SCHEMA THERAPY: EVIDENCE BASED TREATMENT FOR CHALLENGING MENTAL DISORDERS

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Schema therapy (ST) was developed as a transdiagnostic approach for treatment challenging clinical disorders. It also provides disorder specific models for most personality disorders (PD). There are growing number of evidence that show that ST model is very effective for patients with borderline personality disorders (BPD), antisocial personality disorder (ASPD), all cluster C personality disorders. Good results are also reported by number of researchers for chronic depression, post-traumatic stress disorder, including complex PTSD, eating disorders, and complex obsessive compulsive disorders.

Schema therapy derives from cognitive-behavioral therapy (CBT) and considered by majorities of psychotherapists as a third wave CBT approach. ST was developed by Jeffrey Young (student of Aaron Beck) in 2003 for patients, which did not respond to standard CBT. These patients often had a comorbid personality disorders and showed complex, rigid, and chronic psychological problems in emotion regulation and in interpersonal relationships, which in most cases could be followed back into their childhood. These problems also impaired the psychotherapeutic process as those patients had difficulties in forming a collaborative relationship with the therapist and could not be reached with standard CBT techniques due to intensive emotional reactions and coping strategies such as avoidance or surrender. In order to solve these clinical challenges Young integrated ideas and techniques from other theoretical orientations into a classical CBT frame (attachment theory, Gestalt therapy). A strong emphasis was placed on the biographical aspects for the development of maladaptive psychological patterns through traumatization in childhood and frustration of basic childhood needs. The therapeutic relationship was conceptualized as “limited reparenting” meaning that the therapist creates an active, caring, parent-like relationship with the patient.

The major goal in ST is helping patients to understand their emotional core needs and learn ways of getting needs met in an adaptive manner or to help them deal with the frustration if needs cannot be satisfied. This requires breaking through long-standing emotional, cognitive and behavioral patterns, meaning change of dysfunctional schemas, coping strategies and modes.

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PSYCHOPHARMACOTHERAPY OPTIONS FOR PATIENTS WITH ORGANIC BRAIN DAMAGE

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Introduction: Despite considerable efforts of scientists and medical practitioners, neither treatment standards nor complete recommendations for various mental disorders in patients with organic brain damage have been developed so far.

The specific strategies of choice that increase the effectiveness of pharmacotherapy can be: a) considering the level of brain damage (stem, subcortical, hemispheric), b) stimulating interhemispheric connectivity, c) impacting a “weak” neurotransmitter component by stimulating or inhibiting separately different (choline, glutamate, dopamine and GABergic) systems.

However, a main criterion of drug choice has been and remains clinical one which provides a pharmacological strategy needed in the patient’s current state, with identifying not only negative symptoms of mental disorders, but also productive psychopathological symptoms produced by a deficient (due to brain damage) functional system to enable a coherent neuropsychic activity.

Goal: To find clinical options relating to the choice of therapeutic strategy for patients with mental disorders due to organic brain damage.

Subjects and methods: Treatment and side effects were analyzed during 1613 courses of psychotropic medication administered to 365 patients with traumatic or neoplastic brain damage.

Results: After examining the contingent of patients, the following treatment options were suggested for various clinical situations:
1. Emerging from the states of inhibited consciousness (unconscious states, various types of mutism, states with severely limited contact) can be accelerated by neurometabolic, primarily neurotransmitter, means. If a blockade of voluntary activity is followed by reduced muscle tone, weakened reflexes and sensitivity, Ipidacrine is most effective at a dose of 20 to 120 mg; with increased tone and reflexes, Amantadine at a dose of 100 to 400 mg is a drug of choice; with hyperesthesia, hyperkinesis, vegetative paroxysms, or unfocused motor agitation, Aminophenylbutyric acid at a dose of 500 to 3000 mg is preferable.

2. Consciousness reintegration (in cases of confusion, severe amnestic syndromes) is more probable during the treatment with GABAergic agents (Aminophenylbutyric acid at a dose specified above, D-, L-hopantenic acid at a dose of 500 to 2000 mg per day) and polypeptide drugs (Semax, Selank, Cortexin).

3. Cognitive disorders in cases of clear consciousness regress more quickly and efficiently, if the impact of medication specific to functions of the left or right brain hemisphere is taken into account. With left hemispheric dysfunction, Ipidacrine, Donepezil, Memantine are more effective; with right, Ethylthiobenzimidazole, Aminophenylbutyric acid and D-, L- hopantenic acid, as well as Semax. It is also necessary to consider the patient’s profile of personal asymmetry, as side effects and paradoxical reactions are more often found in patients with motor, sensory or family left-handedness.

4. Asthenic disorders are more likely to regress, if their specific clinical manifestations are taken into account. Hyposthenia with weakened reactivity to the environment, decreased sensitivity, difficulty in focusing attention, excessive sleepiness is better treatable by nootropics with a stimulating effect (Piracetam, Pyriditol); hypersthenia with excessive reactivity, hyperesthesia, difficulty holding attention, dyssomnia due to anxiety, “stream of thoughts”, by tranquility nootropics (Aminophenylbutyric acid and Selank) or Tetramethyltetraazabicyclooctandion, a daytime tranquilizer.

5. Psychotic disorders are most often treated by neuroleptics, with preference to atypical ones due to a lower probability of side-effects. In case of persistent unfocused agitation, Quetiapine is administered at a dose of 50 to 300 mg per day; if behaviour is affected by psychotic disorders (hallucination, delusion), Risperidone at a dose of 1 to 6 mg per day.

6. Aggression is eliminated, depending on the clinical context, by non-benzodiazepine tranquilizers (Aminophenylbutyric acid, Buspirone, Tetramethyltetraazabicyclooctandion), valproates or adrenoblockers. Only if those are inefficient, small doses of neuroleptics can be used: Periciazine (preferably in drops, up to 10-15 mg per day) or Risperidone (up to 1-2 mg per day).

7. Emotional and neurotic disorders (depression, anxiety and phobia) are treated with tranquilizers and antidepressants. To avoid multiple side effects, serotonergic drugs (Rexetine, Citalopram, Escitalopram, Vortioxetine), dual-action drugs (Venlafaxine, Duloxetine, Mirtazapine) or others (Agomelatine) should be preferred over tricyclic antidepressants. For the same reason, tranquilonootropics (Aminophenylbutyric acid, Selank) or other non-benzodiazepine drugs (Buspirone, Tetramethyltetraazabicyclooctanedione, Hydroxyzine, Fabomotizole) should be used whenever possible instead of benzodiazepines (Diazepam, Alprazolam, etc.).

**Conclusion:** The further collecting and compiling of clinical data in patients with organic brain damage are necessary to define and develop the options listed above. It will enable to conduct theoretically and practically well-grounded randomized clinical trials and, thus, to formulate recommendations and standards for its differentiated pharmacotherapy.