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Teaching withdrawal of antipsychotics and antidepressants to professionals and recipients

Summary: Problems at withdrawal from neuroleptics ("antipsychotics") and antidepressants are well known from animal research and experiments in normal subjects. The existence of dependence is denied from pharmaceutical industry and mainstream medicine. People withdraw with or without problems. Older studies did not distinguish between withdrawal problems and so-called true relapse. Meanwhile there is some knowledge about reducing risks in withdrawal, especially on the side of ex-users and survivors of psychiatry. But there is still a mass of open questions for physicians and natural healers, carers, therapists, social workers, jurists, pharmacologist, relatives and experts by experience. Until today, teaching withdrawal is passing knowledge and experiences, but also teaching the open questions and staying involved decision processes of future research.

Introduction

Teaching withdrawal of neuroleptics and antidepressants to professionals and recipients is not easy. It seems easy for professionals to persuade people in emotional distress to take these drugs. Sometimes they use simple power and administer them by the use of violence. Sometimes they praise their drugs as harmless or alternativeless helpers in painful emotional states, (mis)using the trust they have as physicians. Sometimes people in emotional distress suffer so much that they would do nearly anything or they do not care about the risks of the chemical substances they swallow only to get some relief. And sometimes individuals make the experience that they cannot exist in their current life-conditions without taking these psychiatric drugs. The discussion always does into the direction of intake, not of stop. The impression might be there, that on the side of psychiatric mainstream professionals there is no knowledge about withdrawal problems, they had to be thought. This is wrong.

It was evident since the introduction of neuroleptics and antidepressants and into the "market", that withdrawal of both drug groups can be a serious problem. And with the time, that there are receptor changes, supersensitivity problems, rebound problems, tolerance, physical and psychic dependence. It is also well known since decades, that the benefit of longer-term use of psychiatric drugs, basis of the development of drug dependence, has to be questioned. Following his long-term study in 1972, for example, psychiatrist Manfred Bleuler from Switzerland saw no indication of an improved course or outcome in patients following long-term treatment with neuroleptics. In fact, the opposite seemed to be the case, he wrote:

> "Not one single patient who – healed or improved – lived outside of the clinic over years or permanently, has ever taken long-term drugs. The assumption, that the majority of improved schizophrenics would stay improved on the long term only under the influence of neuroleptic drugs, is an error. First of all, it is an error to assume that relapses after remissions could be avoided by neuroleptic drugs. There are many cases of permanent remissions and there are many cases of relapses under the influence of neuroleptics" (p. 366).

By the way, the difference of neuroleptics and antidepressants is hard to define exactly. No standard criteria exist for the assignment of a single psychiatric drug to a group of substances. The classification of a drug can depend on its pharmaceutical basic schedule, biochemical mechanism, produced effects or the administrator's subjective intention. For example, oxypertine was marketed in the USA as an antidepressant, in the UK as a neuroleptic, flupentixol in the UK as a neuroleptic and antidepressant.

Animal Research and Experiments in Normal Subjects

It is known from animal studies that the sudden withdrawal of neuroleptics can be life threatening. For six months, Helma Sommer and Jochen Quandt (1970), two neurologists in past GDR, tested the neuroleptic prototype chlorpromazine at rabbits. After those animals that had received the highest dosage died after a brief fit of cramping (probably due to irreversibly blocked metabolic processes), they wrote that similar observations in human beings have been published in which death followed a brief stage of cramping. In a self-experiment with a ten-day regimen of thioridazine and following three weeks of administering chlorprothixene to tuberculosis patients who were given it because of its antimicrobial effect, the sudden withdrawal caused, in all test persons, more or less severe reactions of lack of concentration and nervousness about their depressed mood, diarrhoea and lack of sleep, even leading to manic states which lasted up to two weeks (Hollister et al., 1960; Degkwitz, 1964).

Self-experiments with antidepressants showed similar experiences. For her dissertation, the German Gisela Rautmann took the antidepressant prototype imipramine for three weeks, and reported about her withdrawal symptoms which occurred already at small efforts on day 3 after stopping the drug:

> "The subject of the trial responded with dizziness and dry mucous membranes of the mouth. Hot and cold feelings alternated with physical efforts rapidly. (...) During driving her car she recently felt ill. She suffered from sweating; the tip of the nose remained unchanged cold" (1964, p. 29).

Other people, who undertook "long-term" self-experiments (1-3 weeks), reported about loss of concentration, weight and appetite, restlessness, jumpiness, bitter taste in the mouth, sweating and hot flashes, nausea, enlarged pupils, enhanced intestine activity and especially sleeping problems. Rautmann's colleague Gerhard Seuwen (1964) took amitriptyline for two weeks, and after stopping the drug, in his dissertation he reports about heavy pain in the heart area, sweating, headaches, sleeping disorders and a continuously enhancing state of fear and restlessness, which only declined after ten days (pp. 27-28).

Denial of Dependence

Over decades, the existence of dependence is denied by the pharmaceutical industry and mainstream medicine (although there are some exceptions to this), as it was denied over decades in benzodiazepines. Representative for many of his colleagues, the German pharmacologist Gerd Glaeske (1989) justified the replacement of tranquillizers through neuroleptics and antidepressants, "because no problem of dependence can be expected in these drugs." Discussing dependence from neuroleptics and antidepressants often leads to reflex-like responses by physicians, that these drugs do not produce addiction, that only benzodiazepines produce dependence, that neuroleptics and antidepressants might have been misused, that, as the Royal College of Psychiatrists in the United Kingdom stated,

> "...there is potentially overprescribing of these medications, particularly SSRIs, where there is no particularly strong clinical indication…" (quoted after: BMA, 2015, p. 25).

If at all a discussion starts, as initiated from the British Medical Association in 2014, then perhaps it is discussed if antidepressants might produce dependence; the potential of neuroleptics to produce dependence keeps factored out (BMA, 2015) or starts to be addressed with shudder and a delay of 65 years (Fachausschuss, 2016). In their guide for drug-dependent people and their relatives, the physician Wolfgang Poser, the psychiatrist Sigrid Poser and the social economist Dietrich Roscher describe the typical definition of dependence:

> "There is drug dependence when a continuous medication or even increasing drug doses is required to suppress the symptoms sufficiently and to control the condition, and / or drugs-holidays lead to enforced occurrence of the initial and additional complaints. Signs of chronic intoxication become noticeable" (1985, p. 34).

Referring to Heribert Czerwenka-Wenkstetten and colleagues from the University Clinic of Vienna, the term addiction is used in compulsive us of substance

> "… for the adjustment of an unbearable state of body and mind, if necessary by rolling back all other aims and by overriding each hindering circumstances. As criterion of an addictive drug we see primarily the occurrence of withdrawal symptoms at withdrawal as well as the patient's inability to waive out of its own power, secondarily the euphoric effect and the mostly existing need for dose escalation to maintain the effect" (1965, p. 1013).

Dependency and tolerance building is a dark area not least because psychiatrists strictly deny its existence in public. In their own magazines they speak differently, as the example of Rudolf Degkwitz, a former President of the German Association for Psychiatry and Neurology, and his colleague Otto Luxenburger shows, which stated already more than half a century ago:

> "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" (1965, p. 175).

If there is no diagnose of dependence, then there is no possibility to get refunds from health assurance companies for physicians, who might settle up their work, and there is no possibility for patients who got dependent from neuroleptics and antidepressants to receive rehabilitation facilities, therapy and compensation for damaged people. Drug companies and medical practitioners who do

not inform about the risk of these drugs seem protected against criminal prosecution. No diagnose of dependence also means no awareness about the problem, no education about this danger, no information, no warning of the patients. They let them run into their undoing.

Withdrawal Symptoms in Neuroleptics

In principle, all kinds of phenomena of irritation can occur in the withdrawal of neuroleptics, including states of fear and confusion, hallucinations, withdrawal psychoses, sleep disorders, breaking out in a sweat, and much more. In their advertisement for Risperdal, Janssen Pharmaceutica Inc. / Smith Kline Beecham mentioned a "withdrawal syndrome" (1996), but without further description.

Psychological withdrawal symptoms like tension, restlessness, destructiveness, aggression, irritability, and excitability, as well as outbreaks of sweating, racing heartbeats, inner restlessness, vomiting, and diarrhoea, can trigger massive anxiety and may also develop into withdrawal psychoses and delirious states. In 1963, Helmut Selbach of the Psychiatric University Hospital Berlin described the state of shock caused by the sudden withdrawal:

> "After cold turkey withdrawal from a high dose of neuroleptics (which produced extrapyramidal symptoms), extreme euphoria accompanied by a rapid pulse ('choc en retour'*[backfire]*) can be caused solely by the withdrawal, in contrast to the previous affective indifference" (p. 67).

Fritz Reimer, 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" (1965, pp. 446-447).

His American colleague George Brooks at the Vermont State Hospital in Waterbury reported "severe withdrawal reactions indistinguishable clinically from a moderate withdrawal reaction following long-term ingestion of morphine" (1959, p. 931). Degkwitz and Luxenburger compared the withdrawal symptoms of psycholeptics (neuroleptics and antidepressants) with those from alkaloids; morphine belongs to this substance group. Sleeping pills considered working addictive, and it is well known, that their withdrawal can go along with life-dangerous convulsions. The both psychiatrists wrote:

> "As described above, the reduction or withdrawal from psycholeptics leads to considerable withdrawal symptoms that cannot be distinguished from those symptoms occurring with the withdrawal of alkaloids and sleeping" (1965, p. 161).

Roy Lacoursiere and his colleagues at the Veterans Administration Hospital in Topeka, Kansas, explained how to distinguish withdrawal symptoms such as insomnia and restlessness from the original psychiatric symptoms. They optimistically assumed that the withdrawal symptoms subside more quickly; these

> "... (1) tend to occur earlier after drug withdrawal than schizophrenic exacerbation, (2) may be accompanied by other medical withdrawal symptoms, and (3) usually clear up spontaneously or with supportive treatment within a few days" (1976, p. 292).

There are many reports in the medical literature about central nervous system withdrawal symptoms, such as headaches, insomnia, nightmare, feelings of numbness, and disturbed taste, even convulsions. The vegetative withdrawal symptoms affect organ systems and functions and include feeling hot or cold, neuroleptic malignant syndrome (a syndrome with fever, muscle stiffness, impaired consciousness), dizziness, fainting, binging, pronounced sweating, heavy nasal discharge, excessive mucous and saliva secretion, anorexia (or a lesser loss of appetite), ravenous appetite, diarrhoea, gastritis, stomach ache, colic, nausea, vomiting, pronounced nasal discharge, sebaceous gland discharge, cardiovascular (i.e. heart and circulatory system) problems such as a racing heartbeat and physical collapse. The symptoms can last in some cases for months. Thomas Gualtieri and colleagues from the North Carolina Neuropsychiatry Clinics in Chapel Hill described the vegetative withdrawal symptoms as anorexia, nausea and vomiting – symptoms that occurred during the reduction of neuroleptics or within two weeks after their complete cessation for the first time and could not be attributed to other causes, for example, viral diseases or food poisoning (1984, p. 21).

Withdrawal from neuroleptics can also cause various muscle and motor disturbances, so-called withdrawal dyskinesias. Known motor symptoms are: the inability to move, increased or initial tremor, joint pain, inner agitation, hyperkinesia and dystonia such as tongue-throat syndrome. Parkinsonian disturbances caused by neuroleptics occur sometimes more frequently; neuroleptics not only trigger Parkinsonian disorders – symptoms of the brain disorder resulting from the use of the neuroleptics – but also suppress their expression. Mental problems, which appear as a reaction to the muscular disturbance, are often interpreted as a relapse.

The withdrawal symptoms can lead relatives and physicians to believe that patients are suffering relapses. Brooks was concerned that the severity of the withdrawal symptoms may mislead the clinician into thinking that he is observing a relapse of the patients' mental condition (1959, p. 932). The patients themselves may believe in the need for maintenance treatment with neuroleptics. Degkwitz commented on such secondary dependence:

> "Such patients do not increase the dose but believe that they can no longer exist without the 'crutch' of the psychotropic. This is clearly not an addiction, but a medication dependence resulting from the patient's own insecurity" (1967, p. 162).

Similarly, the Swedish physician Lars Martensson looked at the mental consequences for the people around the patients:

> "Neuroleptic drugs induce specific changes in the brain that make a person more psychosis-prone. It is like having a psychosis-inducing agent built into the brain. The effect of neuroleptic drugs may subside more or less with time if the drug is discontinued. Buy by then it may be too late. Because of psychotic symptoms, which are after effects of the drug, the conclusion has already been reached: 'He needs the drugs.' The trap has become a fact" (1998, p. 107).

The confusion can have fatal consequences, since withdrawal symptoms may be warning signs of permanent damage. The sudden occurrence of psychotic symptoms when reducing dosage could be an indication of developing supersensitivity psychoses, which then become chronic following the further administration of neuroleptics.

Receptor-changes, Supersensitivity, Tolerance

Neuroleptic effects consist mainly in the disruption of the nerve impulse transfer with dopamine. The subsequent withdrawal problems are due to the changes in the nerve conduction system, a natural reaction to the disturbed nerve impulse transmission, and can trigger rebound, supersensitivity and withdrawal psychoses. Animal experiments have demonstrated that continuous administration of neuroleptics can lead to supersensitisation of the dopamine receptors, dopamine turnover and tardive dyskinesia. In other words, the animals have mobility impairments during the course of administration of neuroleptics, on their withdrawal, or afterwards. The pharmacologist Guy Chouinard and his colleague Barry Jones at the University Clinic in Montreal made a lot of research and concluded:

> "The authors suggest that dopaminergic supersensitivity also occurs in the mesolimbic region after chronic neuroleptic exposure, resulting in the development of a supersensitivity psychosis. (...) An implication of neuroleptic-induced mesolimbic supersensitivity is that the tendency toward psychotic relapse in such patients is determined by more than just the normal course of the illness" (1980, p. 16).

Kenneth Davis and Gordon Rosenberg of the Veterans Administration Hospital in Palo Alto, California, tested fluphenazine and summarized the results of their study in *Biological Psychiatry*:

> "Long-term administration of antipsychotic drugs to animals induces super-sensitive mesolimbic [referring to nerve tracts from the midbrain to the cortex – P.L.] postsynaptic dopamine receptors. It is possible that a similar process can occur in man. Following a reduction in the dose of antipsychotic medications, or their complete discontinuation, mesolimbic dopamine receptor supersensitivity could be reflected in rapid relapse of schizophrenic patients, the development of schizophrenic symptoms in patients with no prior history of schizophrenia, or in the necessity for ever-increasing doses of long-acting depot fluphenazine to maintain a remission" (1979, p. 699).

It has been known since the 1950s that neuroleptics can lead to tolerance to the so-called antipsychotic effects because of experience with chlorpromazine. In 1958, Stefan Hift und Hans Hoff of the University Hospital of Vienna explained:

> "A further key factor is the speed at which tolerance to the substance develops, which then becomes less and less efficacious, as is the case with chlorpromazine" (p. 1046).

Development of tolerance cannot be avoided (Meyer, 1953, p. 1098). The dose has to be constantly increased to achieve a continuous effect; this is an indication of the dependence potential of psychiatric drugs. Tolerance and occurs mostly with low potency neuroleptics and also at relatively low doses (Haase, 1982, p. 214), including in non-psychiatric uses (Broglie & Jörgensen, 1954). As a reaction to the neuroleptic blockade, within a few weeks or months the dopamine receptors form additional receptors, a response known as up-regulation.

As a result of the increased receptivity to psychotic reactions, supersensitivity psychoses, also known as breakthrough psychosis, can occur, and finally lead to tardive psychoses. 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 long-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" (1987, p. 44).
>
> "Thus, a tolerance to the antipsychotic effect seems to develop" (1991, p. 53).

The frequent damage caused by typical neuroleptics like haloperidol arises from changes in dopamine-D_2-metabolism, observable as motor disturbances; the usual damage caused by "atypical" neuroleptics like clozapine, sertindole or quetiapine 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.

In 1977, Urban Ungerstedt and 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. 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" (p. 199). Since then, medical journals have steadily published findings on supersensitivity, rebound and withdrawal psychoses. In particularly clozapine, the prototype of "atypical" neuroleptics can cause irreversible psychoses (Chouinard & Jones, 1980, 1982; Ekblom et al., 1984; Borison et al., 1988).

Withdrawal Symptoms in Antidepressants

When stopping antidepressants, massive withdrawal symptoms can be expected, they are rather similar as in neuroleptics. The longer an antidepressant has been taken and the shorter its half-life is, the more likely withdrawal symptoms can be expected, for example, gastrointestinal symptoms with or without anxiety, sleep disturbance, Parkinsonian disorders, convulsions, paradoxical activation, aggression, or worsening of the underlying depression. Dilsaver and Greden clarified, that anxiety, agitation or impending panic are common withdrawal symptoms in antidepressants (1984a, p. 240). The *British National Formulary* listed more withdrawal symptoms:

> "Gastrointestinal symptoms of nausea, vomiting, and anorexia, accompanied by headache, giddiness, 'chills', and insomnia, and sometimes by hypomania *(elevated mood below mania)*, panic-anxiety, and extreme motor restlessness may occur if an antidepressant (particularly an MAOI) is stopped suddenly after regular administration for 8 weeks or more" ("BNF", 2008, p. 205).

In 2012, the register added movement disorders, muscle pain and manias and noted:

> "Withdrawal effects may occur within 5 days of stopping treatment with antidepressant drugs; they are usually mild and self-limiting, but in some cases may be severe. Drugs with a shorter half-life, such as paroxetine and venlafaxine, are associated with a higher risk of withdrawal symptoms" (2012, p. 243).

In the case of newer antidepressants such as the "selective" serotonin-reuptake-inhibitors (SSRIs), for example paroxetine, and the serotonin-noradrenalin reuptake-inhibitors (SNRI), for example venlafaxine, a special withdrawal syndrome has to be expected:

> "Gastrointestinal disturbances, headache, anxiety, dizziness, paraesthesia *(sensation of tingling, burning, pricking, or numbness of a person's skin)*, electric shock sensations in the head, neck, and spine, tinnitus, sleep disturbances, fatigue, influenza-like symptoms, and sweating are the most common features of abrupt withdrawal of an SSRI or marked reduction of the dose; palpitation and visual disturbances can occur less commonly" (ibid., p. 250).

Receptor-changes, Supersensitivity, Tolerance at Antidepressants

Rebound phenomena and receptor changes are assumed as reasons for withdrawal symptoms in antidepressants. Eventually a sub- or supersensitivity of the postsynaptic receptors might have developed in the course of the drug administration. After the withdrawal, the altered sensitivity can trigger a relatively abrupt change of the neurotransmitters' effect – into the opposite direction (Dilsaver & Greden, 1984b, 1986; Mirin et al., 1981). Dennis Charney and colleagues from the Psychiatric University Clinic New Haven, Connecticut, discussed different models of receptor changes resulting from continued administration of antidepressants, which could cause the withdrawal symptoms (1982). They also addressed rebound phenomena, which where proved in the nervous system of animals, for example, in rats and bear macaques after the administration of antidepressants (Sugrue, 1980; Svensson & Usdin, 1979) and made responsible for fear reactions (Redmond & Huang, 1979). With remarkable similarity such symptoms are found also in opiate withdrawal in humans.

In the early 1970s, physicians expressed the suspicion that antidepressants lead to chronic depression (Irle, 1974, pp. 124-125). Now Paul Andrews (2011) and his team at the Department of Psychology, Neuroscience & Behaviour at the McMaster University in Hamilton, Ontario (Canada), are finding that synthetic antidepressants interfere with the brain's natural self-regulation of serotonin and other neurotransmitters and the brain can overcorrect once the medication is suspended. Thus, depression would be triggered, Andrews explains:

> "We found that the more these drugs affect serotonin and other neurotransmitters in your brain – and that's what they're supposed to do – the greater your risk of relapse once you stop taking them. (...) All these drugs do reduce symptoms, probably to some degree, in the short-term. The trick is what happens in the long term. Our results suggest that when you try to go off the drugs, depression will bounce back. This can leave people stuck in a cycle where they need to keep taking anti-depressants to prevent a return of symptoms" (quoted after "Patients", 2011).

Andrews and colleagues conclude that it is important to inform patients about the risk of dependence before the administration of ADMs (antidepressant medications):

> "Drugs that promote the risk of relapse or withdrawal upon discontinuation can cause dependence on the drug to prevent the return of symptoms. Consequently, such drugs must be managed carefully and patients must provide informed consent for their use. ADMs are sometimes prescribed to people with alcohol or illicit drug dependencies, because the use of such substances to medicate feelings of anxiety and depression is thought to play a role in the dependency. Ironically, the use of ADMs to help people wean off such substances might merely replace one dependency with another" (p. 15).

The reason for this dependence lays in the down-regulation of the serotonin and noradrenalin receptors as a reaction to the artificial levels of transmitters in the synapses caused by the antidepressants; the receptors become insensitive and degenerate. In 2012, Andrews and colleagues explained once more:

> "It is a principle of evolutionary medicine that the disruption of evolved adaptions will degrade biological functioning. Because serotonin regulates many adaptive processes, antidepressants could have many adverse health effects. For instance, while antidepressants are modestly effective in reducing depressive symptoms, they increase the brain's susceptibility to future episodes after they have been discontinued. Contrary to a widely held belief in psychiatry, studies that purport to show that antidepressants promote neurogenesis are flawed because they all use a method that cannot, by itself, distinguish between neurogenesis and neuronal death. In fact, antidepressants cause neuronal damage and mature neurons to revert to an immature state, both of which may explain why antidepressants also cause neurons to undergo apoptosis (programmed death)."

Referring to the SSRIs, the Swiss physician and psychotherapist Marc Rufer warned:

> "In the long-term, the effect of serotonin is weakened. If the serotonin deficiency hypothesis of depression were correct, then the SSRI should cause very severe depression" (1995, p. 144).

Even with antidepressants a development of tolerance that indicates a processes of dependence is reported (Moldawsky, 1985; Tornatore et al., 1991, p. 108). At the example of Tofranil (imipramine), Selbach recommended in 1960 to solve the problem of tolerance building – without further ado with the administration of electroshocks:

> "The effectiveness of Tofranil may decrease with increasing number of depressive episodes. It is known that in the course of life the amplitude will shallow, but the ill system often freezes for therapy resistance, and the ability to spontaneous or therapeutically provoked counter-regulation loses or is greatly reduced. Here often only one or a few electroshocks can bring a relief of the counter-regulatory insufficiency, often with a strikingly fast homeostasis. Well, within seconds the seizure causes the same with extreme amplitude what Tofranil brings unspecifically with stretched and flat amplitude over a much longer time, but milder and more gently" (p . 267).

Internally, psychiatrists address extensively the problem that withdrawal symptoms in antidepressant correspond with the withdrawal symptoms of street drugs. Although antidepressants do not have euphoric or subjectively pleasant and calming effects, withdrawal symptoms, tolerance building and psychological dependence would justify the term "dependence," as it is used in other addictive substances. Some psychiatrists were convinced of the necessity to expand the term of dependence to a new type to come clear with the problem of dependence of neuroleptics and antidepressants. As early as 1960, the antidepressants pioneer Roland Kuhn wrote that withdrawal symptoms

> "… can look really turbulent, under certain circumstances bringing on severe headaches, profuse sweating, tachycardia attacks *(racing heart beat)*, sometimes going along with vomiting, and disappear within a half hour of resuming the

> medication. This is a phenomenon that looks at least very similar to the 'withdrawal symptoms' of toxicomania *(drug dependence)*..." (p. 248).

In the same year, psychiatrist Chaim Shatan (1966) asked, using an example of the discussion of a case-report with imipramine in the *Canadian Psychiatric Association Journal*, if the drug dependence definition of the World Health Organisation from 1950 should also be used for antidepressants, since there is development of tolerance, psychic, and bodily dependence, as well as characteristic withdrawal symptoms. According to Shatan, it is remarkable that the withdrawal reactions in course and symptomatology are nearly undistinguishable from those following average opiate dependence.

Raymond Battegay from the University Hospital Basel, Switzerland, explained that the term dependence needs to be expanded in order to clearly describe the problem of dependence on antidepressants and neuroleptics. Battegay referred to an article by John Kramer (1961) of the Psychiatric Hospital Glen Oaks in New York City on the subject of antidepressants and tranquillizers. In a comparison of withdrawal symptoms with those of tranquilizers such as meprobamate or chlordiazepoxide, conducted at the hospital,

> "... the main difference lay in the fact that following withdrawal of the neuroleptic substances no craving was triggered (...). Furthermore the two groups differed from each other whereby in the case of the neuroleptics, in contrast to meprobamate, there was no muscle twitching in the withdrawal phase and no epileptic fits. (...) In those patient we examined who had been treated over months, or in most cases years, neuroleptics caused physical dependence as the occasional occurrence of abstinence symptoms demonstrated, but not psychological dependency. Withdrawal symptoms or rather physical dependency appeared especially with combined administrations of neuroleptics and anti-Parkinson drugs. (...) Going by the experience of Kramer et al., who observed similar symptoms during the withdrawal of imipramine which had been administered at high does for over two months, the same criteria would apply also to antidepressant substances, so that one can speak of a neuroleptic/antidepressant type drug dependency" (1966, p. 555).

Frequency of Withdrawal Symptoms in Neuroleptics and Antidepressants

The medical and psychiatric literature includes widely varying information relating to the frequency of withdrawal symptoms associated with neuroleptics. According to diverse studies, up to 84% of the people withdrawing have vegetative, especially gastrointestinal, problems, 22% have withdrawal dyskinesias and 60% muscle pain (see Lehmann, 2016, pp. 421-455). Chouinard and Jones reported that they had found signs of supersensitivity psychoses among 30% of the 300 patients they studied, many of which had not necessarily gone through an abrupt withdrawal (1982).

Withdrawal symptoms are to be expected for all neuroleptics. Low potency neuroleptics tend to cause stronger vegetative effects, so withdrawal symptoms are expected here first. Since all neuroleptics can potentially cause receptor changes, there is always the possibility, if not the certainty, that withdrawal symptoms can occur. In their withdrawal study, Lacoursiere and colleagues could not identify a link between the drug dose and the severity of the withdrawal symptoms (1976). Brooks had come to the same conclusion in his study in 1959: "There seemed to be no correlation of the intensity of the reaction with the level of dosage..." (p. 932).

Dilsaver and Greden delivered an overview of the available literature to withdrawal symptoms in antidepressants to 1984 delivered. They came to the conclusion that withdrawal symptoms often occur surprisingly, namely 21% to 55 % of adults. Some authors even came to 80%; the frequency found was connected with the hanging with the diligence of the investigation together (Dilsaver & Greden, 1984a).

The Current Situation

Ever since the emergence of psychiatric drugs, many people who have taken prescriptions have made their own decision to quit. Many had quit the drugs successfully and were never seen again by physicians. Degkwitz informed long ago:

> "Unquestionably, some patients tolerate the abrupt discontinuation of psycholeptics *(neuroleptics and antidepressants)* readily, while others experience considerable discomfort" (1967, p. 162).

What remains is a percentage of people which attract attention or ask for new psychotropic drugs and become patients again. I think it is safe to say that a great number of attempts to quit would have been more successful if those wishing to quit and those around them had been better informed as to the potential problems that may arise as well as of means for preventing the often-prophesied relapse. How often are these withdrawal-problems misdiagnosed as relapse into psychoses? Brigitte Woggon of the Zurich University Psychiatric Hospital, a strong proponent of psychiatric drugs, saw problems with the lack of differentiation made 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" (1979, p. 46).

What was meant by "relapse," usually was not defined, or "relapse" was seen as "return to active medication" or the "deterioration in performance."" In addition, often there was no double-blind experimental design, that means, in open trials the expectations of the physicians coined the results decisively.

Withdrawal with and without Physicians

Mostly, physicians are not very helpful for patients who decide to withdraw. Single exceptions cannot hide this fact. Many consumers of psychiatric drugs are convinced they need their physician's absolute agreement to withdraw. But people who stop taking psychiatric drugs against their physician's advice are just as likely to succeed as those who come off with physician agreement. This was the finding of the research project "Coping with Coming Off," commissioned by the national organization Mind in England and Wales. Funded by the British health ministry, a team of users and survivors of psychiatry carried out 250 interviews to investigate experiences with coming off psychiatric drugs. The forms of support found most helpful were: support from a counsellor, a support group or a complementary therapist; peer support; information from the internet or from books; and activities such as relaxation, meditation and exercise. Physicians were found to be the least helpful group to those who wanted to reduce or come off psychiatric drugs (Read, 2005; Wallcraft, 2007). Following this study, Mind changed its standard advice to patients – but only for a short period. Historically, their advice was not to come off psychiatric drugs without consulting a physician first. After the publication of "Coping with Coming Off"-study, people were

reminded of the indoctrination of physicians by Big Pharma (Darton, 2005, p. 5) and advised to seek information and support from a wide variety of sources (Read, 2005). This warning meanwhile is deleted with the justification by Mind, the situation would be different now.

Withdrawal on Your Own and Eventually with Competent Support

Finally – now – it is to begin with the overdue building of a network to support consumers of psychiatric drugs who take their right to terminate the additional intake of these substances with words and deeds. On the basis of certain knowledge and with knowledge of existing uncertainties, all people involved in withdrawal processes should be enabled to deliver competent support.

Some stakeholders in the psychosocial field have more or less isolated from each other started as pioneers to offer aids in coming off psychiatric drugs. The time has come to gather knowledge how people came off these substances without ending up once again in the doctor's office and made productive for all. Gaps in knowledge and developing mistakes have to be identified. An overview on withdrawal symptoms from psychotropic drugs has to be built. Research of withdrawal problems and damages caused by dependence through psychotropic prescription drugs should be defined and suggested to foundations, universities as well as to organisations of users and survivors of psychiatry for user-controlled research. Patients' experiences should be integrated into relevant programs. Pilot projects how local organizations can accompany withdrawal and be financed and continued should be stimulated, and regular first aid telephone hotlines should start without delay.

There is much knowledge already about reducing risks in withdrawal, especially on the side of ex-users and survivors of psychiatry. There are some publications by critical (ex-) users and survivors of psychiatry and their supporters with some tips for risk reduction at withdrawal (NAPA, 1984; Caras, 1991; Lehmann, 1998 (2004); Breggin & Cohen, 2000; AGIDD-SMQ et al., 2003; Breggin, 2012; Icarus Project & Freedom Center, 2012; DGSP, 2014).

No question, there is a lot of good will on the side of ex-users and survivors of psychiatry and their supporters, but sometimes they offer problematic tips, too. For example, the German Federal Association of Users and Survivors of Psychiatry informs, that if withdrawal problems arise people can take a high dosage of neuroleptics spontaneously to "bring you down," but without warning about the enormous burden of the body through an extreme blood level change and the risk of a delirium of organic nature (Krücke, 2014, p. 11). It is well known that rapid doses changes are a risk factor for deliria, and that such syndromes can end deadly. Another example: A service provider in Berlin summarized the publication of the German psychiatrist about the high mortality of psychiatric patients, unwanted effects of neuroleptics, alternatives, risk-lowering ways of withdrawal and the task to dosage as low as possible for people who cannot do without neuroleptics with the headline: "Neuroleptics can and must be administered in minimal dose" (Pinel, undated). Coming off then is no issue any more; the further intake of neuroleptics seems alternativeless. To give drugs not in doses higher than required is a standard in mainstream medicine, psychiatry included. Some psychiatrists use the unwanted effects in antidepressants, dependence included, to praise electroshock as less dangerous – despite their effect to destroy brain nerves irreversible. Enhancing figures of electroshock administration are one of the results of their way dealing with drug dependence.

A Mass of Open Questions

For medical practitioners and natural healers, carers, therapists, social workers, jurists, pharmacologist, relatives and experts by experience a mass of open questions remains. Especially the last-called ones' task it is to raise the open, non-pleasant and huge questions and to keep the power of definition of the problem. Especially their task is to raise all the open, unpleasant and huge questions and to ensure that they do not lose the power to define the unresolved issues. All the knowledge and the answers to the open questions should lead to a curriculum and be used for education in the psychosocial field and in the self-help sector and be offered to patients who decide for themselves to quit their psychotropic drugs and the relatives and friends who want to support them.

Especially you should further on work on these topics:

- Psychiatric-medical aspects: Which doctrines of the need for continuous administration of psychotropic drugs make it difficult for physicians and in particular psychiatrists to provide assistance in self-determined withdrawal of psychiatric drugs? How can physicians ensure support in coming off, as long – except the diagnose of dependence on benzodiazepines – no appropriate diagnoses and therefore no billing codes exist? Which stationary possibilities of support do exist? What risk factors require a stationary support at withdrawal? Is a sporadic, needs-adapted support at withdrawal?
- Which unwanted psychotropic effects require an immediate withdrawal? Which physiological and psychological withdrawal symptoms can be expected? How can you cope with them? Which withdrawal symptoms may occur especially during the transition from mini doses to zero? Is in an insurmountable situation a so-called very-low dosage useful, and if so, what residual risks does it include? Who makes and controls the decision on the need for a minimum dosage? Can, similar as in alcohol, a premature offer for minimum dosage jeopardize the decision to withdraw? Which check-ups are also useful in minimum dosage, which symptoms point on the development of long-term damages?
- How to find competent and user-orientated physicians?
- Homeopathic aspects and aspects in natural healers' practice: Which methods are used to relieve withdrawal symptoms, to stabilize in particularly the vulnerable period immediately after withdrawal? What are the options for detoxifying and "emergency pharmacies" use in homeopathic or naturopathic practice?
- Aspects in social work, nursing, psychotherapy, service providers: How can psychotherapists, social pedagogues and social workers, social workers and psychiatric nurses be trained to give competent help? Are there psychotherapeutic methods especially suitable for monitoring at withdrawal? What psycho-educational processes hamper aid at withdrawal? How to find competent and user-oriented psychologists?
- Which opportunities and problems have provider of community-based services and of wards in hospitals and of psychiatric clinics to offer support in coming off psychiatric drugs on a secure institutional and financial basis?
- Pharmacological and pharmaceutical aspects: How to withdraw combinations? Which role do various forms of administration play in the gradual reduction? How can dosages be reduced beyond predetermined product units? When can you use pill cutters? What, if

capsules contain pellets (balls, beads)? How can pellets be protected from breaking down in the stomach, if they require an intestinal absorption? How can doses be reduced by time stretching, and which psychotropics do not deliver this possibility? What is the ratio of half-lives for speed of withdrawal? Where do patients find practical advice if their physicians do not respect their wish to withdraw and deny any technical assistance in dose reduction? What to do, at in the neuroleptic withdrawal process the sensitivity faster returns than the suicidal tendencies disappear?

- Legal aspects: Which risks have physicians to face who – eventually in spite of the occurrence of early warning symptoms, which point on the development of long-term damages – do not initiate any step to reduce or withdraw? Can physicians be prosecuted, who do not inform about the risk of dependency and withdrawal symptoms at the beginning of the administration, during its course and at the transition to long-term administration? What if they deny aid and do not want to treat patients if they demand assistance in gradual withdrawal? Which professional and civil right risks have service providers, nurses and other psychiatric professionals to face who assist patients in their withdrawal process but without medical instruction or against it? Which possibilities does the UN Convention on the Rights of Persons with Disabilities, other human rights declarations, and laws deliver to enforce people's demands to withdraw psychiatric drugs against the decision of their legal guardians?
- Self-help and family aspects: Where can patients, their relatives and friends inform themselves balanced about possibilities and problems at withdrawal? What can they do if physicians to not support self-determined withdrawal of if there is a lack of sufficient knowledge about the possibilities and problems at withdrawal on the side of the physician? How can they plan the withdrawal process, and which issues should they keep in mind? Where do patients who are alone find support? Which substances lower withdrawal symptoms? How useful and promising are advance directives to guarantee self-determination at least partially in case of withdrawal symptoms and relapses? Which environment, lifestyle, diet and physical activity support a successful withdrawal? How to cope with sleeping problems cause by the withdrawal? Which support options, but also which risks, can be expected in the self-help sector?

Drug companies make money by selling drugs; stock prices depend on new drugs in the pipeline. Profit maximisation correspondents not always with maximal ethical standards (Gøtzsche, 2013, 2016). To warn from dependence would lower profits, same as the warning from withdrawal problems or providing information how to lower withdrawal risks would do. Then withdrawal outcomes would be more positive, people would recover more likely. Their bodies would be missed as business market. As long as there is no general demand for the renunciation of toxic synthetic substances in the nature, the living area, nutrition and medicine, as long there is the illusion that psychiatry as a scientific discipline can do justice to the expectation of solving mental problems that are largely of a social nature, as long there is no warning on blurbs against the risk of dependence from neuroleptics and antidepressants, as long as there is no duty for Big Pharma to pay for compensation and rehabilitation (comparable to tobacco industry), as long as Big Pharma finances professionals, "self-help"-groups like GAMIAN (Global Alliance of Mental Illness Advocacy) and organisations of relatives to get them on board for PR-strategies, and as long there is no involvement of independent organisations of users and survivors of psychiatry into psychiatric drug

registration, evaluation and monitoring: drug companies can go on undisturbed to merchandise their drugs and conceal the fact of physical dependence from neuroleptics and antidepressants.

Meaningful involvement in drug issues would require involvement in licensing processes in order to participate in decision-making about the granting and withdrawal of licenses (Lehmann, 2004). This involvement might be directly or via trusted experts and end with recommendations to governmental committees on the safety of medicines. Compared to other medical patients, a specific involvement of organisations of users and survivors of psychiatry would be required. This is due to the current discrimination and missing changes to eke out compensation for the generated damages.

Teaching Withdrawal – A Task for Decades

Until today, teaching withdrawal is passing knowledge and experiences, but also teaching the open questions and the need for participation of independent users and survivors of psychiatry in deciding the direction of future politics and research – a task for decades. Teaching withdrawal, too, is teaching alternatives beyond psychiatry and the need for co-operation of all organisations and people of good will with the meaningful involvement of users and survivors of psychiatry on all levels. (It is questionable if organisations and persons taking money from Big Pharma, even if they maintain to deal critically with dependence from neuroleptics and antidepressants, can be assigned to the organisations and people of good will, especially when they do not uncover their financial enmeshment with the pharmaceutical industry.)

In the light of the mass of problems, stakeholders believing to be able to solve withdrawal problems seem not to work promising. Reflecting the lack of clear borders between black and white and the altering in convictions and beliefs in opinion leaders on all sides, who could arrogate to deliver a patent recipe how to solve the problem with dependence from neuroleptics and antidepressants?

Remark

Translations of the citations into English and explanations in italic brackets within citations by Peter Lehmann.

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