of a patient whose objective mind, realizing the danger of habitual drug ingestion and in full possession of his sound mentality, desires to discontinue the use of the habit-forming drug. Every drug-steeped cell of his system cries out against the sober judgment of his mind, demanding gratification of the habit, the memory. So in epilepsy, the longer the patient's brain cells are held free from epileptic toxins or ferments, the longer the spell-free intervals, the deeper will the non-epileptic habit be established.

All this applies, of course, only to epilepcy not suppressed by drugs. This epileptic habit demands recognition in connection with Jacksonian epilepsy. Here the reputed cause being pressure upon the cortex, does not give rise to continuous convulsions (status epilepticus) in the afflicted individual. The attacks recur with irregular periodicity and severity, sometimes several times daily, then again not for weeks or months, exactly as in other cases (the idiopathic type) of epilepsy. One is justified in asking why the Jacksonian epileptic is not all the time, while the pressure upon the cortex persists, under the influence of such pressure, why not in a "status epilepticus"? The confusion increases (if we credit the pressure theory) when we notice that after trephining, exact location of cortical pressure and removal of the same, the attacks do by no means decrease, and that after the operation it is required that the "epileptic habit" be treated. Persistent administration of the accepted standard remedies directed against this "epileptic habit" remains too often utterly fruitless.

May one not reasonably assume that in case of Jacksonian epilepsy, the damaged cortex presents a locus minoris resistentia, hence a hypersusceptible area for epileptogenic toxins coursing in the patient's blood and also creative of epileptogenic ferment production by the injured brain cells. The weakened brain cortex offering no resistance to the responsible substance, whether ferments or toxins, are poisoned first or perhaps alone of all the organs and thus enable the observer to notice the registering of the toxins by release of convulsions and mental derangement. Does not treatment of the epileptic habit merely mean to afford the recently damaged cortex time to repair, thereby placing it upon an equal basis of resistance with the cortex of the idiopathic epileptic? Does not this point to some other force in the physiology of the epileptic, some other factor that possesses the pwer to record its presence upon the cortex by releasing epileptic attacks; a cause remaining and still controlling unabated after removal of the supposed cause? This, too, supports the contention that in the blood, perhaps in the brain (carried to it by the blood) of the epileptic, is contined a epileptogenic material, a toxin which poisons the cortical layer of cells and