PSYCHIATRIC MORBIDITY AFTER CEREBROVASCULAR ACCIDENTS

Thesis submitted for partial fulfillment of the requirements of the M.D. Degree in Psychiatry

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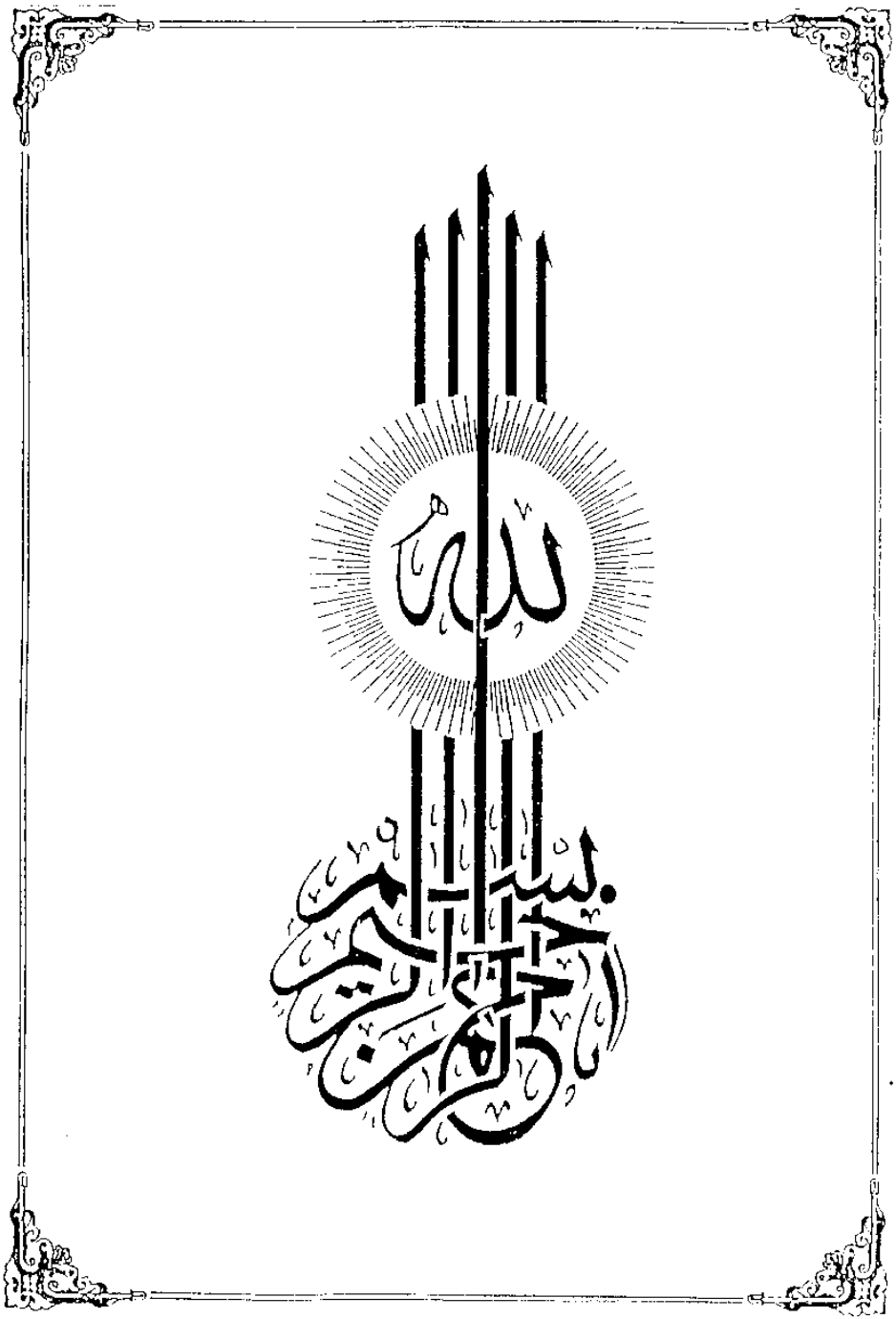
قالوا

سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا

إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

صدق الله العظيم

سورة البقرة (الآية ٣٢)



TO MY PARENT

AND

TO MY WIFE

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INTRODUCTION AND AIM OF THE WORK

Literature Review

One of the great controversies in psychiatry is the dichotomy between the, so called, "functional" versus "organic" nature of the various psychiatric disorders or in other words, the "psychological" versus the "biological" model.

The same controversy applies to psychiatric symptoms that follow cerebrovascular accidents, where the question is whether to consider these symptoms as pure "psychological" reactions to the resulting handicap or as specific "endogenous" complications of the resulting cerebral damage.

One of the factors that favor the "biological" nature of these symptoms is their relation to specific lesion locations.

The aim of this work is to study of relation between the site of cerebral dysfunction in patients with cerebrovascular accidents and the resulting psychiatric morbidity, in order that better understanding of the nature of these symptoms, and subsequently more appropriate management, would be possible.

So as to reach more precise definition and elaboration of "localization of dysfunction" in those patients, more than one aspect of localization were aimed to be studied: i.e. anatomical (morphological), physiological and neuropsychological.

To achieve this aim, the planned strategy entails first diagnosing the various psychiatric syndromes that tend to follow cerebrovascular accidents and secondly examining the site of dysfunction in those patients anatomically, neurophysiologically as well as neuropsychologically, correlating the resulting psychiatric syndrome with the various findings regarding the different aspects of localization.

DEPRESSION FOLLOWING CEREBROVASCULAR ACCIDENTS

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e) factors related to the management prescribed in stroke.

* Aetiology and pathogenesis of P.S.D.

(1) Psychological model.

(2) Biological model.

- anatomical data.
- inter. hemispheric interaction.
- biogenic amine mechanism.
- experimental evidence.

(3) Toward a psychobiological model.

* Why it is important to recognize depression in stroke patients.

* Biological markers of P.S.D.

* Management of depression following stroke:

(1) pharmacotherapy.

(2) electroconvulsive therapy.

(3) non-physical treatment.

Historical Survey of the Concept of "Post-Stroke Depression"

Mood disturbances following stroke have long bean noted by clinicians:

(1) *Kraeplin* (1921) recognized an association between manic depressive insanity and cerebrovascular disease.

(2) *Bleuler* (1951), in his "Textbook of Psychiatry", wrote that following stroke: "Melancholic moods lasting months and sometimes longer appeared frequently".

(3) *Gainotti* (1972) has pointed out the importance of the hemispheric side of the lesion in the production of mood disorders: catastrophic reactions and anxious depressive orientation of mood seemed to be more commonly associated with left hemisphere lesions, while right hemisphere damage was associated with apathetic stage and undue cheerfulness as described by *Hecan et al* (1951).

(4) The hypothesis that depression may be a specific complication of stroke emerged only recently (*Folstein et al., 1977*).

In an important series of works in animals and man, a group from Baltimore has developed the concept of "post-stroke depression" (*Robinson, 1987*), which is considered to be related to biochemical changes induced by the cerebral damage due to stroke, rather than a mere psychological reaction to the resultant disability.

Prevalence of Depression Following Cerebrovascular Insults

Marked controversies and variations do arise, as regards the prevalence of depression following cerebrovascular accidents:

* Numerous studies have found that depression is a frequent sequela of stroke and up to 50% of stroke patients may develop depression during the acute post-stoke period. Among out patient stroke population, the prevalence of depression was found to be close to 30% *(Starkstoin and Robinson, 1989)*.

* In another study, depressed mood was present in about 25-30% of surviving and assessable patients at anytime up to one year after stroke.

Less than half of those depressed in the early stages were free of depression at one year. *(Wade et al., 1987)*.

* *House et al* (1991). in their study for mood disorders in the year after first stroke found that depression in stroke survivors did not differ from that found in patients with other physical disorders.

They considered the phrase "post-stroke depression" to be misleading, since it implies the existence of a discrete, particularly common and specific clinical syndrome. They found only two cases of major depression out of 128 stroke patients for the year following their stroke, so, argued that the frequency of depressive disorders and their persistence after stroke, had been over estimated.

* The marked discrepancy in the statistical data. Concerning the prevalence of mood disorders after stroke could be

explained as follow:

(1) Great variations in the methodological issues encountered, do play a role. The measurement and definitions of the psychiatric disorders employed, are not universal, hence, the findings are variable. Reliability of psychiatric diagnosis tends to vary according to the criteria used.

(2) Another factor is the population studied or the patients' selection. The patients' sample has not the same criteria in each study. Some studies are restricted to outpatients, who are more likely to be less disabled and less socially deprived, which represents a bias in generalizing the epidemiologic results to all stroke patients.

(3) The symptoms of depression may be masked or mimicked by other symptoms as cognitive impairment, aphasia or even other emotional problems. Depression is usually used as a "catch all" term for all emotional problems following stroke (*House et al., 1991*).