly produce hysteria and catatonia, we must allow in some manner for the influence of suggestion. When symptoms change historically, it is doubtlessly owing to the suggestion that cultural change creates. Thus, suggestion influences behaviour – and either we must find the 'suggestion centre' in the brain, or we must allow that culture matters.

This issue of historical change in supposedly organic symptoms places the history of hysteria and catatonia front and centre. There is in mental medicine a tradition of seeing hysteria and catatonia as allied conditions, though few investigators are mindful of this today. As French psychiatrist Henri Baruk, chief of the Charenton mental hospital, said in 1970 about the relationship of catatonia and hysteria: in both there is some kind of

> disturbed personality, that is to say, no longer acting of free will but acting under the influence of a pathological force. … These are the characteristics that distinguish psychiatric disorders from organic and neurological disorders, and it is why the explanations of catatonia, as those of hysteria, have oscillated between two opposing poles: on the one hand, between the tendency to neurologize psychiatry and to reduce these two types of

Edward Shorter 20/10/06 10:54 Page 3

> disorders to a probably functional lesion of the brain centres. … On the other hand, [there is] the psychogenic tendency, which consists of explaining every-thing by psychological motives and to treat illness as a kind of simulation. Each of these two tendencies is in error …' (Baruk, 1970: 1719).[1]

After Baruk, the kinship between the two disorders tended to be forgotten. Yet two recent developments have brought the related nature of hysteria and catatonia onto the table again. One is the budding neuroscience of hysteria. Sean Spence and coworkers at Cornell Medical Center in New York and at Queen Square in London found that patients with hysterical motor deficits on the left side exhibited hypofunction in the left anterior prefrontal cortex, and feigners on the right (Spence, Crimlisk, Cope, Ron and Grasby, 2000). Matthew Broome (2004) has recently summarized research into motor hysteria, pointing to hypotheses that indict deficits in the formation of the intention to move, or the preparation of that intention before the intention is actually executed as movement.

The other development is the budding awareness that not only does hysteria change historically, but that catatonia seems to change as well. Measles does not change historically, at least as regards the pathophysiology of viral illness. For catatonia, the most presumably organic of all psychiatric syndromes, to change significantly its presentation over the years means that a good deal more mental processing of its symptoms is occurring than we have conventionally assumed.

In a previous work (Shorter, 1992) I have dealt at length with probable changes in the presentation of hysteria over the years, and here the question may be quickly reviewed. Basically, before the last decades of the nineteenth century, hysteria seems to have had a formidable motor component, phantom paralyses magicked away by the saints, and conjured away by the hypnotherapists of the nineteenth century being familiar territory for medical historians.

It goes without saying that even today we know hysteria has a significant motor component in the form of neurologically unexplained symptoms – tremors, dyskinesias, and paralyses of all kinds. Yet the dramatic forms of motor hysteria of the past seem unusual nowadays. It is really beyond the compass of quotidian medical experience today for young women to wake up unable to move their legs after receiving bad news the previous evening. It does happen, to be sure, but it is an object of such medical curiosity as to bring the entire department flocking for a view after the patient is admitted. Similarly, the 'sofa cases' of yore – upper-middle-class women who would 'take to their beds,' as the saying went, and remain 'shut-ins' for decades – are now unfamiliar except for those with major psychiatric diagnoses such as chronic psychosis. The cloistered world of chronic fatigue syndrome is a bit of an exception to this generalization. Yet even they do not take to their beds in the belief that they are paralysed but remain mobile enough at least to attend meetings of their chronic fatigue support groups.

Edward Shorter 20/10/06 10:54 Page 4

In hysteria, the main historic change seems to be a shift of symptoms from the motor side of the central nervous system to the sensory side, in the form of such symptoms as chronic fatigue and headache. The reasons for this change are complex, but doubtlessly include the introduction in 1896 of the reflex named after Joseph Babinski (that made it possible to 'disprove' a hysterical paralysis); there was also a new social style for women after World War I that elevated the energetic, loosely clad, tennis-playing young 'flapper' and sent the corset-gusseted Victorian 'lady' into exile. The dynamic young women of the 1920s did not develop the florid hysterical paralyses of the past. It was incompatible with their image.

A caveat: this hypothesized transition in the symptoms of hysteria has never been validated with solid epidemiological data. But clinical evidence of the frequency of hysterical paralyses in the nineteenth century is overwhelming, and may not simply be ignored. And such functional sensory complaints as fatigue and chronic pain did not loom large in the daily experience of nineteenth-century physicians, who had to cope with an avalanche of infective illness. It is possible that the claimed transition is an artefact of ascertainment: what patients do not present to the doctor will not be medically reported; what the doctor does not see as clinically significant will likewise be ignored. Yet at the moment, the evidence on behalf of the symptom transition in functional illness seems pretty sound.

Catatonia, by contrast, is a quite different kettle of fish. No observer, of whom I am aware, has ever claimed that the symptoms of catatonia have changed significantly. Yet there does seem to have been one major shift: catatonic negativism has been vastly reduced. When Kahlbaum introduced the diagnosis of catatonia in 1874 he said, 'What is notable about the symptoms in the sphere of activity and volition is an early tendency to negation.' He commented on the 'prominence of negative mental declarations and negative behavioural traits.' 'There is probably no patient', he said, 'in whom this symptom of the tendency to negation is not present in some manner' (Kahlbaum, 1874: 46–7).

In his *Allgemeine Psychopathologie* in 1913, Karl Jaspers at Heidelberg placed this 'negativism' in the context of the larger symptom picture of catatonia. The 'catatonic complex of symptoms', as Jaspers called it, included: (1) either stupor or excitement; (2) either verbigeration in speaking and writing, stereotypies and mannered movements, or bizarre fixed positions such as Schnauzkrampf; (3) either negativism or automatic responses to commands (Jaspers, 1913: 281). Thus not all patients exhibited negativism, meaning oppositional behaviour; some answered instantly to orders and suggestions (some also switched back and forth between the two responses). Yet for Jaspers, negativism in the sense of doing the opposite of what one was told was part of a fixed behavioural layer in the syndrome of catatonia.

What did this kind of negativistic behaviour consist of? Emil Kraepelin, in the fifth edition of his famous textbook, gave quite an expansive characterization of

Edward Shorter 20/10/06 10:54 Page 5

Negativismus, consisting of mutism, the patients being 'unresponsive to every external intervention'. 'Any effort to alter the position or movement of the patient encounters … resistance.' 'Their muscles tense up when you try to move them. The patients give contrary responses to orders, and often do the opposite of what they are told. They often refuse food, pressing their lips together at the spoon's approach.' Some even refuse to walk, 'letting themselves topple stiffly over as soon as you get them upright. In other cases they march with rigid knees, on the tips of their toes, on the foot's lateral margin, with legs spread apart, the upper body starkly bent over …' (Kraepelin, 1896: 444–6). Some of this behaviour could be redistributed to other catatonic symptoms such as mannerism. Yet for Kraepelin the essential point was the patients' resistance to correction. Karl Kleist later referred to passive and active resistance of the muscles to movement, not just in catatonia but in a number of focal neurological lesions, as 'Gegenhalten' (Kleist, 1927).

Also part of their negativism, Kraepelin said, was their unhygienic toilet behaviour. 'They retain urine and faeces, often for a long time, and then suddenly let go without taking the slighest notice of where they might be in order to avoid unpleasant consequences' (Kraepelin, 1896: 444). This lament about catatonics, who would retain faeces for long periods and then suddenly discharge their bowels, was common.

Yet such comments on contrarian behaviour in catatonia, especially on disagreeable behaviour with excrement, were not confined to Kraepelinians. Among catatonia specialists, few were as sceptical of Kraepelin's grand constructs as Warsaw psychiatrist Maurycy Urstein. Yet Urstein's catatonic patients could well have been Kraepelin's. Urstein (1912: 636) wrote:

> Of diagnostic interest [in catatonia] is above all the general tendency to negation. A comment on the pleasing appearance of the female patient has the result that she immediately begins refusing food. Although the patients wish a restoration of health, they are fearful of being considered recovered and militate for sick status. … Similarly the catatonic patient manages to achieve, even given otherwise orderly external appearances, the evacuation of urine and faeces in his clothes …

Thus, negativism in these strikingly contrarian forms arrested the attention of the founding students of psychopathology. In his 1916 textbook, Eugen Bleuler, Professor of Psychiatry in Zurich, considered negativism 'a very common and very unpleasant symptom of schizophrenia'. 'If the patients are supposed to get up, they want to stay in bed; if they are supposed to stay in bed, they want to get up. In response to orders or according to the routines of the institution they neither want to get dressed, nor to get undressed, nor to come to dinner, nor leave the table.' In catatonic negativism the patients did, in other words, the exact opposite of what they were told. 'In some cases you can reliably get the patients to perform the desired activity if you forbid it or order them to do the opposite.' (Bleuler, 1916: 305–6).

Edward Shorter 20/10/06 10:54 Page 6

How common was negativism? In Johannes Lange's catalogue of catatonic symptoms, among 100 patients with manic-depressive illness, *Negativismus* occurred 13 times (Lange, 1922: 10). The commonest catatonic symptom in this group was mannerism (in 37 of the 100 patients). Thus *Negativismus* was about a third as frequent as manneristic behaviour. (By contrast, only 5 of the 100 had catalepsy.)

Negativism began relatively early to be seen more as a neurological than as a behavioural symptom. In 1900 Breslau neurologist Carl Wernicke rebaptized Kahlbaum's catatonia 'akinetic motility psychosis' (or, alternatively, 'akinetic and hyperkinetic cyclical motility psychosis'). Wernicke (1906: 395–424) limited the negativism component of the syndrome to *active* opposition to changes in muscle position, so that the patients, for example, would strenuously resist clinicians' efforts to open their eyelids or widen their jaws for purposes of feeding.

The *passive* act of oppositional hypertonus, or counterpull, of catatonia became widely noted only after World War I, as we have seen above. But this involuntary phenomenon of passive resistance to movement did indeed become part of the symptom picture of negativism. In his 1927 textbook Johannes Lange mentioned 'Gegenhalten'. 'In Gegenhalten we find a group of disorders in which psychological explanations fail completely. Here the issues are neurological in nature.' Lange commented on the frequency of Gegenhalten in organic brain patients, a phenomenon that 'obliges us to look at psychotic negativisms from this aspect' (Lange, 1927: 534).

Even as late as World War II, Kleist's student Karl Leonhard found negativism common in catatonia. This kind of contrary behaviour, he said, seemed completely involuntary. Even if you were specially nice to the patients, they would still behave negativistically, but with something of an embarrassed smile. There is 'a willingness predominantly in the smile, but an unwillingness in behaviour' (Leonhard, 1942: 120). Thus negativism did not come from negative affect; it was driven by deeper neural sources.

After World War II, negativism in the sense of contrary behaviour seems to disappear from catatonia, at least from careful clinical descriptions of it. To be sure, Willy Meyer-Gross's 1954 textbook does comment on negativism in 'catatonic schizophrenia', with particular emphasis on the patients' soiling of their beds and linen (Mayer-Gross, Slater and Roth, 1954: 248). Yet Mayer-Gross's clinical experience drew heavily upon his Heidelberg days, and the passage does not necessarily represent the reality of clinical life in British mental hospitals in the 1950s.

The term 'negativism' has, of course, been conserved. But it has become the object of a classical historical switch. Used mainly today in schizophrenia, it has come to mean affective flattening, quasi mutism and avolitional apathy (Peralta and Cuesta, 2001*a*). It no longer refers to what the classic authorities called negativism, or *der Negativismus*, which means doing the opposite of what one is asked and deliberately making oneself a nuisance

Edward Shorter 20/10/06 10:54 Page 7

(through such behaviour as refusing to eat and then stealing one's neighbour's food). Some authorities, do use 'oppositionism' for what the heritage texts termed negativism, but neither the term oppositionism nor classical oppositional behaviour is very common today: 14% in a sample of catatonic patients in Spain, for example (Peralta and Cuesta, 2001*b*). One rating scale of catatonia buries oppositionism (active and passive *Negativismus*) together with 14 other 'behavioural' characteristics (Northoff, Koch, Wenke, Eckert, *et al.*, 1999). When in 2003 Max Fink and Michael Taylor brought out their overview of catatonia, they did mention negativism, but mainly in the context of Gegenhalten (the example of behavioural negativism they offered was from 1815) (Fink and Taylor, 2003: 2–3, 20).

What have we learned from this exercise in comparing hysteria and catatonia, aside from the need not to be overly dogmatic in juxtaposing 'psychogenic' and 'somatogenic'? It is no surprise when such phenomena of suggestion as hysteria change over the years, in response to changed cultural circumstances. But it is a bit eye-opening to see syndromes with a profound organic basis such as catatonia changing as well. Could catatonia respond to changed circumstances? And if so, what might some of these changes be?

One must distinguish between 'internal' and 'external' changes. Internal changes refer to the medical management of catatonia and its vicissitudes over the years. Thomas Ban (2005), a veteran psychopharmacologist, calls attention to the introduction of the neuroleptics. 'At the time I started psychiatry [1950s], catatonic manifestations were blatantly present. But by the time I did my research in catatonia in the early 1980s, the symptoms had to be elicited because they were decreased in intensity by neuroleptics.' Ban thought the introduction of the barbiturates might have initiated a similar change in hysteria.

Yet the mind responds as well to external changes in culture and society, not just to shifts in medical management. Within catatonia, negativism is interesting precisely because it involves behaviour, some of it neurally driven, some mentally driven. Negativism might have made sense to patients in the context of the authoritarian mental hospitals of the nineteenth century, where the patients were given 'orders'. Negativism then perhaps represented an illness-driven act of defiance. Yet in the context of post-World War II psychiatry – with reformed institutions managed by patient-friendly teams where discussions replaced commands – negativism no longer made sense. On open-door wards run by agreeable nurses, defiance might have seemed inappropriate, just as defecating in bed was, somehow, no longer on. We do not expect measles to be culturally edited, but catatonia may be different from measles.

Acknowledgements

For comments on an earlier version, the author would like to thank Thomas Ban, Tom Bolwig, Max Fink, Jon Stone and Michael Alan Taylor.

Edward Shorter 20/10/06 10:54 Page 8

Note

1. All translations are by the author.

References

Babinski, J. (1901) Définition de l'hystérie. *Revue neurologique*, 9, 1074–80.

Ban, T. (2005) Personal communication (28 Dec.).

Baruk, H. (1970) La catatonie de Kahlbaum, la schizophrénie et la révision de la nosographie psychiatrique. *Semaine des hôpitaux de Paris*, 46, 1679–729.

Bleuler, E. (1916) *Lehrbuch der Psychiatrie* (Berlin: Springer).

Broome, M. R. (2004) A neuroscience of hysteria? *Current Opinion in Psychiatry*, 17, 465–9.

Fink, M. and Taylor, M. A. (2003) *Catatonia: A Clinician's Guide to Diagnosis and Treatment* (New York: Cambridge University Press).

Jaspers, K. (1913) *Allgemeine Psychopathologie* (Berlin: Springer).

Kahlbaum, K. (1874) *Die Katatonie oder das Spannungsirresein: Eine klinische Form psychischer Krankheit* (Berlin: Hirschwald).

Kleist, K. (1927) Gegenhalten (motorischer Negativismus), Zwangsgreifen und Thalamus opticus. *Zeitschrift für Psychiatrie und Neurologie*, 65, 317–96.

Kraepelin, E. (1896) *Psychiatrie*, 5th edn (Leipzig: Barth).

Lange, J. (1922) *Katatonische Erscheinungen im Rahmen manischer Erkrankungen* (Berlin: Springer).

Lange, J. (1927) *Allgemeine Psychiatrie*, Vol.1 (Leipzig: Barth); this was considered part of the 9th completely revised edition of Kraepelin's *Psychiatrie*.

Leonhard, K. (1942) Zur Unterteilung und Erbbiologie der Schizophrenien, Iste Mitteilung: Die 'typischen' Unterformen der Katatonie. *Allgemeine Zeitschrift für Psychiatrie*. Reprinted in Karl Leonhard (ed.), *Internationale Wernicke-Kleist-Leonhard Gesellschaft: Das wissenschaftliche Werk*, Vol. 1 (Berlin: Verlag Gesundheit, 1991), 313–24.

Mayer-Gross, W., Slater, E. and Roth, M. (1954) *Clinical Psychiatry* (London: Cassell).

Northoff, G., Koch, A., Wenke, J., Eckert, J., Böker, H., Pflug, B. and Bogerts, B. (1999) Catatonia as a psychomotor syndrome: a rating scale and extrapyramidal motor symptoms. *Movement Disorders*, 14, 404–16.

Peralta, V. and Cuesta, M. J. (2001*a*) Motor features in psychotic disorders. I: Factor structure and clinical correlates. *Schizophrenia Research*, 47, 107–16.

Peralta, V. and Cuesta, M. J. (2001*b*) Motor features in psychotic disorders. II: Development of diagnostic criteria for catatonia. *Schizophrenia Research*, 47, 117–26.

Shorter, E. (1992) *From Paralysis to Fatigue: A History of Psychosomatic Illness in the Modern Era* (New York: Free Press).

Spence, S. A., Crimlisk, H. L., Cope, H., Ron, M. A., Grasby, P. M. (2000) Discrete neurophysiological correlates in prefrontal cortex during hysterical and feigned disorder of movement. *Lancet*, 355, 1243–4.

Taylor, M. A. (2006) Personal communication (3 Jan.).

Urstein, M. (1912) *Manisch-Depressives und periodisches Irresein als Erscheinungsform der Katatonie* (Berlin: Urban & Schwarzenberg).

Wernicke, C. (1900) *Grundriss der Psychiatrie* (Leipzig: Thieme); 2nd edn published 1906.