PSYCHOLOGICAL APPROACHES TO CHRONIC CATATONIA-LIKE DETERIORATION IN AUTISM SPECTRUM DISORDERS

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The psychological dysfunctions that may underlie catatonia-like deterioration in autism spectrum disorders are discussed. Clinical observation suggests that an important factor is ongoing stress. The evidence for this from research and clinical observation is considered. The lack of evidence concerning the most appropriate medical treatments is discussed. A psychological approach designed for individual needs by relevant professionals and applied by parents and/or caregivers is described. This can be helpful whether or not medical treatments are used. It involves detailed holistic assessment of the individual and their circumstances to highlight possible precipitating stress factors in view of their

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underlying autism and cognitive/psychological functioning. The overall aim of this approach is to restructure the individual's lifestyle, environment and resolve cognitive/psychological factors to reduce the stress. An eclectic approach is used to find individual strategies in order to provide external goals and stimulation to increase motivation and keep the person engaged and active in meaningful and enjoyable pursuits. The approach describes ways of using verbal and physical prompts as external stimuli to overcome the movement difficulties and emphasizes maintaining a predictable structure and routine for each day. The importance of educating caregivers and service providers to understand the catatonia-like behavior is emphasized. Advice is given on management of specific problems such as incontinence, freezing in postures, eating problems, and episodes of excitement.

> Whether a particular disorder is precipitated or relieved by psychological factors has no bearing on whether a neurological or psychological paradigm is more appropriate for understanding it.
>
> Rogers, 1992

I. Introduction

As described in Chapter 2 by Wing and Shah, in a small minority of people with autism spectrum disorders (ASDs), some catatonia-like and some parkinsonism-like features become very marked or appear for the first time and are severe enough to interfere with the activities of everyday life. This usually happens in adolescence or adult life, though can rarely be seen in childhood. In most cases the onset is gradual and the presence of all the features of classic stupor appears to be rare (Wing and Shah, 2000). This will be referred to here as "catatonia-like deterioration," to avoid the premature conclusion that it is the same as catatonia. A prevalence of 17% was found among people aged 15 years or above who were referred to a diagnostic service (Wing and Shah, 2000).

Because of our special interest in catatonia-like conditions in autistic disorders (ADs), the present authors have seen a large number of individuals with these clinical pictures. The problems of movement, speech, and behavior in catatonia-like deterioration in ADs are similar to those found in acute and chronic catatonic states. But, in our experience, typical catatonic stupor is rare, and we have not seen waxy flexibility in anyone referred to us. The onset of the deterioration in those we have seen was characteristically slow. However, individual cases of autism and catatonic stupor have been reported in the literature (Dhossche, 1998; Ghaziuddin *et al.*, 2005; Realmuto and August, 1991; Zaw *et al.*, 1999).

The precise neuropathology of ASDs is as yet unknown, and there are no known treatments that can cure the characteristic impairments of social interaction, communication, and imagination. At the time of writing, no systematic studies of the causes, nature, and treatment of catatonia-like conditions in ADs have been published. The few papers in the literature that possibly have some bearing on this condition will be discussed, but the results they describe tend to make confusion worse confounded.

II. Effects of Stress

Our clinical observations suggest that ongoing stressful experiences are a major precipitating factor in many individuals who develop catatonia-like deterioration. We have identified various diverse stress factors. These include:

1. External factors, such as being in unstructured environments and programs, which are not autism-friendly. The loss of routine, structure, and meaningful occupation that occurs after leaving school is a common factor. In adolescence, the pressure of major examinations becomes unbearable for some individuals. In adults, an increase in social, adaptive, and independence demands leads to stress and noncoping and eventually giving up. Significant life events, such as bereavement, family break up, house move, and so on may also have a severe effect on some individuals.
2. Psychological factors such as experience of conflict, pressure, and confusion. This can be due to diverse factors such as not having a diagnosis or explanation for one's difficulties or alternatively not accepting a diagnosis or an accumulation of failure experiences. In high functioning people with Asperger Syndrome (AS), an awareness of their limitations and differences from peers/siblings can cause psychological pressure and stress. In some cases, the conflict between parental expectation and the individual's capacity and lack of motivation adds to the pressure.
3. Biological factors, such as illness, pain, and hormonal changes (e.g., during puberty), are also implicated in some cases.

The mechanisms through which the experience of stress leads to catatonia-like deterioration are completely unknown. At a neuropsychological level, it can be speculated that in vulnerable autistic individuals, functions and systems that are weak are further weakened. Catatonia-like deterioration is most commonly seen in individuals who have shown the "passive" type of social impairment and functioning (Wing and Shah, 2000). Individuals in this "passive" subgroup have ongoing difficulties of motivation, volition, and initiating activity and interaction. Uta Frith's theory of central coherence deficit and frontal lobe

executive dysfunction in ASDs leading to difficulty in controlling and regulating behavior offers a possible explanation. (Frith, 2003; Hill, 2004). It is possible that individuals in the "passive" subgroup have a weaker system of central control of actions. Ongoing stress may further disrupt this function and give rise to the characteristic problems of initiating and ceasing actions and speech, poor control of behavior, and increased dependence on external prompts.

These speculations give no help in understanding those cases of catatonia-like deterioration that follow a course in which slowness, movement difficulties, and muteness are very evident on some days, but are not seen at all on other days. In instances when the individual is able to "miraculously" overcome the inability to initiate an action or movement for a specific sudden stimulus, it can be hypothesized that the power/shock of the external stimulus jolts the weak central control system into temporary "action." One such example relates to a young man described in Wing and Shah (2000) who was permanently confined to a wheelchair due to catatonia-like deterioration. On one occasion, when his elderly father stumbled and nearly fell, the young man was able to leap from his wheelchair, save his father from falling, but then returned to immobility.

At a psycho-physiological or neurological level, there is no research yet that has identified any specific mechanisms relating stress and catatonia-like deterioration in autistic individuals. The following discussion considers studies and hypotheses that have some bearing on the subject.

Dhossche and Rout (Chapter 4) have offered a biochemical theory involving abnormal gamma-aminobutyric acid (GABA) function that may link autistic symptoms and catatonia. Adaptive and maladaptive stress responses are regulated by GABAergic processes in the hypothalamus (Kovacs *et al.*, 2004). Hypothalamic abnormalities may be present in autism and catatonia.

There is an interesting case study by Loos and Miller (2004) of the link between stress and "shut-down" in a child with high functioning autism. Their description of the "shut-down" state has a lot of overlap with catatonia-like deterioration. These authors have suggested that chronic "stress instability" can lead to "shut-down" episodes and cause nervous system damage and developmental impairments with the autism spectrum. These authors have suggested that a corticotropin releasing factor (CRF) that is important in stress functions may be unstable in some individuals within the autism spectrum.

The subjective experience of anxiety and physiological correlates of anxiety may have some relevance to the mechanisms relating stress and catatonia.

It has been suggested, in relation to typical catatonic stupor, that extremely high anxiety is an important factor. Northoff (2002) investigated retrospectively the subjective experiences of patients who had had episodes of classic acute catatonic stupor. The dominating emotion reported was anxiety that was overwhelming in its intensity. Northoff contrasted these retrospective subjective experiences with those of people with parkinsonism. He noted that the people

who had had catatonic episodes seemed unaware that they had had problems with movements, whereas those with parkinsonism were fully aware of this aspect of their condition. People with ASDs, especially those with catatonia-like deterioration, have difficulty verbalizing their feelings. However, one young man in the study by Wing and Shah (2000) who was described in Chapter 2 and another man seen subsequently were able to give accounts of their experiences during catatonia-like deterioration. Both described clearly that they wanted to move, tried to move, but their bodies would not obey. They did not describe feelings of anxiety.

A different picture was found in another young man assessed by Shah. He had high functioning AS and developed catatonia-like deterioration after leaving school. During the psychological assessment it was noticed that this young man showed no signs of movement disorder associated with catatonia when he was focusing on verbal and nonverbal cognitive tests, which he enjoyed and found extremely easy. However, when the assessment required him to express and verbalize his emotions and feelings, he seemed to become very anxious, confused, and immediately showed movement problems such as freezing, slowness in action, and difficulties in talking fluently. It was as if the inability to express his emotions and feelings caused intense anxiety and stress, which had a direct effect on his movements and speech. This young man, despite having above average verbal and nonverbal intelligence was unable to verbalize any aspect of his catatonia-like deterioration or anxiety.

There are, specifically for this group, no published studies of the physiological measures that have been found to be associated with feelings of anxiety (Lader and Wing, 1966). However, with hindsight, it now appears likely that some, perhaps many people, who in the past were diagnosed as having catatonic schizophrenia, in reality had undiagnosed ASDs (Nylander and Gillberg, 2001; Wing and Potter, 2002; Wing and Shah, 2000). People with ADs, who have catatonia-like deterioration, would easily meet the criteria for "catatonic schizophrenia" in Diagnostic and Statistical Manual, 4th ed. (DSM-IV) and International Classification Diseases, 10th revision (ICD-10). Ungvari *et al.* (1999) found that adults diagnosed as having chronic catatonic schizophrenia did not report experiencing anxiety. If they did have ADs, it is unlikely that they would have reported their feelings. However, Venables and Wing (1962) examined skin potential and 2-flash threshold in a group of adults living in institutions and diagnosed as having various types of schizophrenia. They found that the most socially withdrawn, mute or almost mute participants, some diagnosed as having catatonic schizophrenia, had the highest levels of skin potential and lowest 2-flash thresholds of any of the subgroups. The authors suggested that these measures indicated events in the brain, referred to in the paper as "high levels of arousal," which led to marked withdrawal from the environment (that is the poor speech, slowness, and unresponsiveness of chronic catatonia-like conditions). The authors

also noted that, in people with "chronic schizophrenia," increased arousal had been found to impair the normal selectivity of perception, making them especially vulnerable to events in the environment. This observation is interesting in the light of the oversensitivity to sensory input that is so characteristic of people with ADs and subjective reports of "switching off" when overloaded with sensory input (e.g., Williams, 2003).

This study left many questions unanswered. Were the brain events associated with the physiological measures and the speech, motor, and social problems a response to environmental pressures? Were the brain events a response to the distress caused by the speech, motor, and social problems? Were the brain events indicated by the physiological measures directly or indirectly the cause of the catatonia-like clinical picture or not related at all? Most important of all, what were the neurological, biochemical, and psychological pathways that led from environmental (or internal, e.g., hormonal) stress to catatonia-like conditions in vulnerable people? Further systematic research is needed to answer questions concerning individual differences in tolerance of stress and experiencing and verbalizing anxiety.

III. Effect of Medical Treatments

Northoff (2002) noted that 60–80% of people, who had had acute catatonic stupor and reported severe anxiety, responded rapidly to high doses of lorazepam, but those who had not been anxious were not helped by this medication (Northoff *et al.*, 1998). Ungvari *et al.* (1999) in the study mentioned earlier carried out a double-blind placebo-controlled crossover trial of lorazepam in a group of people diagnosed as having chronic catatonic schizophrenia. They found that lorazepam in doses of 6 mg/day over 6 weeks was ineffective. As mentioned earlier, these researchers had also found that the participants in the trial did not report subjective anxiety.

The few studies that have been published on the treatment of catatonia-like states in ADs have reported individual cases with acute catatonic stupor that responded to medication and/or electroconvulsive therapy (ECT) (Dhossche, 1998; Ghaziuddin *et al.*, 2005; Realmuto and August, 1991; Zaw *et al.*, 1999). In contrast, the paper by Brasic *et al.* (2000) described the clinical course over 7 years of chronic catatonia-like deterioration in a young man with pervasive developmental disorder. After the first 6 years, he received 25 treatments with ECT with no therapeutic effect. Whether there were any adverse effects was not reported. Among the participants in the study by Wing and Shah (2000), only two had had ECT. One individual had had 3 courses. The first gave 6 months remission of the catatonia-like symptoms, the second gave 3 months remission,

and the third was ineffective. The ASD was not affected. One other person had one course of ECT with no effect. We have never seen, in chronic catatonia-like states in ASDs, the dramatic recoveries reported following the administration of lorazepam and/or ECT for typical catatonic stupor (Fink and Taylor, 2003; Northoff, 2002).

IV. Problems with Assessing the Effects of Medical Treatments

It has been pointed out that there are major problems in assessing the effects of medical interventions that have been used for catatonia in people with ADs (Dhossche, personal communication).

First, none of the people in our study (Wing and Shah, 2000), any seen since, or those in the Ungvari *et al.* (1999) study had been given the high doses of lorazepam that are recommended by specialists in the field of catatonia. Also, it is not known whether the administration of ECT in the two cases described earlier followed the modern guidelines for treatment of catatonia (Fink and Taylor, 2003). There are no reports in the literature of clinical trials in which these guidelines for treating typical catatonia have been used in catatonia-like deterioration in ADs. Until research of this kind has been carried out, the questions remain unanswered. But the present authors' clinical experience does not give rise to any optimism concerning the value of the available medical treatments in ASDs.

Second, the effect on response to treatment of the chronicity of the catatonia-like deterioration in ASDs, even when accepted treatment guidelines are followed, is unknown. Chronic "catatonic schizophrenia" is a notoriously treatment-refractory condition (although there are a few case reports describing unexpected and significant gains in function in such patients). Acute-onset catatonia on the other hand, even in its most severe forms, is reported to have a good prognosis, at least at short-term follow-up (Fink and Taylor, 2003). It is possible that early recognition and prompt, adequate treatment may be essential to avoid chronicity. This important issue warrants systematic study in people with catatonia with and without ASDs. Current data do not allow any definite conclusions as it is unlikely that people with ADs with chronic catatonia-like deterioration have received treatment reported to be optimal at the onset of the catatonia or later stages. However, it must be emphasized that catatonia-like features are part of the clinical picture of ASDs from early childhood onwards. Catatonia-like deterioration appears against this background. This raises the question of when and how to recognize the onset of the deterioration. Also in our current state of knowledge, there is no certainty about the relationship between slow-onset catatonia-like deterioration in ASDs and acute catatonia.

The mechanism of action of ECT is unknown. ECT has been effective in individual cases reported in the literature mentioned earlier of children or adolescents with ADs who had typical catatonic stupor (but not for two individuals, known to the present authors, who were not in stupor but had catatonia-like deterioration). Dhossche and Rout (Chapter 4) have offered a biochemical theory involving abnormal GABA function that may link autistic symptoms, catatonia, and the mechanisms of action of ECT.

V. Psychological Methods of Intervention

It appears from the few studies of individuals in print that catatonic stupor in autism may respond to lorazepam in appropriate doses and/or ECT. It is important to emphasize that these treatments should be used if the catatonia is life threatening.

However, for chronic catatonia-like deterioration without stupor, for at least some individuals, even the most appropriate medical treatments may not work or produce only partial remissions. As things are at the time of writing, very little help is available for people with ADs and catatonia-like deterioration. Faced with seemingly intractable problems in the person for whom they are caring, many parents and caregivers have turned to us, the authors, for help because of our special interest in the field. Over the past 10 years, we have discussed ideas on psychological interventions with professionals, care staff in residential homes, and parents who have practical experience of catatonia-like deterioration. We have observed the results of different approaches and collected feed back from those involved.

It is important to point out that medical and psychological approaches are not incompatible. People with catatonia-like deterioration have to be helped and supported throughout the day (and sometimes the night) while awaiting medical treatment, the treatment to take effect, and if it proves ineffective. The psychological approach is appropriate in all these situations. Furthermore, having an understanding and a clear plan to follow give parents and caregivers the confidence that allows them to be calm and supportive, instead of experiencing the anxiety and despair of not knowing what to do. The cognitive reconceptualization of the problem as neuropsychological which is not due to the individual being lazy, willful, stubborn, or manipulative, gives rise to renewed hope and a constructive approach to appropriate interventions. This in itself reduces stress for all concerned, including the affected individual, and has positive benefits.

We have found that the most effective approach is to adapt and extend the principles that are known to be effective in understanding and supporting individuals with ASDs. The approach for each individual depends on the premorbid pattern of autistic impairments and how these have been affected by

the catatonia-like deterioration and the precipitating stress factors. The relative rarity of this catatonia-like condition and the resulting wide geographical spread of cases known to us have made it difficult to conduct field trials and carry out a scientific evaluation of the results. From the information we have collected, the psychological techniques of management described later appear in practice to be helpful in improving quality of life for some, if not all, affected individuals. In some cases, they have resulted in complete remission of the deterioration.

It is also true that for some individuals with catatonia-like deterioration, who are of high ability and/or have always been rigidly stubborn and noncompliant, the psychological approaches are difficult to apply and may not help. In our experience, such people may also adamantly refuse to take medication. The presence of a severe comorbid psychiatric condition can also make treatment difficult. The psychological approach appears to be most likely to be helpful in people who had, prior to the deterioration, tended to be passive in their social interactions (Wing, 2005; Wing and Gould, 1979). Predictors of favorable (and nonfavorable) response to the proposed psychological approach should be assessed in future studies.

VI. General Principles of Psychological Treatment and Management

The general principles of treatment and management are discussed in later sections. However, clinicians must always be aware that the program must be adapted to the needs of each individual, on the basis of the clinical picture. It must also be modified in the light of the individual's progress.

A. Initial Assessment

Our clinical experience suggests that early recognition of the onset of catatonia-like deterioration is as important for psychological as for medical treatment. Once a pattern of disability has become chronic, it appears that it is more difficult for any type of intervention to have an effect, though this must not deter clinicians, parents, and caregivers from giving the most appropriate treatments available.

For some people, the diagnosis of an ASD has been made before the appearance of catatonic features. In others, the presence of an AD has not previously been identified. In either case, assessment should include a detailed developmental history, current clinical picture, and psychological examination of skills and disabilities. It is essential to collect information in a systematic form from informants, preferably parents if available. One instrument that can be used is the National Autistic Society's Diagnostic Interview for Social and Communication Disorders (DISCO). This is a semistructured interview that

covers areas of development as well as behavior found in ASDs (Wing *et al.*, 2002). It elicits information for the diagnosis of autism and related conditions and catatonia-like deterioration. It also identifies the degree to which the catatonia-like deterioration affects the person in everyday life. This information, together with that obtained from detailed psychological assessment, observation, and other available sources of information, can be used for identifying individual stress factors and needs, and devising an individual program of treatment and management, and as a baseline for its evaluation. Diagnostic criteria indicating a specific associated psychiatric condition should be identified, if present.

The psychological assessment should include appropriate tests of cognitive ability. Tests, such as the Wechsler (1997) Adult Intelligence Scale, Wechsler (1992) Intelligence Scale for Children, and Leiter (1990, 1997) International Performance Scale (a nonverbal cognitive test), are very useful for obtaining quantitative estimates of cognitive level and pattern of abilities, and qualitative assessment of the individual's approach and particular motor difficulties (Shah and Holmes, 1985). It is important to ascertain whether the severe movement difficulties are masking high cognitive abilities. These can be obscured though they are not impaired by catatonia-like deterioration. The information about cognitive level and profile is essential for providing advice about the appropriate level of cognitive stimulation and for selecting structured activities likely to motivate the individual concerned.

The motor components of catatonia-like deterioration can appear in a wide variety of forms. Clinical investigation should cover other identifiable causes of motor problems, any of which could occur in a person who also has an AD. Thorough medical and dental examinations are also needed in case there is a physical condition causing chronic distress. People with ADs, who have poor language, have great difficulty in talking about or showing physical symptoms.

As emphasized by Fink and Taylor (2003), long term administration of neuroleptic drugs can lead to motor disorders including tardive dyskinesia. Continuous mouth and tongue movements, as well as catatonia-like and parkinsonian-like features are described as characteristic of this condition. However, the same phenomena, including continual mouth and tongue movements, can also occur in catatonia-like deterioration in a person with an AD who has never received neuroleptics. Careful consideration should be given to past and present medications as possible causative agents of catatonia-like motor problems.

B. Dealing with Stress Factors

A fundamental aim of the psychological approach for catatonia-like deterioration in ASDs is to identify and reduce the stress being experienced by the person concerned. This will involve restructuring the individual's lifestyle,

environment, daily program, and resolving cognitive/psychological sources of stress. Professionals need to draw from a variety of psychological/behavior therapies and use the most appropriate eclectic approach to achieve change for the individual. A related important aim is to provide external goals and stimulation to increase motivation and meaningful, enjoyable activities for the individual. The programs must be adapted to the individual concerned and may involve change at various levels (individual, carer, systems, programs, residential, day services and staffing level, and intensity of support). Hare and Malone (2004) described a detailed program for helping one young man overcome his extreme difficulty in ascending and descending stairs. They emphasized the importance of planning such a program specifically for each individual, rather than having a standardized approach to everyone with "autistic catatonia," to use the terminology they preferred.

C. Conceptualization of the Nature of Catatonia-Like Deterioration

Everyone concerned with the affected person needs to understand that having catatonia-like deterioration is a neurological complication that can occur in ASDs (Wing and Shah, 2000). The effects on movement are not under voluntary control. The person is not being deliberately manipulative, stubborn, willful, obstructive, or lazy. The condition must cause severe distress and frustration to those affected, and they need a sensitive, sympathetic, and understanding approach. Caregivers must aim to be calm, gentle but confident, and positive in their approach. Psychological techniques of reframing information, cognitive restructuring, and shared communication models can be used to inform, motivate, and influence parents, care providers and service providers in finding individual proactive strategies and solutions.

D. Use of Prompts as External Stimuli/Signals

People with catatonia-like deterioration often need to be given verbal or physical prompts to move on or complete an action or a specific activity. The amount, level, and type of prompting necessary will vary for different people, situations, and actions for the same person. The aim of the prompt is to enable the person to carry out movements and actions as smooth as possible. For people with severe catatonia-like deterioration, prompts may not be enough, and they may, at least initially, need substantial physical assistance.

Verbal prompts can vary from quietly calling the person's name to giving specific instructions for each action required. Each prompt may need to be

repeated several times before the person is able to respond, and the person will require time to respond.

Physical prompts should start with a minimal light touch. This may be enough to enable the person to begin a movement or action. If not, the physical prompt should be extended so that it guides the movement in the right direction. As the person makes progress, the type and level of prompt can be adjusted and reduced.

Staff members and parents, who care for the person, may find it difficult to decide whether he or she should be assisted by prompts or left to carry out actions in his or her own time. Caregivers may feel that there is a danger of making the person dependent on prompts and thereby reducing the capacity of the person for independent action. Sometimes, staff members feel uneasy about prompting or assisting because this seems to be encroaching on the person's right to privacy and dignity. To resolve this dilemma, it is necessary to understand the behavior in the context of catatonia-like deterioration and the possible long-term negative effects on independence and functioning if the condition progresses. Prompts are necessary to enable individuals with catatonia-like deterioration to overcome the difficulties in the central control of voluntary movement and gradually to regain their independence. If left to their own devices and expected to learn the "hard" way, the person concerned may become increasingly unable to initiate movements and gradually less active and less independent. The inability to initiate and complete movements causes frustration, resulting in stress and tension that, in turn, is likely to exacerbate the difficulty with voluntary movements.

E. Maintaining and Increasing Activity

The effects of catatonia-like deterioration can be reduced substantially by keeping the person active, mobile, and stimulated without putting pressure on them, and by reducing additional demands

Physical activities involving rhythmic, repetitive movements are particularly beneficial. Such activities may include walking, swimming, trampolining, cycling, roller-skating, or roller-blading, ice-skating, dancing, music, and movement. Any other activities that the person seems to find meaningful and enjoyable and is able to carry out relatively easily should be included. Activities that require excessive physical effort and those that the person finds very difficult should be avoided. It may be useful for the person to join in specific physical activities with a small group of people. The momentum of the group is often helpful in enabling the person to begin and continue the activity. They may still need one-to-one support, prompt, and guidance through the activity.

The aim is to keep the individual active goal-oriented and stimulated. It may be necessary to have a one-to-one carer most of the time who can encourage,

prompt, and direct throughout the day. Such intensive support may be required in the initial treatment phase and reduced gradually as progress is made.

As well as having problems initiating actions, people with catatonia-like deterioration may find it difficult to stop a repetitive action once started. Examples are continual brushing of hair, repetitively putting a cup to the lips after the drink has been finished, even continuing to ride a bicycle round a track to the point of exhaustion. Again, gentle prompting, verbal or physical, may be necessary to help the person to stop.

F. Structure and Routine

A structured plan of activities of the kind suggested in the earlier section and a predictable routine are necessary for people with catatonia-like deterioration to develop the habit of participation. Habitual actions are much easier for such people, in contrast to new or sporadic activities that are hard, even impossible for them to start. Unpredictability, ad hoc plans, and uncertainty will increase stress and may be the trigger for episodes of freezing or excitement. Principles of the TEACCH approach are useful in devising structured programs, organizing the physical environment, making expectations clear and explicit, and using visual plans for daily schedules (Mesibov *et al.*, 1994).

VII. Management of Specific Problems

A. Incontinence

People with catatonia-like deterioration may show signs of incontinence. This is usually related to the difficulty of getting up from a chair or bed and reaching the toilet in time or the inability to ask to go to the toilet. Such incontinence is puzzling and distressing for a person who previously was fully independent in this respect. This needs to be understood and managed in a discrete and sensitive way. Simple methods, such as regular, frequent physical and/or verbal prompts to go to the toilet and giving enough time to use the toilet, are useful. The person may need assistance with clothes and then be given a verbal prompt to use the toilet. Depending on the severity of catatonia-like deterioration, the person may need physical assistance with personal hygiene.

People with severe catatonia-like deterioration who have difficulty using the toilet independently will benefit from a regular toilet use program. The use of the toilet should be linked to the daily routine by slotting it in at appropriate times, such as immediately on waking, at the beginning and end of each period of

activity, before or after each meal time, and before going to bed. This will work only if there is a consistent daily routine that is preplanned and adhered to as strictly as possible. As the person makes progress, the amount of prompting and physical assistance should be adjusted accordingly.

B. Eating Problems

Some people with catatonia-like deterioration develop severe difficulties with eating. The complex motor coordination required for eating with a knife and fork or with a spoon seems to trigger particularly severe difficulties in initiating and completing movements, and ritualistic and repetitive behavior. There are also difficulties with the movements of lips, jaws, and tongue required to take the food off the fork or spoon and to chew and swallow it.

The eating difficulties are often misinterpreted as "playing up," being deliberately slow, or having a poor appetite. Parents and caregivers may be advised by professionals involved not to be concerned, to leave the person to eat if they want to and when they want to. The result may be severe loss of weight and an exacerbation of the other aspects of catatonia-like deterioration.

The problems can be reduced by using verbal and physical prompts and making the process of feeding as easy for the person as possible. Depending on the extent of the difficulties, any of the following methods may have to be used:

1. Using a spoon instead of a fork and knife
2. Adjusting the type and consistency of food so that it is easily scooped onto a spoon. Some people may need liquidized food, which can be fed from a cup
3. Verbal prompts required for each action or each mouthful
4. Physical prompts that may range from touching the elbow lightly to giving hand on hand support and guiding the person's movement in the direction required
5. If prompts are not sufficient, the person may have to be fed
6. If the person is having difficulty opening the mouth, a light touch on the cheek, or touching the lips with the loaded spoon may be effective.

C. Speech and Communication Problems

The planning and execution of the movements required for speech may be as difficult for a person with catatonia-like deterioration as any other motor activity. It is important not to put pressure on them to answer questions or to talk. Instead, others should talk to the person, focusing on the current activity. It is more

important to relate to the person through physical activities than through verbal discussions.

Having to verbalize or indicate choices is particularly difficult for people with ASDs, and this is exacerbated by the motor problems of catatonia-like features. Caregivers may need to make the decisions and choices for the person on the basis of their knowledge of the person, their interests, and their likes and dislikes. Gentle suggestion and encouragement without asking directly what the person wants to do or whether or not they want to do something, is the most helpful approach. Visual communication systems and nonverbal personal communicators may be useful for some individuals whose speech is severely affected by catatonia-like deterioration.

D. Difficulty with Walking

Usually, a person with catatonia-like deterioration will be able to walk without stopping if he or she is holding on to or linking with the carer's arm. If the person stops suddenly, a light physical prompt on the back or a verbal prompt will help them to start again. Sometimes, walking as part of a group enables the person to walk at a steady pace without stopping. Some individuals have been helped by being encouraged to take the dog for a walk and hold on to the lead. This provides a continuous external stimulus and goal and can be very effective in keeping a person with catatonia-like deterioration moving at a steady pace.

E. Standing Still or Adopting Fixed Postures

Fixed postures of any kind will be extremely tiring for the muscles, but the person will not be able to express this, or do anything about it. The person who appears to be standing still and staring in space is probably unable to initiate movement needed for a particular activity. A verbal prompt or a light touch may enable them to move. They should then be involved immediately in a different activity.

Some individuals hold up one or both arms and may continue to do this for long periods if left alone. If verbal prompting to lower the arms does not work, physical assistance may be needed.

F. Catatonia-Like Excitement

Episodes of uncontrollable, frenzied, and inappropriate behavior may occur. These may, wrongly, be interpreted as outbursts of "challenging" behavior. This misconception can lead to a fruitless search for "triggers" and "communicative

functions" and the application of behavioral methods to discourage the inappropriate behaviors. In the clinical experience of the authors, this has not proved to be a useful approach for the management of catatonia-like excitement. Even more unhelpful is telling the person to stop behaving in that way, to stop being silly, or asking them why they are doing this.

If these episodes are short lived and do not affect the safety of the person, other people, or the environment, the best strategy may be not to intervene, though supervising to ensure safety and give support when the incident is finished.

If these episodes are more severe and longer lasting, intervention at different levels may help the person to calm down more quickly. Verbal suggestions to do something different, mild physical restraint, or simply physically leading the person into a different environment and prompting them to sit down may work. Different strategies are needed to suit different people. When the episode is over, the person should be reassured and encouraged to carry on with the normal routine.

VIII. Implications for Services and Staffing Levels

People with ASDs, who develop catatonia-like deterioration, need an intensive program of help in the right environment, as outlined earlier. Even those who show some response to medical treatment are liable to recurrence if they are in a setting that is not suited to their needs. They require 24-hour care in a setting in which a structured, organized daily program can be planned and carried out consistently. A high staff ratio and sufficient trained staffs are essential. There must also be full access to stimulating physical and occupational activities, of an appropriate kind, in suitable environments.

In the initial stages, the person will require a full time, one-to-one carer for the waking hours in order to carry out the program described. Ideally, each person should have one or two key caregivers who can build up a rapport and a relationship with the person concerned.

IX. Conclusions

As was noted in Chapter 2 by Wing and Shah, the few studies that are relevant or possibly relevant to the unraveling of the nature of catatonia-like deterioration in ASDs generate more questions than answers. The relationships among typical acute catatonic stupor, chronic catatonia, catatonia-like

deterioration, and parkinsonism remain unsolved. The relevance of measures of autonomic activity is still to be explored. Stress seems to have a role in the causation but the neurological, biochemical, and psychological mechanisms through which it operates have not been elucidated.

The place of medical treatments also remains a problem. The only published evidence comes from single case studies of individuals with ADs who develop catatonic stupor. We have seen many different medications tried, mostly with no or only short-lived beneficial effects. Our limited experience of ECT in these conditions (and, for Wing, considerable experience of its use for "catatonic schizophrenia" in the old institutions) gives no cause for optimism. Detailed clinical research is called for, carried out with caution, humanity, and respect for each individual concerned. Until clear results are available, the present authors will remain pessimistic about the medical approach, though appropriate medical treatment has to be tried in life-threatening conditions. The psychological methods of helping described in this chapter are safe, humane, person centered, and do lead to improvement in varying degrees in many people affected by catatonia-like deterioration. The suggestions in the Blueprint for assessment and treatment (Section IV) are fully in line with the cautious approach recommended in this chapter.

Many mysteries remain to be solved in this field. It is to be hoped that this book will be a stimulus to research to find the solutions.

Appendix A

Individual Case History

Bernard was 23 at the time of referral. He had attended a special school. He had always been quiet and passive and from 13 to 15 years, had received treatment for social withdrawal and selective mutism. The team treating him had concluded that Bernard was socially withdrawn and selectively mute due to his mother's over involvement with him and his resentment of this, though this was never evident in his overt behavior. The main part of this treatment consisted of psychological therapies aimed at enabling Bernard to express his repressed anger toward his mother. This approach was unsuccessful. At the age of 16, Bernard was diagnosed as having autism.

From 16 to 19 years, he attended a school for children with autism. The report from the school described Bernard as being unable to communicate normally and being able only to whisper in a soft voice when asked a question, provided the questioner was prepared to wait a long time for a response. He was also described as being extremely slow in his movements, often becoming fixed in a stationary

position for long periods. After leaving the education system, Bernard was not offered any further training or community service. His mother could see that he was distressed by the absence of any structure to his day. She tried to refer him to the local community team for people with learning disabilities. However the referral was rejected on the grounds that Bernard had a recorded nonverbal IQ in the low average range. His mother had to work hard to care for him at home without any support. Bernard gradually deteriorated in his ability to carry out any movements or activities without help or prompting. During a 5-month period of eating very little he lost a great deal of weight. He was then referred to a mental health psychiatrist. A diagnosis of depression was made, and Bernard was prescribed antidepressants. At a review after a year, the psychiatrist was struck by the deterioration in Bernard's condition and referred him to us.

At the point of our involvement, Bernard, who used to be fully independent in all aspects of self-care, had become totally dependent on his mother. The morning routine of getting up, washing, and dressing was taking up to 5 hours. Bernard was only able to finger-feed bite size pieces of dry foods. However, he frequently became "frozen," and each small meal was lasting for hours. He was severely underweight and still losing more weight. Although previously he had been able to travel independently, he could not now travel on his own as he was unable to get up from his seat on the bus to alight.

Bernard hardly talked at all. When asked questions, he took a long time to respond and even then was able to answer only in monosyllables. He whispered the answers very softly. He seemed unable to lift his head to make eye contact or to interact. He had a fixed expression on his face, and was not able to acknowledge people in any way.

Bernard was spending most of his waking time on a sofa in the living room, in a fixed stooped posture with his head bowed and his arms hanging by his sides. He tried to respond to instructions, but each movement seemed agonizingly difficult. Before each action, his body twitched and jerked, he blinked continuously, and made repetitive movements with his mouth.

A comprehensive assessment was carried out. This included a detailed investigation of developmental history and past patterns of behavior, detailed observations of the current behaviors, responses and movement difficulties, and psychometric assessment using the Leiter International Performance Scale (a nonverbal test). A diagnosis of an ASD and severe catatonia-like deterioration was made. As described earlier, Bernard was showing a full range of catatonia-like phenomena. Stress factors identified included not coping in mainstream school, unsuitable therapies focusing on eliciting emotions, and lack of structure and occupation.

After the diagnosis, a case was made for the local services for people with learning difficulty to be involved and provide support to the family. An intervention plan based on the principles of reducing stress, providing appropriate

environment, program, and gradually increasing motivation and activities as outlined earlier was put into practice. This included training of caregivers to understand that Bernard's difficulties were due to catatonia-like deterioration. One-to-one support was provided. He was helped with physical and verbal prompting and a gradual increase in his activity level. Slowly Bernard started to become less rigid in his postures and movements. He was able to complete more and more movements with prompts, which were gradually reduced. He was able to feed himself a range of foods with minimal physical and verbal prompts. He began to respond more quickly. He was able to walk upright and the jerks, twitches, and repetitive mouth movements decreased and then stopped completely. As he progressed, the local health authority was persuaded to find a placement at a small specialist day centre, which provided a highly structured program of activities, one-to-one support and a calm quiet atmosphere. Bernard has continued to make progress and is regaining his mobility, speech, and independence.

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