On the clinical significance of autogenic discharges
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Early in the development of Autogenic Training, some unexpected manifestations, often unpleasant, were reported during the practice of the autogenic standard exercises. Schultz (1) does not specifically mention those phenomena in his early book, but duly acknowledges its existence in a later one (2). Not much attention was paid to those "training symptoms", other than occasional anecdotal reports in the German autogenic literature. The main consensus was that those manifestations were non-relevant side effects, secondary to idiosyncratic characteristics of the trainee or to minor technical errors, and that they would disappear with appropriate training (Luthe, personal communication).

However, in 1961 there was a major breakthrough in the understanding of autogenic phenomena. This year, in the third World Congress of Psychiatry in Montreal, Luthe presented his seminal paper "The Clinical Significance of various forms of Autogenic Abreaction".

After analyzing over 2000 hours of therapy of 100 patients, Luthe was able to demonstrate a link between the nature of the training symptoms and the difficulties, complaints and traumatic (physical and psychological) events in the life of his patients. Because its unexpected presentation and its equally sudden disappearance during the autogenic state, Luthe renamed the training symptoms "autogenic discharges", and devised a method, Autogenic Abreaction, to facilitate its evolution and final dissipation.

The fact that clinical improvement ensued when the autogenic discharges were allowed to proceed to its final dissolution, led Luthe to formulate the hypothesis that they were the expression of a self-regulatory activity of the brain, a process he termed "autogenic neutralization" (3,4,5). He reported further confirmation of his discovery in two special groups of trainees: psychiatric patients that had undergone electroconvulsive therapy, who presented "electrical" discharges (6) and clergy personnel, whose discharges we familiarly grouped under the name of "the ecclesiogenic syndrome" (7).

My own studies with temporal-lobe epileptic patients in the Montreal Neurological Institute provided clinical and electroencephalographic support to Luthe's theory. Two of the criteria for patients' selection were a) persistence of epileptic seizures despite appropriate neurological treatment and b) presence of psychiatric symptoms that justified their referral to my psychosomatic unit. Autogenic standard exercises were modified (reduction of time and body area) because some

patients showed an increased risk of epileptic seizures with the regular protocol. After nine months of regular daily practice of autogenic training, the treatment group showed a significant improvement of their psychiatric symptoms *and* of their neurological status, both in the frequency and in the severity of their seizures. No psychiatric drugs were prescribed during the procedure, and some of the patients had a reduction of their antiepileptic medication.

The baseline EEG recordings taken prior to and after treatment did not show any significant differences. However, the practice of the autogenic exercise during the EEG recording was accompanied by increased epileptic manifestations, mainly medium amplitude slow sharp waves on the temporal lobes. This electrical epileptic activity was not accompanied by external manifestations, nor did the patients report any special discomfort during the proceedings. The EEG recording returned to its previous pattern at the termination of the autogenic exercise (8)

The intriguing observation that autogenic training produces both clinical improvement and subclinical epileptogenic electrical activity lends support to Luthe's discharge theory. It is well known that clinical epileptic seizures are preceded by a progressive increase of neuronal hypersynchronization, which ends in the sudden discharge that produces the clinical seizure. It is our hypothesis that the subclinical discharges during the autogenic state maintain the accumulation of neuronal excitation under the clinical threshold, preventing thus the clinical seizure. On the precautory side, it has to be said that undue prolongation of the autogenic state may induce a clinical seizure, reason why the exercises have to be short and partial, and always performed under the supervision of a qualified expert. Despite its proven beneficial effects, epilepsy is listed by ICAT as a relative contraindication for autogenic therapy.

Autogenic discharges, in general, may obey to similar brain dynamics as those observed in our study with epileptic patients. Certain brain areas may sustain undue chronic activation, which is released during the autogenic state.

Obviously, any activity that could increase the activation of the affected brain areas is to be avoided. On the contrary, facilitation of spontaneous discharge is an appropriate intervention during treatment with autogenic neutralization methods. The so called pro-homeostatic interventions are technical devices to eliminate blockings and resistances to the normal development of spontaneous autogenic discharges. They should not be applied with the aim of voluntarily inducing or provoking any kind of manifestations that are not required by ongoing autogenic neutralization dynamics. Putting it easily, it is not that crying (or vomiting, or trembling or whatever) is good, but that *repressed* crying (or whatever) is bad.

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