Section 1 **Clinical issues**

Chapter 1

Introduction to the psychiatry of conversion disorders

Fred Ovsiew

Here is a description of a psychogenic movement disorder from the clinical literature.

> A 34-year-old woman fell down a flight of stairs some two weeks prior to the reported examination. No serious injury was evident. However, immediately thereafter she developed dystonia of the left lower extremity. The limb was described as rigid with no voluntary movement; passive movement was equally impossible. The thigh and lower leg were held straight out and the foot was plantar flexed at the ankle, so that the three segments of the extremity were held in a straight line "like a rigid bar." The extremity was internally rotated so that the patella and foot pointed inward. Additionally present was a left hemianesthesia involving limbs, trunk, and face.
>
> This patient had a history of epilepsy, with nocturnal convulsions featuring incontinence and biting of the tongue occurring up to weekly and showing a catamenial predominance. In addition, she had a history of psychogenic nonepileptic seizures, which had been almost completely in remission for about 5 years. Frequently, the nonepileptic seizures had been followed by a right lower extremity dystonia and right hemianesthesia lasting weeks. Nothing was reported regarding her mental or social state beyond her living in a residential facility.

What should we call this disorder, the condition in which factors other than organic disease of the nervous system lead to symptoms that mimic those produced by nervous system disease? Many terms have been used, and each new proposal has been superseded, perhaps because the proposals for new names rested on "the, surely vain, hope that old confusions were but word deep" (Porter, 1993 [1], p. 230). The term "psychogenic," used widely and in the title of this book, was reviewed by Lewis, who found its philosophical underpinnings confused and referred to the "shimmering, unfocused quality" that made it "speciously attractive." He thought it should be "given a decent burial" (Lewis, 1972 [2], p. 214). The current official term is conversion disorder (CD), which derives from a now-rejected early Freudian theory about "conversion" of affect into somatic form. The framers of DSM-III preferred that term to its older and more capacious rival "hysteria" [3]. The concept of hysteria was deliberately "split asunder" [4] in the formulation of DSM-III, with the somatic symptoms placed into the somatoform disorders category and the mental elements considered as aspects of personality disorders (notably histrionic personality disorder) or dissociative disorders. More recently, symptoms such as those shown by the patient described above have been called "functional" or "medically unexplained."

Whatever term we use, patients such as the one described are still with us, widespread psychiatric opinion to the contrary notwithstanding. They are often complicated and difficult patients both diagnostically and therapeutically, and an improved understanding of the basis of their psychopathology and of its manifestations will be welcome. They are difficult diagnostically partly because confident distinction of CD from the organic conditions it mimics is required. Fortunately, the differential diagnosis can be adequately made, as Stone and his colleagues showed and as is discussed elsewhere in this volume [5].

The patients are diagnostically complicated as well because they often have other psychopathology. For example, Lieb and colleagues showed that adolescents and young adults with CD were far more likely than their peers to have a variety of psychiatric disorders (see Table 1.1) [6]. Patients with CD are also more likely than comparison subjects to have dissociative symptoms or to meet criteria for a dissociative disorder [7]. The disproportions are so marked that we may wonder whether "splitting asunder" the category of hysteria is genuinely cutting nature at the joints. Perhaps the

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Table 1.1 Odds ratios for additional diagnoses in patients with conversio disorder in a population sample of adolescents and young adults

Disorder	Odds ratio (all significant)
Substance dependence	8.19
Dysthymia	11.69
Generalized anxiety disorder	11.81
Any eating disorder	29.44
Any psychiatric disorder	5.23
Two or more disorders	6.48

Source: adapted from Lieb *et al*., 2000 [6].

patients can be understood properly only by taking other psychopathology into account.

Patients presenting with a conversion symptom are likely to have other non-organic somatic symptoms. Followed over the long-term, patients with CD are likely to show multiple medically unexplained symptoms in multiple organ systems [8]. Clinicians must particularly keep this in mind because of the low accuracy of the history provided by the patient at the point in the course when a given clinician happens to see the patient. Schrag *et al*. compared the accounts of patients with non-organic neurological symptoms with the records of their primary-care physicians. Only 22% of self-reported diagnoses were confirmed [9]. Simon *et al*. found that 61% of medically unexplained symptoms and 43% of all symptoms reported at a first interview were not reported on inventories of "lifetime" symptoms on a second interview one year later [10]. Consequently, a comprehensive view, over time and with adequate medical records, may show that patients with CD make more extensive use of somatization than is initially apparent.

Two other non-organic conditions need to be considered in the differential diagnosis of CD: factitious disorder (FD) and malingering. These two diagnostic groups comprise patients whose non-organic symptoms are voluntarily produced, in contrast to the definitional involuntary nature of symptom production in CD. By definition, in FD the goal of producing these symptoms is the assumption of the sick role; in malingering, by contrast, although the symptoms are equally under voluntary control, the goal of their display is an external incentive, such as monetary gain. Although it is likely that these three domains – CD (with involuntary production of symptoms), FD (with voluntary production of symptoms for the purpose of assuming the sick role), and malingering (with voluntary production of symptoms for an external, practical goal) – broadly capture separable phenomena, it might well be doubted that the dividing lines between them are bright and that doctors are reliably capable of recognizing what is going on in patients' minds. The varieties of deception may form a continuum, on which the deceptions seen in patients with malingering and FD lie at various positions in regard to the degree and nature of self-deception involved [11]. The self-deception central to CD can be particularly puzzling: how could they possibly *not* know what they're doing, we may wonder. Symonds [12] (p. 408) quoted the London psychiatrist Birley as posing the issue in this way: the self-deception of the hysteric is a particular kind of mental deficiency.

Kanaan and Wessely reviewed neurological presentations of FD [13]. They suggested that neurologists' diagnosis of FD and perhaps patients' choice of which symptoms to manufacture are affected by the available border with CD. Patients may avoid production of symptoms that could be diagnosed not only as genuine neurological disease, with the attendant gain of sympathy and care, but also as hysteria, with its attendant opprobrium. Doctors, by comparison, perhaps partly for fear of litigation, are especially reluctant to diagnose deliberate production of symptoms in the absence of observational proof (such as seeing the patient heat the thermometer to produce factitious fever) when the diagnosis of CD is easily at hand as an alternative.

The distinction of malingering from unconscious simulation or from conscious simulation for purposes other than practical gain is often problematic. Observational proof that neurological impairment is malingered – for example from video recordings of behavior inconsistent with the claimed impairment – is usually unavailable and is obtained by lawyers, almost never by doctors [14]. Kaanan *et al*. showed that neurologists in the UK, while aware of the distinction between malingering and CD, do not consider it within their purview to make this distinction [15]. A large neuropsychological literature has grown up on identifying exaggerated cognitive impairment. The combination of findings inconsistent with brain disease and the presence of an external incentive is recognized to be common, but some neuropsychologists doubt that they can objectively attribute the excessive impairment to an internal state that is hard to assess [16]. As a "best

practices" guideline points out (p. 136), "Although symptom validity tests are commonly referred to as malingering tests, malingering is just one possible cause of invalid performance" [17]. While other clinicians may feel that it is, therefore, up to the psychiatrist to discern the patient's private intentions and secret goals, psychiatrists too lack telepathic powers.

For everyday social interaction, we all confidently believe we can usually distinguish between voluntary actions and involuntary ones, by ourselves or others, and we believe we can usually infer others' goals from their actions or their statements. Social life would be impossible without fair accuracy in these respects. But how well can we do in pathological cases at recognizing whether symptom creation is "deliberate" or "voluntary," or has one thing or another as its goal? Indeed how well do concepts such as deliberateness or voluntariness or goal, comfortably used in ordinary language under ordinary circumstances, capture experience in pathological circumstances or in unconscious mentation?

The diagnosis of CD under DSM-IV criteria requires the identification of psychological stressors deemed to be responsible for the symptoms [18]. This identification, the criteria say, is confirmed by the temporal sequence, symptoms following stressor. Some psychiatrists insist that the diagnosis of CD should be made only on the basis of "positive psychiatric findings," such as *la belle indifférence* or the presence of a symbolic meaning for the symptom [19]. Can these modes of inference genuinely validate the assignment of symptoms to a conversion reaction caused by a particular psychological stressor? Can clinicians reliably and validly confirm that a patient meets this diagnostic criterion?

Few data support an affirmative answer to these questions [20,21]. Perusal of case reports of incorrect diagnoses of CD suggests that undue diagnostic reliance on presumed psychological stressors is a frequent cause of error [22]. Stress, like meaning, is simply too easy to find, irrespective of the medical diagnosis. Here is an example of the appeal of psychodynamic explanations.

> Mrs. N., a woman of about 40, was left to raise two daughters after her husband died of a stroke early in the marriage. Deaths of family members ran through her history: of her 13 sibs, only four were still alive; she had memories from ages seven and nine of seeing her sister and then her aunt in their coffins. She had symptoms of anxiety and depression, and she suffered phobias and vivid pseudohallucinations. It was possible in a detailed way to trace these symptoms to her experiences.
>
> She had motor symptoms as well. These included "spastic interruptions" of her speech, "ceaseless agitation" of her fingers, and "frequent convulsive *tic*-like movements of her face and the muscles of her neck, during which some of them, especially the right sterno-cleido-mastoid, stood out prominently. Furthermore, she frequently interrupted her remarks by producing a curious 'clacking' sound from her mouth."

Many clinicians today would be likely to consider that Mrs. N. had Gilles de la Tourette syndrome. As it happens, the clinician who described this case knew Gilles de la Tourette well and surely was familiar with the syndrome he described [23]. The point here is not that Sigmund Freud – for it was he who described the case of Frau Emmy von N. ([24] (quotations from p. 49)– misdiagnosed a case of Tourette syndrome as hysteria. The boundaries between the two have shifted over time, and Freud's diagnostic thinking was appropriate to his time [23]. The point is that psychodynamic explanations – that is, explanations of symptoms in terms of their meanings to patients based on the patients' life experiences – can be magnetically attractive, irrespective of diagnosis [25]. If the diagnosis is indeed of symptom-causation by such psychological mechanisms, then recognition of the meanings of the symptom to the patient forms the core of psychological treatment. A necessary servant, but a treacherous master, is psychodynamic thinking.

Patients themselves may use psychological explanations to deny medical illness [26]. I vividly recall the patient who at the point of admission for workup of dysphagia explained to me how the symptom arose from the stresses in his life. He had motor neuron disease.

Non-physiological neurological signs, such as give-way weakness or asymmetric vibratory sensation on the sternum, also can be misleading [27,28]. The exception is when the non-physiological finding directly reflects the abnormality that is the patient's complaint. For example, when the Hoover sign is present in a patient who complains of leg weakness, the complaint is shown to be non-physiological. Even here, false positives can occur because of limitation of effort by pain or because of a mixture of organic and non-organic weakness [29]. But when a patient complaining of abnormal movements shows non-physiological sensory abnormalities on examination, all that has been demonstrated is the patient's suggestibility. Suggestibility is widespread in

the normal population and lacks diagnostic validity as a marker of CD.

The author of the case reported at the beginning of this chapter used the term "hysteria," but he knew no better because he was writing at the dawn of the modern age of the study of hysteria. In fact, he can lay claim to having initiated the modern age. Jean-Martin Charcot, who provided the case description [30] (p. 35) that was recast here, inherited a diagnostic category with carefully catalogued symptoms but no satisfactorily understood pathophysiology. Charcot believed that the disorder arose from a hereditary disturbance of the functional state of the nervous system, what he referred to as a "dynamic lesion." As the historian Toby Gelfand put it, "He believed that he had captured hysteria for the specialty of neurology" [31]. (How dismayed Charcot would be that his *Clinical Lectures on Diseases of the Nervous System* was reissued in the series of Tavistock Classics in the History of Psychiatry! [30].) Although Charcot (p. 210 [30]) insisted that he believed that "the psychic element plays a very important part in most of the cases" of hysteria, perusal of his case descriptions yields the impression that he interested himself but little in the emotional or mental state of his hysterical patients. Indeed, he appears to have had little conversation with them, as this description [32] of a clinical examination suggests:

> He sits down at a bare table and at once calls for the patient. The intern reads the history while the master listens attentively. Then, there is a long silence during which he looks and looks at the patient while drumming his fingers on the table… All the while, Charcot says nothing. Finally, he orders the patient to make a special movement, makes him talk, asks for his reflexes to be tested, his sensory system to be explored. And again a mysterious silence.

Freud, Charcot's acolyte from the time of his visit to the Salpêtrière in 1885, took the crucial steps of abandoning the search for a brain lesion and instead listening to the patient: no more the "mysterious silence" between doctor and hysterical patient. These steps played a large role in determining the future shape of the specialty of psychiatry, arguably not entirely for the better. However, what we have learned subsequently about hysteria, notwithstanding new information about the brain state that corresponds to it [33], has put us on a path toward a developmentally based, psychological understanding of somatization in relation to trauma, dissociation, and self-awareness [34–38]. These advances confirm that Freud's hesitant steps in this direction were necessary for the understanding of patients with non-organic symptoms [24] (pp. 160–161):

> Like other neuropathologists, I was trained to employ local diagnoses and electro-prognosis, and it still strikes me myself as strange that the case histories I write should read like short stories and that, as one might say, they lack the serious stamp of science… The fact is that local diagnosis and electrical reactions lead nowhere in the study of hysteria, whereas a detailed description of mental processes…enables me…to obtain at least some kind of insight into the course of that affection.

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Section 1 **Clinical issues**

Chapter 2

Phenomenology of psychogenic movement disorders

Anthony E. Lang

This brief chapter will highlight the phenomenology of psychogenic movement disorders and will serve as a springboard for the review of the accompanying DVD. There are a large number of reviews describing the clinical aspects of psychogenic movement disorders including their phenomenological features (e.g., [1–5]). Psychogenic movement disorders largely overlap with conversion disorders (see Chapter 1 for diagnostic criteria) although there are differences, including the less common possibility of malingering and factitious disorders causing psychogenic movement disorders. Psychogenic movement disorders are common in neurological practice and particularly in subspecialty clinics. One of the earliest surveys by Factor *et al.* estimated that these patients accounted for 3.3% of consecutive movement disorder cases seen over a 71-month period [6]. It has been the experience of many movement disorder specialists working in tertiary referral clinics that the prevalence of these disorders is greater in recent years, presumably because of better recognition.

Psychogenic counterparts may be seen for all types of movement disorder. Table 2.1 provides an estimate of the relative frequencies of the different types of psychogenic movement disorder phenotype. Tremor and dystonia are the commonest phenotypes. Probably one of the least common of these is typical chorea; indeed, this author has only seen one or two such cases over the past 25 years. One patient with strong family history of Huntington's disease and psychogenic chorea was recently described [8]. Surveys of movement disorder clinics provide quite variable figures for the relative frequencies of the different types of psychogenic movement disorder, in part related to differing ascertainment methods (some emphasizing only the dominant movement disorder and others all movement disorder types seen in an individual patient) or referral bias (some clinics emphasizing dystonia or parkinsonism).

Table 2.1 Relative frequencies of psychogenic movement disorder phenotypes

Psychogenic movement disorder	Approximate percentages (range)[a]
Tremor	40 (14–56)
Dystonia	31 (24–54)
Myoclonus	13 (0–19)
Gait disorder	10 (0–50)
Parkinsonism	5 (0–12)
Tics	2 (0–7)
Other	5 (0.4–30)

[a] Methods of classification and designation varied from center to center contributing to these figures (e.g., listing only the dominant movement disorder versus all movement disorders). Referral bias also plays a role in some centers' classifications.
Source: Lang 2006 [7].

The classification of psychogenic movement disorders has generally followed the original approach described by Fahn and Williams in their initial report on psychogenic dystonia [9]. There have been a small number of further attempts to provide newer diagnostic criteria. For example, Shill and Gerber developed a scheme involving primary and secondary criteria [10]; however, the application of these is problematic [11]. Recently a revision of the Fahn/Williams criteria has been proposed, emphasizing the ability to establish a diagnosis based on the presence of definitive clinical features alone ("clinically established minus other features") and the importance of adding a category of "laboratory supported definite" [12]. Table 2.2

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Table 2.2 Diagnostic classification of psychogenic movement disorders

	Traditional classification of degrees of certainty in diagnosis[a]	**Proposed revision of classification of degrees of certainty in diagnosis**[b]
1. Documented	Remittance with suggestion, physiotherapy, psychotherapy, placebos, "while unobserved"	As in original
2. Clinically established	Inconsistent over time/Incongruent with clinical condition plus other manifestations: other "false" signs, multiple somatizations, obvious psychiatric disturbance	(a) Clinically established plus other features, as in original (b) Clinically established minus other features: unequivocal clinical features incompatible with organic disease with no psychiatric problems
Clinically definite	Documented and clinically established as above [13]	Documented and clinically established as above (2a + 2b)
Laboratory-supported definite	–	Electrophysiological evidence proving a psychogenic movement disorder (primarily in cases of psychogenic tremor and psychogenic myoclonus)
Probable	(a) Inconsistent/incongruent: no other features (b) Consistent/congruent + "false" neurological signs[c] (c) Consistent/congruent + multiple somatizations[c]	
Possible[d]	Consistent/congruent + obvious emotional disturbance	–

[a] From Fahn and Williams [9].
[b] From Gupta and Lang [12].
[c] It has been proposed to reclassify these patients under "possible" [12].
[d] The utility of retaining the "possible" category is questioned since this generally represents patients with organic movement disorders with additional psychiatric problems rather than a true "possible psychogenic movement disorder" [12].

outlines the original Fahn/Williams criteria as well as the proposed revision of Gupta and Lang [12].

There are a variety of clues that may assist in the diagnosis of psychogenic movement disorders. These can be subdivided under those obtained in the patient's history and those evident on the clinical examination. Tables 2.3 and 2.4, respectively, outline the more important historical and clinical clues. It should be emphasized that all of the points listed are no more than clues, and exceptions to each of them can be found in patients with organic neurological dysfunction. Importantly, a detailed history may require obtaining extensive records involving previous assessments, hospitalizations, and so on. Patients may not volunteer the full details of their previous assessments or may report inaccurate or inexact information related to these [14,15]. In some patients where the clinical features are uncertain or questionable, repeated assessments may need to be conducted over several visits. Video surveillance has been used to document the absence of the movement disorder [16], but, of course, this approach has to be used carefully. Where available and appropriate, the electrophysiological laboratory may be extremely helpful in confirming the diagnostic suspicion [17,18], although occasionally it can provide somewhat misleading information [19].

A detailed description of each of the psychogenic movement disorder phenotypes is beyond the scope of this introductory chapter (see Hallett *et al.* [20]). Tables 2.5–2.9 summarize most of the important clinical characteristics of the commonest psychogenic movement disorders and, where possible, contrast these features with the abnormalities seen in patients with their organic counterparts. Possibly two of the most important descriptors that apply to psychogenic movement disorders are their inconsistency and their incongruency. The abnormal movements are generally inconsistent over various time frames (either over the course of an individual examination or at different times on repeated assessments). Equally important

Table 2.3 Historical clues suggesting that a movement disorder may be psychogenic

Common historical clues	Exceptions/caveats
Abrupt onset often triggered by minor injury	Slow onset occasionally seen; "organic" movement disorders occasionally begin abruptly
Static course, early development of maximal or near maximal severity	Progressive course sometimes seen, possibly more often in psychogenic parkinsonism than others
Spontaneous remissions (inconsistency over time)	Spontaneous remissions occasionally seen in "organic" movement disorders such as cervical dystonia
Obvious psychiatric disturbance	Caution: overt psychiatric disturbances may not be evident and psychiatric problems are not uncommonly present with "organic" movement disorders
Multiple somatizations	
Employed in health profession	Obviously does not exclude the possibility of an "organic" movement disorder
Pending litigation or compensation	As above, i.e., does not exclude an organic disorder by any means
Presence of secondary gain	May not be evident
Young female	Psychogenic movement disorders may be seen at all ages in both genders

Table 2.4 General clinical clues suggesting that a movement disorder may be psychogenic

1. Inconsistent character of the movements (amplitude, frequency, distribution)
2. Movements increase with attention or decrease with distraction
3. Inconsistencies between performance on examination (often movements are most prominent) and times when the patient is not actively being examined (e.g., while giving a history or observed surreptitiously)
4. Selective disabilities not typical of "organic" task-specific movement disorders
5. Ability to trigger or relieve the abnormal movements with unusual or non-physiological interventions implying suggestibility (e.g., trigger points on the body, tuning fork; encouraging spread of movements to unaffected regions while restricting movement in the originally affected area [i.e., immobilizing])
6. Paroxysmal movement disorder[a]
7. Deliberate slowness of movements; performance of requested movements may appear to require extreme effort (often with excessive sighing or hyperventilation); commonly there is a major dissociation between this performance (on examination) and spontaneous performance of movements at other times (e.g., when not formally being examined)
8. Suffering or strained facial expression (particularly when asked to perform various tasks on physical examination)
9. Active resistance to passive movements (particularly with dystonic postures; also may account for "pseudorigidity" in psychogenic parkinsonism)
10. Movement abnormality that is bizarre, multiple, or difficult to classify
11. Functional disability out of proportion to examination findings
12. False weakness
13. False sensory complaints
14. Self-inflicted injuries[b]
15. Response to placebo, psychotherapy, isolated physiotherapy

[a] Must consider organic paroxysmal movement disorders.
[b] This may be seen in some "organic" movement disorders including tic disorders and neuroacanthocytosis.

Table 2.5 Clinical features of psychogenic versus "organic" tremors

Psychogenic tremor	Organic tremor
Often rest = posture = action; sometimes posture = action without a rest component	Variable depending on cause; rest tremor of Parkinson's disease diminishes/abates with action (may re-emerge in the new position); typically in other disorders rest < posture < action
Often marked variability in direction, joint and muscle involvement	Generally consistent but may vary depending on posture and activity
Variability in frequency; irregular	Frequencies generally consistent, usually regular; dystonic tremors may be irregular
Fingers uncommonly involved particularly in isolation	Fingers not infrequently involved
Often subsides or becomes more irregular with stressful tasks (e.g., mental arithmetic)	Amplitude frequently increases with stress while frequency remains constant
Complex physical tasks often cause tremor to subside (distractibility) or become more irregular (changing frequency); repetitive rhythmical task (e.g., tapping to a constant frequency using a metronome) may entrain the tremor to the new frequency or simply change the original frequency. Slow, side to side movements of the tongue may be associated with two possible outcomes: distractibility with changes in the frequency of the tremor or persistence of the tremor but extremely poor performance of the tongue movements in the absence of any dysarthria or other disturbances of orolingual function	Frequency remains relatively constant while amplitude often increases
Common features of the tremor: absent isolated finger tremor; flapping movements with variable direction; tremor often at physiological clonus frequency (distractibility and entrainability may be less in this circumstance) Leg tremors: foot plantar flexed with heel lifted slightly from the floor; leg partially flexed at knee with tremor in the thigh causing flexion/extension movements below the knee. May appreciate the "co-contraction sign" in evaluating tone	

Table 2.6 Clinical features of psychogenic versus "organic" dystonia

Psychogenic dystonia	Organic dystonia
Inconsistent/variable	Consistent and relatively stereotyped; may be action specific or largely action induced
Fixed dystonic postures common and often early in the course	With certain exceptions, dystonia is usually "mobile" and often purely action specific or action induced initially
Response to sensory tricks exceedingly rare	Response to sensory tricks (gestes antagoniste) common particularly early in the course (more typical of idiopathic than symptomatic dystonias)
Pattern usually inconsistent with organic counterparts	Recognized patterns of dystonia typical in different age groups (e.g., generalized and segmental forms more common in children while focal involvement [most often cranial, cervical, upper limb] more common in adults)
Pain may be a prominent feature, often associated with profound tenderness	Often painless (the main exception is cervical dystonia)
Often associated with marked resistance to passive movement even giving the sense of actively resisting the examiner	Tone may be normal or increased at times that the dystonic postures are most evident (dystonic rigidity)
Common features of dystonia: fixed dystonia at onset; leg involvement beginning in adult life (no evidence of additional neurological deficit such as parkinsonism); tonic downward pull of the mouth (unilateral or bilateral) Tonic posturing often persists despite attempted distracting maneuvers (i.e., distractibility is far less common in psychogenic dystonia than in more "mobile" psychogenic movement disorders such as tremor or myoclonus)	

Table 2.7 Clinical features of psychogenic versus "organic" myoclonus

Psychogenic myoclonus	Organic myoclonus
Often variable in distribution	Apart from multifocal myoclonus, distribution is more consistent
Distractibility, suggestibility	No influence of these maneuvers
If movements are stimulus induced (including excessive response to startle), the latency may be obviously long or quite variable (formal electrophysiology testing may be required to confirm this); jerks may be triggered by the threat of stimulus (e.g., following repeated taps with the reflex hammer a subsequent tap may be held up before touching the patient)	Consistent short latency evident
Characteristic features of myoclonus: large amplitude synchronous flailing of the arms from the sides or crossing the chest; pronounced trunk flexion (caution – organic propriospinal myoclonus); pelvic thrusts Electrophysiological characteristics: latency within voluntary reaction time, variable duration of bursts (usually > 300 ms), varying patterns of muscle involvement); activity may be preceded by a Bereitschaftspotential in the trace	