# Introduction

According to the literature, anyone trying to stop taking psychiatric drugs must reckon with withdrawal problems. Ample evidence is provided for this statement in my book "Schöne neue Psychiatrie" *("Brave New Psychiatry,"* Lehmann 1996b, pp. 356ff.). In this book, "Coming off Psychiatric Drugs," I only provide a summary of the withdrawal symptoms. They may or may not occur.

When one discusses the issue of dependency with psychiatrists, most often their first reflex is to deny the danger of dependency for users of anti-depressants and neuroleptics. For example, the psychiatrists of the German Pharmaceutical Watch Group for Psychiatry have defined dependency as follows:

> "Psychological dependency is understood as the irresistible longing for a medication in order to increase a feeling of well-being or to reduce uncomfortable symptoms. Physical dependence can be established with the appearance of withdrawal symptoms after the reduction or withdrawal of a medication." (Grohmann / Rüther / Schmitz 1994, p. 279)

The risk of further problems developing in addition to the usual withdrawal symptoms, for example a rebound effect or hypersensitivity, should be taken into consideration when deciding whether or not to withdraw. Rebound effects are counter-regulatory adjustment reactions that lead to a temporary pronounced recurrence of the original symptoms.

Rebound effects have a mirroring-effect that make it particularly difficult to recognize withdrawal symptoms as distinct from the original problems. Because a prolonged use of psychoactive drugs raises the probability of various withdrawal problems—in addition to the usual damage of using the drugs—it is wise to consider sooner rather than later whether or not the time has come to limit these risks and to withdraw in a safe manner.

Taking psychoactive drugs can, under certain circumstances, lead to a temporary relief of psychological stress; but at the same time this leads many peo-

ple slowly into dependency without their knowing it and with the support of the medical establishment. Many people attempt to stop taking psychiatric drugs, for a variety of reasons: lack of "therapeutic" effect; unwanted effects; pregnancy; lack of insight into their "illness." Generally they have been left to cope with withdrawal problems on their own. In popular medical advice books (Curran / Golombok 1985; Neild 1990; Trickett 1991; Gadsby 2000) there is no mention of withdrawal from carbamazepine, lithium, antidepressants or neuroleptics. A few specialist journals published apparently random reports of severe withdrawal symptoms. But until recently, textbooks and information leaflets aimed at psychiatric drug users and their families still claimed withdrawal symptoms only arose with tranquilizers. In the past few years withdrawal effects of the newer antidepressants have gained some public notoriety. It is even possible to find information about dependence on "atypical" neuroleptics like Zyprexa (Support4Hope 2003). You only have to search long enough in the internet.

Typical withdrawal studies demonstrate quite serious methodological deficiencies, which have also been noticed by doctors: double-blind studies, in other words studies where neither the subject nor the treating physician knows what substance is actually being administered, are as rare as the administration of a placebo to a control group (which is also problematical). Furthermore, there has been a lack of systematic follow-up, a lack of information on the duration of hospitalization and of prior treatment as well as of the strength of dose of the psychiatric drug being withdrawn. Also, the period covered by the studies is too short, and finally, what is meant by any "relapse" mentioned is left completely unclear (Andrews / Hall / Snaith 1976). Those being treated were considered "improved" if in the eyes of the administrator of the psychiatric drugs they were not ready to be discharged but caused less trouble on the ward (Glick / Margolis 1962).

As Bertram Karon from the Psychology Department of Michigan State University concluded, the only purpose of some of these studies is simply to justify the prescribing practices of psychiatric drugs (Karon 1989, p. 113). For example, the American psychiatrist Philip May in his "California Study" (May 1968), much quoted among his circle of colleagues, claimed to prove the superiority of neuroleptics, antidepressants and electroshock treatment over psychotherapeu-

tic procedures. However, the report failed to point out that the therapists he used were untrained and unpaid trainees. A further deficit is the fact that in long-term studies only subjects who are motivated to take psychiatric drugs are included (Tegeler / Lehmann / Stockschläder 1980); people who stop taking psychiatric drugs on their own initiative and who live without them do not figure in such studies and their experiences are ignored.

The fact that no distinction is made between withdrawal problems such as receptor changes caused by the treatment, rebound effects or supersensitive reactions and relapse is another serious deficiency. Brigitte Woggon from the Zurich University Psychiatric Hospital, who favors psychiatric drugs, sees problems with the lack of differentiation made, even drugs are abruptly withdrawn, between withdrawal symptoms and the return of the original psychological symptoms:

> "Interestingly, in most studies on withdrawal no position is taken on possible withdrawal symptoms apparently because the studies are not set up to deal with these findings." (Woggon 1979, p. 46)

Nonetheless, doctors continue to refer to their studies and speak with a great deal of pathos of a sudden relapse if the drugs are stopped without their authorization, particularly in the case of lithium, antidepressants and neuroleptics. The situation is somewhat different in the case of carbamazepine and tranquilizers. In contrast to its use in preventing epileptic fits in neurology, carbamazepine is rarely used alone in psychiatry—its claimed antimanic effect is disputed anyway (Lerer et al. 1985)—and there have been practically no withdrawal studies. Partly as a consequence of decisions reached in the law courts on compensation, awarded because patients were insufficiently informed on the risk of dependency associated with tranquilizers, these substances have come to be seen as problematic by orthodox medicine.

### Tranquilizers

Taking tranquilizers involves a risk which should not be underestimated. The development of tolerance and rebound phenomena can occur after taking the psychiatric drugs for only a short time and in low dosage. Massive, life-endangering withdrawal symptoms, especially convulsions, can make stop-

ping psychiatric drugs a very dangerous undertaking. Often withdrawal symptoms are so pronounced that withdrawal can only be done under hospital care. After withdrawal, a rebound-like explosion of feelings that had been chemically repressed may be released.

Depression, in some cases long-lasting, as well as anxiety states or hallucinations and deliria may occur during withdrawal. A "fear of rebound" is a generally recognized withdrawal symptom associated with tranquilizers. These symptoms are associated with risks, not least the repeated prescription of psychiatric drugs, becoming a psychiatric "case," and the switch to even more risky psychiatric drugs such as antidepressants or neuroleptics.

Withdrawal symptoms can register in the central nervous system as EEG (electroencephalogram) disturbances, difficulty in concentrating, pressure headaches, generalized pain, nervousness, restlessness, disturbed sleep and disturbed perceptions such as an increased sensitivity to stimuli. According to many doctors, withdrawal symptoms such as insomnia and excitability can be traced to a reactive hyperactivity of receptors which have been chemically altered. Many speak of a "rebound insomnia." Sometimes this phenomenon continues for weeks or months until the molecular mechanisms have settled at a new tolerable level.

Various vegetative symptoms can also often be explained as unhealthy physiological rebound effects. These symptoms may last for weeks, making it difficult to interpret whether they are drug withdrawal symptoms or recurring symptoms of the underlying condition. Weight loss, hot-cold sensations, fever, and heart and circulatory problems often occur (the latter for example as a rapid heart beat, shortness of breath, constriction, a rapid pulse, dizziness and weakness). Breaking out in sweats is an accompanying symptom. Intestinal and stomach disorders are another common withdrawal symptom (diarrhea, nausea, loss of appetite). Stomach cramps also occur. Disorders affecting vision sometimes accompany these symptoms as well.

Withdrawal may also bring muscular and motor disturbances, for example, jitters and shaking, limb pain, back tension, an insecure gait, as well as alternating muscle contractions that may cause jerking and shaking.

Withdrawal symptoms occur at a rate of 30–50% even when withdrawal is gradual.

Especially in the case of benzodiazepine tranquilizers, the approaches to withdrawal studies are mixed. Some include reports on the (problematical) administration of carbamazepine, antidepressants or neuroleptics to suppress withdrawal symptoms, although they seldom include warnings of the additional toxic burden (e.g. Klein et al. 1994). The tranquilizers are frequently replaced on a long-term basis by other psychiatric drugs. All the same, in recent years several authors of such studies have spoken not just in favor of a gradual withdrawal of tranquilizers; they also observed good long-term results in avoiding relapses back to the original unproductive mechanisms of dealing with problems and in avoiding problems of new dependencies (Ashton 1987; Rickels et al. 1988). This was also true of people with varying diagnoses (Golombok et al. 1987) and even in the case of repeated relapses (Crouch / Robson / Hallstrom 1988). Even in the eyes of professionals self-help groups have proven to be effective (Tattersall / Hallstrom 1992), as has psychological support in learning non-psychopharmacological strategies (Ashton 1994) when they encouraged people to persevere, to actively confront the problems caused by the continuous use of tranquilizers (Bish et al. 1996) and when psychotherapeutic support was provided during withdrawal and in the ensuing months (Otto et al. 1993; Kaendler / Volk / Pflug 1996).

### Carbamazepine

Withdrawal symptoms associated with carbamazepine appear to be relatively minor. Nonetheless, in the few known controlled studies on withdrawal, a series of psychological, central nervous, vegetative and motor disturbances occurred.

L.A. Demers-Desrosiers and his colleagues from the Montreal Neurological Hospital and Clinic published two case studies of first-time occurrences of psychotic symptoms in two people who had been taking the anti-epileptic drug carbamazepine for three and four years respectively to treat epilepsy and had withdrawn slowly (Demers-Desrosiers / Nestoros / Vaillancourt 1978). The British neurologist John Duncan, together with his colleagues as the Centre for Epilepsy in Chalfont/England, carried out a systematic withdrawal study of the drug carbamazepine. A double-blind study of 24 adults with epilepsy diagnoses was carried out in which some subjects withdrew slowly and some abruptly. According to the 1988 report, although all of them

continued taking a second anti-epileptic drug, 23 persons still showed withdrawal symptoms, including paranoia and confusion in one patient, a lack of energy in four patients, depression and/or irritability in five, tension and feelings of depersonalization in two, states of anxiety in three, a weak memory and loss of concentration in two, insomnia and/or headaches in four, loss of appetite in three, muscular pain in five, jerking in four, muscle jitters and/or an insecure gait in two. Some of these disturbances were accompanied by low blood pressure and a racing heartbeat. According to the neurologists, in comparison with the chemically closely-related tricyclic antidepressants, the carbamazepine withdrawal symptoms were not nearly as pronounced as was found in the first study (Duncan / Shorvon / Trimble 1988).

The biggest withdrawal problem, especially in people taking this substance for epileptic attacks or to suppress psychological states—possibly together with neuroleptics or lithium—, lies in the danger of the recurrence or sudden new occurrence of epileptic attacks.

### Lithium

In the case of lithium the usual vegetative withdrawal symptoms seem not to occur. However, depending on dose, duration of administration, as well as the patient's physical and psychological state, rebound phenomena and states of confusion can be expected which bring with them the danger of renewed hospitalization. Therefore, a gradual approach is recommended when stopping this psychiatric drug.

In 1979 D.G. Wilkinson, a psychiatrist at Bethlem Royal Hospital in London, was the first to report on withdrawal problems, citing states of confusion (Wilkinson 1979). Other authors have since confirmed Wilkinson's statements: a state of fear increases, irritability and an unstable state as well as the rebound phenomena of mania and psychosis result. Y.D. Lapierre and his colleagues at the Royal Ottawa Hospital took into consideration what they found to be an unexpectedly high "relapse" rate after the withdrawal of lithium and came to the following conclusion:

> "Such quick relapses suggest some sort of exaggerated neurochemical or physiological response to abrupt withdrawal. (…) Such rebound pheno-

> mena have also been observed after withdrawal from benzodiazepines." (Lapierre / Gagnon / Kokkinidis 1980, p. 863)

J.R. King and R.P. Hullin from the High Roads Hospital in Ilkley, England found in 1983 that the relatively high number of cases of anxiety states and other withdrawal reactions (irritability and an increase of strong emotions) are obviously withdrawal symptoms and rebound phenomena, and that their occurrence two or three days after withdrawal is characteristic of the delayed reaction typical of withdrawal from medication (King / Hullin 1983). The excessive increase of "relapses" within a few weeks after withdrawal from lithium, which has often been described, was addressed by Janet Lawrence of McLean Hospital in Belmont, Massachusetts, who said:

> "Such withdrawal might trigger psychosis by altering neurotransmitter balance with previously unipolar depressed patients relapsing into depression and bipolar patients into mania. This is of especial interest as it implies that lithium treatment may adversely affect the natural history of the underlying disease in an analogous fashion to the rebound psychosis postulated for antipsychotics by Chouinard and associates." (Lawrence 1985, pp. 873f.)

More recent withdrawal studies with lithium do not show uniform results. In general, it was observed that gradual withdrawal reduces the risk of the recurrence of the same depressive and manic moods which had led to psychiatric treatment and the administration of lithium in the first place (Mander / Loudon 1988; Faedda et al. 1993; Suppes et al. 1993). One study showed that after getting through the first three months following withdrawal, relapses were no more frequent than in those persons who continued to take lithium (Mander 1986). Some psychiatrists expect the occurrence of a withdrawal rebound, in other words an increased risk of relapse with temporarily more severe "symptoms" especially if the psychiatric drugs are stopped abruptly (Hunt / Bruce-Jones / Silverstone 1992; Schou 1993). Others found no such rebound (Sashidharan / McGuire 1983) or at least only a partial one (Klein et al. 1991). The claim that lithium prevents depressive or manic attacks is not undisputed in orthodox medicine ("Lithium" 1969). Publications continue to appear on "individual cases" where the claimed protection provided by lithi-

um proved to be an illusion (Prien et al. 1984), as well as on considerable rates of relapse (Lusznat / Murphy / Nunn 1988) and several cases of suicide including while under the influence of lithium (Schou / Weeke 1988).

### Antidepressants

In 1982 Dennis Charney and his colleagues at University of New Haven, Connecticut described the typical reaction to the withdrawal of antidepressants:

> "Symptoms first appeared approximately 48 hours after the last dose and included excessive anxiety, restlessness, and autonomic symptoms such as diaphoresis, diarrhoea, hot and cold flushes, and piloerection *(goose-bumps)*. In addition, the development of hypomanic and manic symptoms has been observed to occur during the first week after cessation of chronic TAD *(tricyclic antidepressants)* treatment." (Charney et al. 1982, p. 377)

When stopping antidepressants, more psychological withdrawal symptoms can be expected, for instance apathy, social withdrawal, a depressed mood, but also panic attacks, aggression, or delirium. Withdrawal psychoses can also occur.

Besides stomach and intestinal tract disturbances, insomnia probably represents the second most common withdrawal problem, according to Steven Dilsaver and John Greden of the University Hospital in Columbus, Ohio. Further symptoms of the central nervous system include headache, restlessness, hyperactivity, insomnia, a dazed state, apparently paradoxical improvements, tiredness, jittering and nervousness as well as disruptive dreams, for example nightmares. According to the German psychiatrists Otto Benkert und Hanns Hippius, severe cramping, which is known to occur particularly with the withdrawal of tranquilizers, can also develop when antidepressants are withdrawn, especially if the dosage had been high:

> "Favorable conditions for the development of severe cramping include beginning with a high dosage, rapidly increasing the dosage, or a rapid withdrawal from a high dosage. A sudden withdrawal from antidepressants after years of use should always be avoided." (Benkert / Hippius 1980, p. 34)

The occurrence of vegetative withdrawal symptoms has been observed in cases of both gradual and abrupt withdrawal. Intestinal tract and stomach

disorders, for example diarrhea, stomach ache, tenesmus (painful, lasting cramp-like urges to release the bowels or bladder), and associated nausea, vomiting and loss of appetite, are the most common symptoms. In addition, the following should also be mentioned: cold-sweats, weakness, an increase in libido, a racing heartbeat, abnormal heart rhythms, as well as lowered blood pressure and extreme anxiety associated with physical collapse.

Motor disturbances are a more rare. Some individual reports have noted also muscle pain, Parkinson-like symptoms such as slowed movement, cog wheel phenomenon, as well as restlessness and withdrawal dyskinesia.

In 1984 Dilsaver und Greden provided an overview of the available literature on withdrawal. They reached the conclusion that withdrawal symptoms very frequently appear, that is, in 21% to 55% of adults. Other authors reported a rate of 80% (Dilsaver / Greden 1984).

Withdrawal symptoms that include a worsening of the psychological state must carefully be separated from a return or relapse to the original psychological problems that led to treatment in the first place, according to Janet Lawrence at the McLean Hospital in Belmont, Massachusetts. The two are not easy to distinguish. A "relapse" occurs three to 15 weeks after withdrawal, whereas withdrawal symptoms occur within two weeks and usually subside within a week or two (Lawrence 1985). The problems associated with withdrawal from antidepressants are as old as antidepressants themselves. Already in 1960, the antidepressant pioneer Roland Kuhn wrote that withdrawal symptoms:

> "… can look really bad, under certain circumstances bringing on severe headaches, profuse sweating, tachycardia attacks *(racing heart beat)*, sometimes also vomiting, all of which disappear within a half hour of resuming the medication. This is a phenomenon that looks very similar to the 'withdrawal symptoms' of toxicomania *(drug dependence)*…" (Kuhn 1960, p. 248)

Rudolf Degkwitz, former president of the German Association for Psychiatry and Neurology, compared the withdrawal symptoms of psycholeptics (neuroleptics and antidepressants) with those of alcaloids, the group of substances to which morphine among others, belongs. In addition, sleeping pills are considered addictive, and it is known that withdrawal from them can bring on severe, even life-threatening, cramping. According to Degkwitz:

> "The reduction or withdrawal from psycholeptics leads, as described above, to considerable withdrawal symptoms that cannot be distinguished from those symptoms occurring with the withdrawal of alcaloids and sleeping pills." (Degkwitz 1967, p. 161)

The development of tolerance and rebound phenomena even after only short-term usage in moderate doses as well as receptor changes may necessitate a gradual withdrawal. This counteracts the risk of withdrawal symptoms (which can last for several weeks) being confused with the recurrence of the original problems thus leading to renewed prescription of antidepressants and other psychiatric procedures. In those withdrawal studies which deal with antidepressants psychiatrists reported, among other things, relatively good prognoses in the time after withdrawal in older people (Cook et al. 1986), especially if the subjects had been symptom-free for 16 to 20 weeks (Prien / Kupfer 1986). They do not, however, deal with the question of what use that statement is to people who despite or because of antidepressants are suffering from depressive symptoms. Other studies saw higher relapse rates after withdrawing all kinds of antidepressants (Misri / Sivertz 1991; Solyom / Solyom / Ledwidge 1991); but the suspicion that antidepressants lead rather to depression becoming chronic (Irle 1974, pp. 124f.) was not dealt with while the question of how to evaluate the effect of psychotherapy or self-help (which was not offered) was not even posed in the first place.

### Neuroleptics

Psychiatrists have reported the following psychological withdrawal symptoms: a depressed mood, fear, a desire to run away, and fits of crying. Because a reduced dosage may result in motor disturbances and emotional pain caused by the neuroleptics becoming more pronounced and/or particularly intense (due to the fact that the emotional numbing of the drugs has subsided), a temporary—but nonetheless serious—risk of suicide may arise during withdrawal.

Tension, restlessness, destructiveness, aggression, irritability, and excitability may develop into withdrawal psychoses and delirious states. Fritz Reimer, like Degkwitz a former President of the German Association for Psychiatry

and Neurology, concluded the following concerning the possibility of post-withdrawal delirium that may last several days:

> "The ultimate factor in the delirium syndrome is certain to be the psychoactive pharmaceuticals. On the surface, it appears to compare to the withdrawal delirium of the alcoholic." (Reimer 1965, pp. 446f.)

Some psychiatrists deliberately employ withdrawal and its effects to provoke a so-called therapeutic delirium, for example to stimulate those with a "numbed foundation" and to create new target syndromes for the neurolepsy, as they put it (cf. Lehmann 1996a, pp. 125f.).

Tardive dyskinesia, that is, muscle disorders that appear during treatment, withdrawal or thereafter and which are not treatable nor controllable, have in the past been deemed impairments resulting from treatment, and some victims have been successful in obtaining compensation for this. In 1977 George Simpson from the Psychiatric Institute in Orangeburg, New York was the first psychiatrist to warn that:

> "The potential of neuroleptics to produce dyskinesia, a serious complication, in a considerable number or patients would indicate that an attempt should be made to withdraw in every patient." (Simpson 1977, p. 6)

In the same year, Urban Ungerstedt und Tomas Ljungberg at the Karolinska Institute in Stockholm published results of studies in which rats were administered the conventional neuroleptic haloperidol and as a comparison the "atypical" clozapine[1]. They believe that "atypical" neuroleptics modify subtypes of specific dopamine-receptors, produce their supersensitivity and contribute to the risk of new, increasing, or chronically powerful psychoses of organic origin, which can be understood as "counterpart to tardive dyskinesia" (Ungerstedt / Ljungberg 1977, p. 199). Since then, medical journals have steadily published findings on supersensitivity, rebound and withdrawal psychoses (Chouinard et al. 1979, 1984; Chouinard / Jones 1980, 1982; Borison et al. 1988; Ekblom / Eriksson / Lindstroem 1984).

The frequent damage caused by typical neuroleptics like haloperidol arises from changes in dopamine-$D_2$-metabolism, observable as motor disturban-

1 Neuroleptic, marketed as Clopine, Clozapine, Clozaril

ces; the usual damage caused by "atypical" neuroleptics like clozapine, sertindole[1] or quetiapine[2] goes in the direction of changing the metabolism of special subtypes of dopamine-receptors, dopamine-$D_1$ and -$D_4$, seen as producing or increasing mid- and long-term psychotic syndromes of organic origin. Frank Tornatore and his colleagues at the University of Southern California School of Pharmacy in Los Angeles warned of the development of supersensitivity psychoses:

> "There is a worsening of the psychosis (delusions, hallucinations, suspiciousness) induced by Iong-term use of neuroleptic drugs. Typically, those who develop supersensitivity psychosis respond well initially to low or moderate doses of antipsychotics, but with time seem to require larger doses after each relapse and ultimately megadoses to control symptoms." (Tornatore et al. 1987, p. 44)

Supersensitivty should be understood as the result of an increased tolerance to the drugs, as they point out in an additional citation in the German translation of the book four years later: "Thus, a tolerance to the antipsychotic effect seems to develop." (Tornatore et al. 1991, p. 53)

Withdrawal symptoms related to the central nervous system are well known to psychiatrists. In 1960 psychiatrists at the University Clinic in Vienna published their initial observations on the effects of Melleril[3]:

> "What we noticed was that when medication was suddenly withdrawn, even after several months patients experienced insomnia and considerable restlessness as well as occasional states of pronounced excitability." (Hofmann / Kryspin-Exner 1960, p. 900)

Further symptoms in this area include headaches, restlessness, insomnia, nightmares, numbness and taste impairment.

Vegetative withdrawal symptoms that may occur include anorexia (or a lesser loss of appetite), binging, nausea, vomiting, gastritis, diarrhea, stomach

1 Neuroleptic, marketed as Serdolect

2 Neuroleptic, marketed as Seroquel

3 Neuroleptic, active ingredient thioridazine, marketed also as Aldazine, Mellaril, Rideril, Thioridazine

ache, colic, pronounced nasal discharge, sebaceous gland discharge, hot flashes, freezing, pronounced sweating, cardiovascular (i.e. heart and circulatory system) problems such as a racing heartbeat, dizziness and physical collapse. The dangers that proceed from the habituation of a vegetative state and a physical dependence on neuroleptics have been shown in a rabbit study by Helma Sommer and Jochen Quandt at the Psychiatric Clinic in Bernburg/Saale. Their observations were based on noted metabolic changes induced by chlorpromazine that caused a circulatory collapse after withdrawal from the neuroleptic, despite the fact that metabolism was in fact returning to normal. For six months, Sommer and Quandt administered neuroleptics to 20 rabbits. The four animals that had received the highest dosage (16.7 mg/kg) died after a brief fit of cramping:

> "At a dosage of 13.3 mg/kg of chlorpromazine, abrupt withdrawal led to a sudden death within 14 days, probably due to irreversibly blocked metabolic processes that stopped functioning (similar observations in human beings have been published in which death followed a brief stage of cramping)." (Sommer / Quandt 1970, p. 487)

Withdrawal from neuroleptics can cause various muscle and motor disturbances. Parkinson-like disorders are common, and muscle jerking or withdrawal dyskinesia and tongue atrophy often increases or is initiated.

Roy Lacoursiere and his colleagues at the Veterans Administration Hospital in Topeka, Kansas, have stated that the rate of withdrawal symptoms of all kinds is as high as 75%. The observed rate will depend on how closely subjects are observed, how well the withdrawal symptoms can be distinguished from the "mental illness," and how well the psychological condition of the subjects before treatment has been documented. Thus it is no surprise that some psychiatrists have published rates of 0% (Larcoursiere / Spohn / Thompson 1976). Up to 80% of patients experienced vegetative and especially stomach and intestinal problems with withdrawal (Greil / Schmidt 1988, 1989). There is very little evidence available on the rate of supersensitivity psychoses. In 1982 the pharmacologist Guy Chouinard and his colleague Barry Jones at the University Clinic in Montreal reported that they had found signs of supersensitivity psychoses among 30% of the 300 patients they studied, many of whom had not necessarily gone through an abrupt withdrawal

(Chouinard / Jones 1982). Degkwitz has repeatedly reported on withdrawal symptoms—not publicly, but in specialized journals:

> "We now know that it is extremely difficult, if not impossible, for many of the chronic patients to stop neuroleptics because of the unbearable withdrawal-symptoms." (Degkwitz / Luxenburger 1965, p. 175)

George Brooks, psychiatrist at the Vermont State Hospital in Waterbury said:

> "The severity of the withdrawal symptoms may mislead the clinician into thinking that he is observing a relapse of the patient's mental condition." (Brooks 1959, p. 932)

Medical opinion on continued administration of neuroleptics is split. In 1995 Patricia Gilbert and colleagues in the Psychiatric Department of the University of California in San Diego published a meta-analysis in which they looked at 66 studies conducted between 1958 and 1993 on almost 5600 persons. They summed up the problems of the continued administration of neuroleptics for the treating physician:

> "The issue of prolonged neuroleptic treatment in a patient with chronic schizophrenia places the clinician on the horns of a dilemma. Since neuroleptic treatment does not cure schizophrenia, a large majority of such patients need long-term treatment. At the same time, prolonged use of these drugs carries a high risk of adverse effects, including TD *(tardive dyskinesia)*. It is therefore recommended that continued prescription of antipsychotic drugs over a long period not be undertaken without adequate justification for both clinical and legal purposes. This may imply attempts at neuroleptic withdrawal. Drug withdrawal, however, is associated with a risk of psychotic relapse. To complicate matters further, a number of patients withdrawn from antipsychotic therapy do not experience relapse, at least over a short period, while some patients maintained on therapy do experience relapse. Thus, the clinician and the patient have to choose between two unwelcome risks: relapse and adverse effects of continued treatment." (Gilbert et al. 1995, p. 173)

Both psychotherapeutic treatment providers and biologically-oriented psychiatrists admit in internal discussions that they do not know whether neuroleptics

in individual cases actually help or cause damage. William Carpenter and Carol Tamminga from the Maryland Psychiatric Research Center in Baltimore, who provided the opportunity of a controlled withdrawal, came to the conclusion:

> "Although adverse events, such as suicide, dissatisfied patients or relatives, loss of job, deteriorating course, and brain abnormalities, can all be observed during drug withdrawal, each of these is also commonly encountered in the clinical care of medicated patients!" (Carpenter / Tamminga 1995, p. 193)

Hanfried Helmchen from the University Hospital in Berlin, a psychiatrist who can be seen as a strong supporter of long-term neuroleptic treatment, expressed himself back in the 1980s in a discussion among colleagues in a notably skeptical tone:

> "When looking back on the 25 years since neuroleptics have been made available to us, it can be concluded that indication predicators for a neuroleptic treatment have not been found but are essential. There are clearly patients who remain symptom-free even without neuroleptics, and there are those who continue to display symptoms while gaining no benefit from neuroleptic therapy and who become even more handicapped." (Helmchen 1983)

His colleague Karl Leonhard from the Psychiatric Department of the Humboldt-University in Berlin differentiated what he determined to be "nuclear schizophrenias" versus so-called cycloid psychoses (for example anxiety psychoses, confusion psychoses, happiness psychoses or motility psychoses with a catatonic-like state). Based on this, he considered it malpractice if prescribed neuroleptics are not soon thereafter withdrawn again:

> "Today I unfortunately see very many cases of cycloid psychosis that remain in a toxic, pathological state because of constant medication, but which would be perfectly normal without medication. If one could prevent the development of further phases of psychosis with constant medication, then this practice would be justified, but unfortunately that is not the case. Thus those patients who would be healthy for extended periods, or perhaps forever, are held in a permanently toxic state…" (Leonhard 1980, p. 3)

Several further factors should make people think twice before allowing themselves to be pressured by doctors' and psychiatrists' frequent insistence on a long-term administration of neuroleptics:

- The duration of hospitalization is not shortened when neuroleptics are taken (Hartlage 1965); in fact people are discharged earlier if they take none at all (Epstein / Morgan / Reynolds 1962).
- The state of older people taking neuroleptics is worse in comparison with those who are free of psychiatric drugs (Tune 1992).
- The aim for rapid success—quiescence and management—is also considered by psychotherapists as absolutely misplaced: what is important is personal development as well a change in the kind of family relationships which lead to illness and mental disturbance (Haley 1989).
- Neuroleptics probably suppress "self-healing tendencies" (Ernst 1954, p. 588) and prevent "cure" (Stierlin / Wynne / Wirsching 1985; Harding / Zubin / Strauss 1987). Those who weather their crises without psychotropic drugs have better medium and long-term prognoses (Goldberg / Klerman / Cole 1965; Hogarty / Goldberg / Baltimore Collaborative Study Group 1974; May / Goldberg 1978; Wehde 1991, pp. 44ff.), and are less frequently "psychotic" than those treated with psychiatric drugs and land far less often in psychiatric wards (Young / Meltzer 1980; Heinrichs / Carpenter 1985).
- "Relapses" under neuroleptics result in longer periods of hospitalization than "relapses" which occur when no psychiatric drugs are used (Gardos / Cole 1976).
- Neuroleptics contribute nothing to long-term rehabilitation (Niskanen / Achté 1972), generally inhibit everyday "functioning" (Schooler et al. 1967), and often lead to social deterioration (Müller / Günther / Lohmeyer 1986).

Uninformed, isolated and therefore defenseless individuals are understandably afraid to be sent back to the loony-bin and to be forcibly treated with neuroleptics, so they go on taking neuroleptics at the insistence of "their" psychiatrists or their families. It is particularly important that this group of "users" of psychiatric drugs be exposed to the alternative experiences of others.

*Translation from the German by Christina White*