

CLINICAL, MOTOR, AND BIOLOGICAL CORRELATES OF DEPRESSIVE DISORDERS AFTER FOCAL SUBCORTICAL LESIONS

Edward C. Lauterbach, M.D., Joseph G. Jackson, M.D., Spencer T. Price, B.S.
Ami N. Wilson, B.A., Alan D. Kirsh, M.D., G.E. Alan Dever, Ph.D.

The authors studied depression after focal subcortical lesions (SCLS) in 45 highly selected subjects. Secondary major depression (2'MD) occurred in 20.0%, depressive disorder NOS (2'DDNOS) in 4.40%, and 2' dysthymia in 0.0010. 2'MD after SCLs was associated with pallidal lesions (88.9%) and dystonia without geste antagonistique; subjects with 2'DDNOS had nigrosegmental lesions and Parkinsonism. Depressive severity after SCLs correlated positively with severity of parkinsonism and dystonia. Pallidal lesions disrupting neurotransmitter systems and pallidothalamic and parietal input to the frontal lobe may lead to 2'MD, whereas nigrosegmental lesions may predispose to 2'MDforme fruste (2'DDNOS) through disruption of mesocortical frontal or nigrostriatal dopamine tracts. Patients should be closely followed over several years for depression after such lesions, especially when accompanied by parkinsonism or dystonia without geste antagonistique. nigra and basal ganglia. Consequently, we studied the relationship of depression to subcortical circuit disruption and these clinical neuropsychiatric findings in subjects with circumscribed SCLS. We endeavored to determine which subcortical structures, subcortical circuits, and clinical features were associated with depression in our subjects. Specifically, we analyzed the frequency of DSM-III, "DSM-III-R, 2' and DSM-IV" major depression with first onset occurring before (1'MD) or after (2'MD) SCL onset. We focus on 2'MD here because of our interests outlined above. We also examined the frequency of other 2' depressive disorders: depressive disorder not otherwise specified (2'DDNOS) and dysthymia. We compared our rate of 2'MD after SCLs to the rate of MD in matched normative controls and to the rates for 2'MD in identically studied consecutive subjects with two different clinically recognizable basal ganglia disorders, PD and ID. We also compared this rate of 2'MD after SCLs with external control rates of 2'MD after parietal lobe and left frontal infarcts. Next, we surveyed the lesion loci in 2' depressive disorders. Finally, we ascertained whether the observed rate of 2'MD in this select sample exceeded chance alone and examined disabling clinical variables (2' parkinsonism and 2' dystonia) that might be associated with depressive severity in this select SCL sample.

We hypothesized that:

- 1 - The prevalence of 2'MD after SCLs exceeds the rate expected for normative populations.
2. Parkinsonism severity is greater in SCL subjects with more severe depression.
3. Dystonia severity is greater in SCL subjects with more severe depression.

4. Dystonia with geste antagonistique occurs less frequently in 2OMD than in SCL subjects with I OMD.

We were interested in testing these hypotheses because 1) 2'MD after SCLs should be more common than normative rates if the depression is truly a consequence of the lesion; 2) although primary PD and ID may be associated with 2'MD in general, this may not prove to be true of our sample of SCLs with 2' parkinsonism or 2' dystonia; 3) parkinsonism is associated with disruption of limbic dopamine supply;^{24,3'} and 4) dystonic geste antagonistique represents a novel clinical variable associated with reduced symptom control and the inability to modulate motor outflow by sensory input.³² (*Geste antagonistique* refers to postures and other sensory tricks adopted to alleviate dystonic contractions,³² probably requiring the physiological interaction of parietal sensory and frontal motor regions of the brain.)

(The journal of Neuropsychiatry and Clinical Neurosciences 1997; 9:259-266)

Lauterbach EC, Jackson JG, Price ST, Wilson AN, Kirsh AD, Dever GEA: Clinical, motor, and biological correlates of depressive disorders after focal subcortical lesions. Journal of Neuropsychiatry and Clinical Neurosciences 9(2):259-266, 1997.

Article Published in: Journal of Neuropsychiatry and Clinical Neurosciences