

Psychiatric Morbidity After Stroke: A Multidimensional Approach

Okasha A., Asaad T., Zakaria M., Kamel M., Self EL Dawla A., and Ashour S.

Abstract

Psychiatric symptoms after cerebrovascular accidents are quite common. A marked controversy exists as regards the nature, mechanisms and location underlying their occurrence. A multi-dimensional approach has been applied in the present study for a more precise elaboration of the site of dysfunction, making use of brain CT scan, EEG and Brain Electrical Activity Mapping (BEAM) as well as the Luria Nebraska Neuropsychological Battery (LNNB) and clinical examination. The study was carried out on 103 stroke patients and 50 healthy controls. Clinical diagnosis was made according to DSM III R criteria using the SCID patient and non-patient versions. Psychiatric symptoms were more associated with ischemic than hemorrhagic lesions. The results revealed a threshold psychiatric diagnoses in over 50% of stroke patients, most of whom suffered from depression (16.5% major depression and 19.4% dysthymic disorders). Other diagnoses included anxiety, mania, non-affective psychoses and conversion disorder. The multidisciplinary approach tended to localize the lesion in the left anterior cortical and sub-cortical lesions in the case of post-stroke depression, and in the right temporal region in post-stroke mania. In cases of post-stroke anxiety no significant difference was detected between both hemispheres, and post-stroke psychotic disorders tended to be associated with an epileptic activity especially on the left hemispheric region. Also, psychiatric morbidity following stroke was found to be related not only to the site, but also to the extent of dysfunction. Premorbid personality traits seemed to play a role as a vulnerability factor in developing post-stroke psychiatric symptoms especially depression. The study discusses the emphasis on a multidisciplinary approach in studying the site of dysfunction in cerebrovascular accidents.

Introduction Psychiatric symptoms after cerebrovascular accidents are quite common, and marked controversy does exist as regards the nature and mechanism underlying their occurrence. Two major theories have been proposed to explain the relation between stroke and post-stroke psychiatric symptoms. The first considers psychiatric symptoms to be a psychological response to the associated impairment, thus depending on factors related to the patient, the nature of the illness, and the social environment and social support systems (Uyod, 1977). The alternative theory considers the resulting psychiatric morbidity to be a specific disorder directly related to the cerebral lesion (Folstein 1977; Robinson 1987). The relation of specific symptoms to certain lesion locations is one of the factors in favor of a biological nature of those psychiatric sequels. However, such a relation is still a matter of great dispute. Marked discrepancy exists between the studies dealing with this issue as regards the site and laterality of the lesion responsible for the causation of a particular symptom or symptom cluster. Moreover, most of the previous studies considered only anatomical data, usually through brain CT scan, in examining the site of dysfunction, with little or no attention to the accompanying psychological and neuropsychological profiles, which may add to the understanding of the nature and mechanism of psychiatric sequel in such patients. The present study was designed to have a broader descriptive evaluation of cerebral dysfunction in

patients with post-stroke psychiatric symptomatology, considering multiple aspects: anatomical (through brain CT scan), physiological (through EEG and BEAM) and neuropsychological (through Luria Nebraska Neuropsychological Battery - LNNB), in addition to clinical neurological examination.

Material and Methods The study sample consisted of patients who have a history of cerebrovascular accident throughout the previous year preceding the assessment, and who attended the neurology and stroke unit at the Ain Shams University Hospital in 1990.

Exclusion criteria included patients over the age of 65, those with marked disturbance in the level of consciousness, or those with aphasia to an extent that makes verbal assessment impossible, and those with a history suggestive of other neurological disorders that might interfere with the results. We had a total of 103 patients with cerebrovascular accidents, all of whom were assessed through the following:

1. Clinical physical and neurological examination.
2. Structured clinical interview based on DSM-III R criteria: SCID-I Non-patient (NP) version for the assessment of psychiatric morbidity. Those who showed frank psychiatric symptomatology were further assessed by SCID-I outpatients (OP) and patient (P) versions.

SCID gives diagnoses either at threshold (*full criteria met*) or subthreshold (*full criteria not met, but clinically disorder seems likely*).

3. Computed tomography of the brain (CT) for anatomical localization of the site of dysfunction. CT scans are done without contrast using 10 mm cut from the base of the skull. They were examined by the radiologist who reported the site and type of lesion without knowledge of the patient's clinical state. The localization of lesion was determined according to the criteria described by Starkstein et al. Four lesions were classified as "anterior" if the distance from the anterior border of the lesion to the anterior pole of the brain was less than 40% of the greatest anteroposterior diameter of the cut; otherwise they were considered "posterior". Localization terms according to the area of brain affected (e.g. frontal temporal...etc) are also used. The depth and pathology as well as the volume calculated as a proportion of the total brain volume were reported by the radiologist.

4. Electroencephalography (EEG) and Brain Electrical Activity Mapping (BEAM), using power spectral analysis was done calculating for each patient, the percentage activity of the various frequency bands and the relative and absolute powers. Resulting values were compared to those of the controls.

5. Neuropsychological testing was undertaken using Luria Nebraska Neuropsychological Battery (LNNB) localization scales with a cut off point calculated according to the critical level equations. Such scales included:

- L1 for left frontal lesions
- L2 for left sensory-motor lesions
- L3 for left parieto-occipital lesions
- L4 for left temporal lesions
- L5 for right frontal lesions
- L6 for right sensory-motor lesions
- L7 for right parieto-occipital lesions
- L8 for right temporal lesions

6. Harris test for lateral dominance.

7. Barthel index (*Activities of daily living*) for the assessment of the degree of handicap.

8. Structured clinical interview based on DSM-III-R criteria for the diagnosis of premorbid personality (SCID-II).

A control group of 50 healthy individuals with no history of neurological or psychiatric disorder and matched for age, sex and educational level were assessed

by means of EEG, BEAM, LNNB (*localizing scales*), SCID-I (NP) and SCID-II.

Statistical procedures for data analysis included the mean, standard deviation, t-test, chi square with Yates correction and Spearman correlation coefficient.

Results Application of the SCID-II showed that 56 out of the 103 patients (54.4%) had a threshold psychiatric diagnosis and 11 patients (10.7%) had a subthreshold diagnosis - Table 1.

Table 1: Threshold and subthreshold diagnosis according to SCID II

Diagnosis	No.	%
Threshold		
Major depression or Dysthymia	37	35.92
Major depression	17	16.5
Dythymia	20	19.4
Anxiety Disorders	18	17.47
With major depression	9	8.73
Without major depression	9	8.73
Manic or Hypomanic syndromes	5	4.85
Psychotic Disorders	7	6.79
Major depression with psychotic feat	3	3.02
Non-affective psychosis	4	3.77
Conversion disorder	1	1.79
Total	56	54.36
Subthreshold		
Dysthymia	6	5.82
Generalized anxiety disorder	3	2.91
Agoraphobia (without panic)	1	0.97
Hypochondriasis	1	0.97
Total	11	10.67

Thirty-six patients (34.9%) did not show clinically relevant psychiatric symptomatology.

In the group of threshold diagnosis, the majority had a depressive disorder (66.1%), whether major depressive or dysthymic. This means that 35.9% of the total stroke patients population had a depressive disorder of whom 16.5% had a major depressive disorder and 19.4% had a dysthymic disorder. As to the prevalence of other psychiatric diagnoses among stroke patients, 18% had an anxiety disorder, 4.85% had a manic or hypomanic syndrome, 6.79% had a psychotic disorder whether major affective with psychotic features or non-affective psychosis and there was a single case of conversion disorder.

Patients with subthreshold diagnosis were 6 patients with dysthymia, 3 with generalized anxiety disorder, 1 with agoraphobia without panic and 1 with hypochondriasis. The rest of the patients did not show clinically relevant psychiatric symptomatology. In all patients, the period elapsing between the onset of the cerebrovascular accident and the examination time ranged between 4 and 12 weeks.

Application of the Harris test showed all patients to have left cerebral dominance.

The mean ages of the different groups of patients were as follows: patients with post-stroke depression 49.27 ± 9.29 years; patients with post-stroke anxiety 48.33 ± 10.10 years; patients with post-stroke mania 46.2 ± 3.38 years and patients with post-stroke psychoses 49 ± 5.47 years. As to stroke patients with no psychiatric symptomatology the mean age was 49.44 ± 9.2 years and that for the control group was 47.82 ± 8.3 years. There was no statistically significant difference between the different groups as regards their age. Similarly no significant difference existed between the groups as regards sex distribution or level of education.

Concerning premorbid personality, the prevalence of personality disorders, especially compulsive personality (whether threshold or subthreshold) was higher in stroke patients than in controls. Although compulsive traits were observed more in patients with post-stroke depression than in stroke patients without psychiatric symptomatology, yet the difference was not statistically significant ($\chi^2 = 1.14, p > 0.05$).

Regarding the level of physical disability, no difference has been noted between stroke patients with and without psychiatric symptoms ($\chi^2 = 0.9, p > 0.05$). Also no correlation has been observed between the severity of depression and the degree of physical handicap ($r = 0.24, 1 > 0.05$).

Table 2: CT Findings in the Different Patient Groups (%).

Site of lesion	Post-Stroke			Stroke	
	Depression	Mania	Anxiety	Psychosis with no	Psych. Symp.
Left Anterior	23 (62.16%)	..	1 (11.11%)	2 (50%)	11 (30.55%)
Left Posterior	1 (11.11%)	..	6 (16.60%)
Right Anterior	2 (5.4%)	3 (60%)	2 (22.22%)	2 (50%)	8 (22.22%)
Right Posterior	7 (18.9%)	2 (40.1%)	1 (22.22%)	..	6 (16.60%)
Brain Stem	2 (22.22%)	..	2 (5.55%)
Normal CT	5 (15.5%)	..	2 (22.22%)	..	3 (8.33%)

Depression was significantly more associated with left anterior lesions compared to stroke patients with no psychiatric symptoms ($\chi^2 = 9.2, p < 0.01$).

Manic syndromes were significantly associated with right hemispheric lesions ($\chi^2 = 13.2, p < 0.01$). There was no significant localization for patients with anxiety or psychoses following stroke.

Table 2 demonstrates the results of CT scan of patients with post-stroke depression, mania, anxiety, psychoses and stroke patients with no psychiatric symptomatology. The results showed that post-stroke depression is significantly more associated with left hemispheric (especially left anterior) lesions, while post-stroke manias more associated with right hemispheric lesion ($p < 0.05$). No significant difference was found for patients with post-stroke anxiety or psychoses.

As to the EEG findings, there was an increased incidence of right temporal epileptic activity in patients with post-stroke depression and right hemispheric lesion compared to stroke patients with right sided lesion and no depression ($\chi^2 = 4.51, p < 0.05$). Left temporal epileptic activity was observed in patients with post-stroke psychosis and left sided lesions on CT scan [statistical analysis was not suitable because of the small number of the sample (2 patients)].

As to the result obtained from BEAM, significant findings were:

In patients with left hemispheric anterior lesions and depression, there was a significant increase in delta activity in left temporo-parietal regions than in patients with left hemispheric lesions without depression, but in both groups, left frontal delta activity was increased significantly compared to control groups.

In patients with right hemispheric lesions and depression, no significant difference in BEAM was found compared to right hemispheric lesion patients without depression.

In patients with post-stroke mania, maximal dysfunction was noted in the right temporal region.

Figure 1 shows an example of a comparison drawn between patients with left hemispheric lesion with and without post-stroke depression on the one hand and controls on the other.

Results of the LNNB showed a correlation with BEAM findings in depressed patients. Patients with a left hemispheric lesion showed an impairment in scales reflecting left frontal, left sensory-motor and left temporal functions. The same pattern of dysfunction was also observed in the case of post stroke mania, and increased dysfunction in occipital scales bilaterally in anxious patients. Patients with post-stroke psychosis did

not show a characteristic pattern of dysfunction on the Luria battery.

Figure 1 Beam profile in patients with left hemispheric lesion with and without post stroke depression as compared to controls: Values are distributed according to corresponding electrode placement according to 10-20 systems.

R					L				
Fp2					Fp1				
2.40					2.61*				
F8	F4	F2	F3	F7	F8	F4	F2	F3	F7
1.64	1.90	3.64*	3.94*	2.51*	1.64	1.90	3.64*	3.94*	2.51*
T4	C4	C2	C3	T3	T4	C4	C2	C3	T3
1.08	1.44		4.90*	2.74*	1.08	1.44		4.90*	2.74*
T6	P4	P2	P3	T5	T6	P4	P2	P3	T5
1.52		4.03*		3.05*	1.52		4.03*		3.05*
O2	O1				O2	O1			
2.41	2.30				2.41	2.30			

"t" values for patients with stroke due to a left hemispheric lesion and depression (compared to controls).

"t" values for patients with stroke due to a left hemispheric lesion without depression (compared to controls).

* = Significant at a level of $P < 0.05$

TABLE 3 Comparison of LNNB scores above the critical level in patients with post-stroke depression and stroke patients without psychiatric symptomatology in relation to controls.

Scale	Stroke patients with left hemispheric lesion and depression		without psychiatric symptoms
L1 left Frontal	18.7 ± 1.22 P < 0.01	11.1 ± 15.04 P < 0.01	
L2 Secondary motor	7.65 ± 10.39 P < 0.01	3.6 ± 5.18 P < 0.01	
L3 left Parieto Occipital	13.24 ± 14.54 P < 0.01	1.6 ± 9.5 P < 0.05	
L4 left Temporal	5.06 ± 7.77 P < 0.02	1.1 ± 8.2 NS	
L5 Right Frontal	0.94 ± 3.78 NS	0.9 ± 7.3 NS	
L6 Right Secondary motor	2.76 ± 7.4 NS	0.4 ± 9.2 NS	
L7 Right Parieto-Occipital	3.7 ± 9.8 NS	0.2 ± 10.1 NS	
L8 Right Tempora	4.7 ± 8.4 P < 0.05	0.3 ± 9.1 NS	

Table 3 reveals the results of the comparison drawn between patients with left hemispheric lesion with and without depression on the one hand and controls on the other on the different LNNB scales.

Discussion The association of post stroke depression (16.5% for major depression and 19.45% for dysthymia) with left hemispheric lesions (cortical and subcortical) agrees with several other studies (Lipsey et al 1983, Robinson et al 1984, Starkstein and Robinson 1989, Eastwood et al 1989, Stern and Backman 1991). Reference was also made to the importance of the proximity of the lesion to the frontal pole as a correlating factor to the presence and severity of depression (Starkstein et al 1987 Starkstein et al 1988). In a study by Rederoff et al, it was suggested that the mechanism of depression following anterior brain injury may involve the interruption of biogenic amine containing neurons as they pass through the basal ganglia or frontal sub-cortical white matter (Fedoroff, et al. 1992). However, other studies found no support for the association of depression, neither with left nor with frontal lesions. (Dam et al. 1989, House et al. 1991, Sharp et al. 1990). It is to be mentioned that methodological variations between those studies do exist as regards the tools and criteria for diagnosis, patient selection, the time elapsing between the occurrence of stroke and evaluation and considerations of other contributing factors.

Another important point is that most of the previous studies used only CT scanning for studying the site of dysfunction. This is another limitation to be taken into account. In the present study other dimensions for studying the site of dysfunction have been considered in order to give more precise illustration. The degree of cerebral dysfunction, as demonstrated by BEAM, could not be exactly correlated with the structural lesion demonstrated by CT. Although brain mapping demonstrated global and intense cerebral affection in most patients with major depression, CT showed only small, deeply seated lesions in a great number of patients.

EEG dysfunction shown in 64.7% of patients could be correlated to the resulting brain ischemia. However, this is not consistent with the extent of damage shown in CT scan. This agrees with the findings of other studies which showed significant correlation between EEG slowing and brain ischemia (Miyake, et al. 1986, Suzuki, et al 1986). The EEG findings may offer an explanation of the depressive symptomatology following right hemispheric stroke. In those cases, the affective symptoms may be secondary to the epileptic activity in

the right temporal area rather than the ischemic changes themselves.

Our findings in the case of post-stroke mania (4.8% of stroke patients), are consistent with those of Starkstein and Robinson, who found a relationship between post-stroke mania and orbito-frontal and baso-temporal cortical lesions, in addition to the thalamus and head of caudate (Starkstein and Robinson 1989). Seizure activity was reported only in one patient: This is in disagreement with the possibility that the pathogenesis of post-stroke mania may involve seizure foci without hemispheric specificity.

The findings in patients with post-stroke anxiety (17.47% of stroke patients in our study) together with the clinical examination pointed to brain stem activity in a number of those patients. This may be part of vertebro-basilar insufficiency with brain stem ischemia, which might explain the bilateral occipital dysfunction noted in LNNB. The link of brainstem to autonomic activity arousal and anxiety is to be noted.

Our results in the case of non-affective psychoses (3.77% of stroke patients) regarding the involvement of both hemispheres are not supported by other studies. Levine and Finklestein, for example, reported a relation between psychotic illness after stroke and right hemispheric lesions (Levine and Finklestein 1982). However, patients with left hemispheric lesions in the present study were also found to have temporal epileptic activity and have different symptomatology than right hemispheric lesions patients. The symptomatology in patients with left hemispheric lesions was dominated by delusions and hallucinations, preserved affect and no disintegration of the personality. In patients with right hemispheric lesions psychosis was characterized by impaired conceptualization, thought and memory disturbance. This finding may suggest a different pathogenic mechanism for post-stroke psychosis following right and left hemispheric lesions.

A comment has to be made regarding stroke patients with left hemispheric lesion who did not show either depressive or any other psychiatric symptomatology and who constituted 18.44% of our stroke patients. A general observation was that the extent of dysfunction in those patients by BEAM and LNNB was less than that observed in patients with left hemispheric lesion and depression. This finding could suggest that depression might be associated with more extensive cerebral dysfunction. The conclusion could then be that psychiatric morbidity following stroke is not only related to the site, but also to the extent of dysfunction.

It should be finally mentioned that the site of dysfunction should always be regarded as a "vulnerability" factor. If it had been an exclusively causal factor, a question would be justified as to why not all stroke patients with the same dysfunction develop the same psychiatric symptoms.

The application of SCID II for the assessment of premorbid personality in our sample of patients revealed some association with compulsive traits or obsessive compulsive personality pattern, especially in patients with post-stroke depression. Could this personality pattern constitute a vulnerability factor? Federoff et al. suggested that premorbid vulnerability factors that may operate in the development of depression among patients with traumatic brain injury may include premorbid personal psychopathology (Federoff, et al. 1992). The same factor could operate in the case of stroke. Other vulnerability factors could include coping strategies, family history and any other associated medical conditions, each of which should be taken into consideration, a matter left to further investigation.

In the present study, the pathology of the cerebrovascular insult had an effect on the psychiatric symptomatology e.g. ischemic lesions were observed to be associated more with psychiatric symptoms than hemorrhagic lesions, especially in cases of post-stroke depression and left hemispheric lesions. A possible explanation would be that ischemic lesions tend to be associated with more widespread cortical dysfunctions due to the resulting oedema, especially at the beginning. This is in contrast to hemorrhagic lesions, which have a more localized dysfunction. Hemorrhagic lesions, on the other hand, were observed to be associated with psychiatric symptoms mainly when accompanied by epileptic activity. Thus the pathogenesis of psychiatric symptoms might be somewhat different depending on the nature of the pathology.

We could suggest that in cases where the CT failed to show positive findings, neuro-physiological and neuro-psychological tools were able to reveal demonstrable localization of dysfunction. This reflects one advantage of the multi-dimensional approach in diagnosis. Despite the fact that localization by CT was mainly limited to certain circumscribed areas, yet a broader extension of dysfunction could be shown by BEAM and LNNB which were nicely correlated with each other. This may point to a better and more precise understanding of the pathophysiological mechanisms underlying psychiatric symptoms in those patients. On the other hand, BEAM and LNNB fail to demonstrate subcortical localizations. Despite the fact that conventional EEG gave less valuable information regarding localization, yet it is

highly indicated in detecting epileptic activity and its origin which may have some relation to resulting psychiatric symptomatology with subsequent alternation of management. It is suggested that no single tool is meant to replace the other. The multi-dimensional approach however can provide a more satisfactory picture of the dysfunction. Furthermore, the specificity of each tool may help in the detection of a particular vulnerability factor which in every individual patient may be the key factor to a particular clinical presentation.

References

- Dam H.; Pederson H.E.; Ahlgren P. et al. (1989):* Depression among Patients with Stroke. *Acta. Psych. Scand.* 80:118-124.
- Eastwood M.; Rifat S.; Nobbs H. et al. (1989):* Mood Disorders Following Cerebrovascular Accident. *Brit. J. Psychiat.* 134: 195-348.
- Fedoroff P.; Starkstein S.; Forrester A. et al. (1992):* Depression in Patients with Acute Brain Injury. *Am. J. Psychiatry*, 149:918-923
- Folstein M.F. (1977):* Maiberger, R., and McHugh P.R.: Mood Disorders as a Specific Complication of Stroke *J. of Neurol., Neurosurgery, and Psychiatry.* 40:1018-1020.
- House A.; Dennis M.; Mogridge L. et al. (1991):* Mood Disorders in the Year After the First Stroke. *Brit. J. Psychiat.* 158:83-92.
- Levine D. and Finklestein S., (1982):* Delayed Psychosis after Right Temporo-Parietal Stroke or Trauma: Relation of Epilepsy. *Neurology*, 32 267-273.
- Lipsey J.R.; Robinson R.G.; Pearlson C.; et al. (1983):* Mood Change Following Bilateral Hemisphere Brain Injury. *Br.J.Psychiat.* 143: 266-273.
- Llyod G.G.(1977):* Psychological Reactions to Physical Illness. *Brit. J. of Hospital Med.* October 352-358.
- Miyake H.; Okade M.; Nakagawa Y. et al. (1986):* Observation of EEG changes during Main Cerebral Arterial Occlusion. In Matsuoka S.; Soejima T. and Tokota A.. (Eds.) "Clinical Topographic Electrography and Evoked Potentials" Shindars To. Chinyo. Co. Lts. Tokyo
- Robinson R.G. (1987):* Depression and Stroke. *Psychiatric Annals* 17: 731 -740.
- Robinson R.G.; Kenneth L.K.; Lyn B.S. et al. (1984):* Mood Disorders in Stroke Patients; Importance of location of Lesion. *Brain* 107: 81-93.
- Sharp M.; Hawton K.; House A. et al. (1990):* Mood Disorders in Long-term Survivors of Stroke: Association with Brain lesion, Location and Volume. *Psycholoaical Medicine* 20: 518-528.
- Starkstein S.E.; Robinson R.G. and Price T.R. (1987):* Comparison of Cortical and Subortical Lesions, in the Production of Post-Stroke Mood Disorders. *Brain*; 110: 1045-1059.
- Starkstein S.E; Robinson R.; Berlhier M.L.; Parikh R.M. and Price T.R. (1988):* Differential Mood Changes Following Basal Ganglia versus Thalamic Lesions. *Arch. Neurol*; 45: 725-730.
- Starkstein S.E. and Robinson R.G. (1989):* Affective Disorder and Cerebrovascular Disease. *Brit. J. Psychiat.* 154: 170-182.
- Stern R.A. and Backman D.L. (1991):* Depressive symptoms Following Stroke. *Am. J. Psychiat.* 148 (3): 351-356.
- Suzuki, A.; Yasuri N.; and Vashioka K., (1986):* The EEG Slowing in Basal Ganglionic Infarction. In: Matsuoka S. Soejima T. and Tokota A. . (Eds.) *Clinical Topographic Electro-encephalography and Evoked Potential*" Shindars To. Chinyo. Co. Ltd. Tokyo.

AUTHORS

Okasha A.

Professor and Head of Neuro-Psychiatric. Department, Faculty of Medicine, Ain Shams University

Kamel M.

Professor of Psychiatry, Faculty of Medicine, Ain Shams University

Ashour S.

Lecturer of Neurology, Faculty of Medicine, Ain Shams University

Zakaria M.

Lecturer of Neurology, Faculty of Medicine, Ain shams University

Seif EL Dawla A.

Lecturer of Psychiatry, Faculty of Medicine, Ain Shams University

Asaad T.

Lecturer of Psychiatry, Faculty of Medicine, Ain Shams University

ADDRESS OF CORRESPONDENCE

Prof. A. Okasha

Chairman of Neuropsychiatric Dept., Faculty of Medicine, Ain Shams University, Abbasia, Cairo, Egypt.

الاضطرابات النفسية فى مرضى حوادث الأوعية الدموية المخية - دراسة متعددة الجوانب

تشيع الاضطرابات النفسية فى الفترة التى تعقب حوادث الأوعية الدموية المخية. وهناك تباين كبير بالنسبة لطبيعة و كيفية حدوث مثل هذه الاضطرابات و علاقاتها بموضوع الخلل الناجم بالجهاز العصبى.

و لقد تم فى هذا البحث استخدام دراسة متعددة الأوجه لتحديد موضع الخلل بصورة أكثر دقة فى هؤلاء المرضى باستخدام التصوير المقطعى للدماغ بواسطة الكمبيوتر مع رسام المخ الكهربائى و المسح الطبوغرافى للمخ باستخدام الكمبيوتر مع بطارية لوريا-نبراسكا للفحص النفسعصبى (النفـس-عصبى). وأجريت الدراسة على ١٠٣ من مرضى حوادث الأوعية الدموية المخية مع ٥٠ شخصاً من الأصحاء كعينة ضابطة ، و تم تشخيص المرضى باستخدام مقابلة إكلينيكية مبنية على الدليل الأمريكى الإحصائى لتشخيص الإضطرابات النفسية - الجزء الثالث المراجع - الجزء الخاص بغير المرضى.

ولقد أظهرت النتائج شيوع الإضطرابات النفسية بعد حوادث الأوعية الدموية المخية (خاصة الناتجة عن قصور الدورة الدموية أكثر من النزيف) و تبلغ نسبتها أكثر من ٥٠٪ معظمهم يعانون من الاكتئاب (١٦,٥٪ اضطراب وجدانى اكتئابى، و ١٩,٥٪ اضطراب عسر المزاج). و شملت التشخيصات الأخرى القلق، الهوس ، الإضطرابات الذهنية غير الوجدانية والاضطراب التحولى.

وقد لوحظ وجود علاقة بين الاكتئاب واضطرابات الفص الأيسر بينما يكثر الهوس مع اضطرابات الفص الأيمن فى حين لا توجد علاقة واضحة فى مرضى القلق مع موضع معين للإضطراب الوظيفى فى حين كانت الاضطرابات الذهنية مصحوبة بنشاط صرعى فى الفص الصدغى الأيسر.

ومن ناحية أخرى وجد أن هناك علاقة للإضطرابات النفسية مع حجم الخلل الوظيفى وليس فقط موضعه، فى حين تعتبر الشخصية قبل المرض عاملاً مساعداً لحدوث كل من إضطراب الأوعية الدموية المخية والإكتئاب فى الوقت نفسه.

وتناقش هذه الدراسة أهمية استخدام مدخل متعدد الأوجه لدراسة موضع الخلل الوظيفى فى هؤلاء المرضى.

616-082

A Comparative study of inpatient psychiatric service in a private-versus- University General Hospital* in an Arab culture

Lotaief F., Ghanem M., El Mahalawy N., Asaad T., Kahala H.

Abstract

The present study compares the nature of psychiatric service in two different hospitals in an Arab culture (Saudi Arabia); the first is a general private hospital, where the patient pays for himself and the second is a university Hospital, where no direct payment is required from the patient and teaching aspects are considered. The results showed an increased number of psychiatric patients in the University Hospital all over the year of study, with more prevalence of males and younger age groups. More length of stay was found in the University hospital. The nature of psychiatric diagnosis does differ in the two hospitals. Possible explanations for such variation as well as difference in hospital system organizations have been discussed.

Introduction For the last years, there has been an increasing argument about the need for hospital beds for psychiatric patients. The type of mental patients to be admitted and the nature of services supplied to them, all are matters for debate in the focus of cost containment strategies (Knapp and Beechman, 1986). A movement raising the slogan of "psychiatry without hospital beds" or "alternative psychiatry" has been progressively growing (Tyrer and Malone, 1992).

On discussing such a dilemma of hospital versus community treatment, it is worth noting that several factors other than the clinical psychiatric diagnosis do affect the decision of hospitalization and the type of service provided, including cultural, social, economic and other factors. Another important aspect is that hospital system organizations are not one and the same, but they do differ as regards ownership, profit orientation, affiliation and patient population (Kaplan and Sadock, 1991).

They may be general or special psychiatric, government or private, in addition to other hospitals, where teaching purposes are considered.

The present study is meant to compare the nature of psychiatric service in the different hospitals in Saudi Arabia: the first is a general private hospital, where the patient pays for himself, and the second is a university hospital where no direct payment is required from the patient and teaching aspects are considered.

Subjects Psychiatric diagnoses were followed in the two hospitals (private and university), through the period from November 1991 to November 1992.

For each patient, demographic data were collected including age, sex, race, marital status, residency and educational level. The "race" is considered as the society of S.A. is a heterogeneous one in terms of ethnicity and consequently financial status (i.e. involve persons from different communities coming to S.A. to improve their financial status.).

Only the final diagnosis, which is the diagnosis on discharge, was taken into account, as it differed in some cases from the provisional or admission diagnosis. Diagnosis was done according to the DSM-III-R criteria (1987).

The duration of treatment or length of stay in hospital was considered for every patient as well as the condition on discharge whether improved, deteriorated or the same as admission.

The system of psychiatric service regarding facilities, personnel number and qualification was compared in the two hospitals.

* Fakhry Hospital, Al Khobar, Eastern province Saudi Arabia

** Psychiatric department, Medical School, King Faisal University, Eastern province, Saudi Arabia.

Results

Table 1: Hospital system organization

	University Hospital	Private Hospital
(a) Beds		
1. No. of psychiatric beds	18 (constant)	Variable (only 10 constant)
2. Special or isolated beds	Male ward: 10 beds Female ward: 8 beds	isolated male ward: 10 beds (mostly for substance abuse patients). distributed in various other beds (not special psychiatric beds).
(b) Available investigations	EEG C.T. Scan MRI Psychometric testing	EEG CT.Scan Psychometric testing
(c) Available treatment theatre modalities	- ECT use is restricted done in operation theatre. - Psychotropics: limited varieties. - Psychotherapy, group therapy, behavior therapy: limited	- ECT more openly used. - Psychotropics: larger varieties - Psychotherapy, behavior therapy: and group therapy, more openly used
(D) Staff personal		
1. Consultants		
Number	1	4
Qualification	MD. degree in Psychiatry	MD. degree in Psychiatry or MRCP
2. Specialists		
Number	1	1
Qualification	Master degree in Psychiatry	Master degree in Psychiatry
3. Residents		
Number	1	8
Qualification	-	Diploma in psychiatry
4. Nurses		
Number	13	8
Sex	6 (males): 7 (females)	4 (males): 4 (females)
Race	Only few are non Arab.	Good % are non S.A.
5. Psychologists	1	9
6. Social worker	-	1

Statistical analysis of the data was done using the mean, standard deviation and chi square, and student t-test comparing the results obtained from the two hospitals.

Table 2: Demographic Data

A. Age

Age Distribution	Private Hospital		University Hospital	
	No.	%	No.	%
<20 yrs	105	18.61%	20	6.94%
20-30 yrs	159	28.19%	73	25.34%
30-40 yrs	126	22.34%	111	38.45%
40-50 yrs	90	16.48%	54	18.75%
>50 yrs	81	14.36%	30	10.41%
Total	564		288	

The difference between the two groups is statistically significant ($P < 0.05$).

B. Sex

Sex	Private Hospital		University Hospital	
	No.	%	No.	%
Male	385	68.26%	156	54.17%
Female	179	31.73%	132	45.83%

The difference between the two groups is statistically significant ($P < 0.05$).

C. Race / Nationality

Nationality	Private Hospital		University Hospital	
	No.	%	No.	%
S.A.	555	98.40%	274	95.13%
Non S.A.	9	2.60%	14	4.86%

The difference was not statistically significant ($P > 0.05$).

Table 3: Psychiatric Diagnosis

Diagnosis	Private Hospital		University Hospital		χ^2/P
	No.	%	No.	%	
1)Mood disorder (depression)	119	21.09%	89	30.90%	$\chi^2=2.69$ ($P>0.01$)
2)Mood disorder (Mania or hypomania)	11	1.95%	24	8.33%	$\chi^2=3.78$ ($P<0.01$)
3)Schizophreniform	51	9.04%	74	25.69%	$\chi^2=8.8$ ($P>0.01$)
4)Acute psychosis (brief reactivity and schizophreniform)	10	1.77%	12	4.6%	$\chi^2=0.98$ ($P>0.01$)
5)Atypical and other psychoses than schizophrenia	9	1.5%	5	1.73%	($P>0.01$)
6)Substance use disorder	211	37.41%	21	7.29%	$\chi^2=28$ ($P<0.01$)
7)Generalized anxiety disorder	94	16.66%	10	3.47%	$\chi^2=8$ ($P<0.01$)
8)Obsessive compulsive disorder	6	1.06%	10	3.47%	($P>0.01$)
9)Panic disorder	2	0.35%	0	-	
10)Phobic disorder	9	0.33%	0	0	
11)Somato-form disorder	11	1.95%	11	3.81%	($P>0.01$)
12)Organic brain syndrome (dementia and delirium)	10	1.77%	11	3.81%	($P>0.01$)
13)Adjustment disorder	20	3.54%	3	1.04%	($P>0.01$)
14)Epilepsy	5	0.88%	6	2.08%	($P>0.01$)
15)Personality disorder	2	0.35%	7	2.4%	($P>0.01$)
16)Extrapyramidal side effects	0	0%	2	0.69%	-
17)Anorexia nervosa	0	0%	1	0.34%	-
18)Conduct disorder	0	0%	1	0.34%	-
19)Factitious disorder	1	0.17%	0	0%	-
Total	564	100%	288	100%	

Length of stay in hospital.

The mean duration of hospital stay in the private hospital was 13 ± 5.34 days, while in the university 20 ± 6.05 days. The difference was significant ($P<0.05$).

Table 4: Condition on Discharge

Condition on discharge	Private Hospital		University Hospital		P
	No.	%	No.	%	
1. Improved	475	84.21%	223	77.43%	< 0.05
2.Deteriorated	18	3.19%	17	5.90%	< 0.05
3. No. charge	71	12.58%	48	16.66%	< 0.05

Discussion The overall results of the present study do suggest a different profile for psychiatric service in both types of hospitals. The number of patients in the private hospital was nearly double that in the university hospital, which might be attributed to more length of hospital stay in the university hospital. The more length of hospital stay in a university hospital could have several explanations, because long stays in a private hospital is costly from the patient's perspective and rapid turnover is more favorable for profit-oriented private hospital system. Furthermore, patients might be kept in university hospital for teaching purposes, in addition to treatment goals, and availability of beds in the private hospital, where the number of beds was not limited as in the university hospital. It can also be explained by the fact that in state hospital, psychiatrists are less aware of the importance of quick improvement of the state of their patients, and more cautious in using medications for fear of side-effects as well as rapid turnover of patients may cause extra duties and excess work which will add more responsibilities over and above their teaching duties, while in the private hospital cure of patients is the main goal, so new biochemical lines of treatment are available and used as a routine to salvage the suffering souls of their patients. It is observed that psychiatric staff in university hospitals are so cautious to the extent of using the old traditional drugs of the fifties in very small doses.

Second, regarding demographic data, younger age groups as well as males were found more in the private hospital. A possible explanation for both findings could be due to the fact that the private hospital tolerates much more number of patients, which is not the system followed in the university hospital, where neurotic

patients are rarely admitted and only in certain circumstances (e.g. emergency cases, associated psychiatric morbidity, substance abuse...). Substance abuse is known to be commonly young adult males (National Institute on Drug Abuse, 1988). The presence of higher percent of non-Saudi Arabians in the university hospital is explainable by the fact that higher costs of the private hospital repel the foreigners who have come to Saudi Arabia to save money not to spend a lot on treatment costs.

Third, regarding the condition on discharge, more improvement was observed in private hospital. This does mean more effective treatment, and variation in the types of patients admitted from the start. Private hospitals tend to accept cases with encouraging at least short-term prognosis, where university hospital accepts the so called "bad" cases. The dropouts of private hospitals tend to stay more in university hospitals, with need of less effort and their families will not be demanding their discharge, and lastly there will be accepted excuse for their bad prognosis.

Fourth, as regards the nature of psychiatric diagnosis or types of patients admitted, there are significantly more substance abuse patients, more patients with anxiety disorders, with less chronic and manic or hypomanic syndromes in the private hospital. The more prevalence of anxiety disorder might reflect the possible tendency for facultative admission in private hospital, as well as comorbidity of such disorders with substance abuse, which is much more in private than in university hospital. The finding of less schizophrenia could be attributed to the fact that only acute cases of schizophrenia are admitted to private hospital i.e. those with possible favorable prognosis. Chronic as well as bad prognosis cases who need longer periods of hospitalization, and thus more costly are not good candidates for private hospitals.

Regarding substance abuse, it is to be mentioned that cases were to be admitted either from the emergency department during night time and they did not stay more than one night or the diagnosis of drug addiction was revealed later either through the patient himself or through investigations. Some of these cases are not diagnosed from the original screening, but with detailed data collection by the researchers during the preparation for this study.

References

Knapp, M. and Nee Cham J. (1989): The cost-effectiveness of community care for former or long stay Psychiatric hospital patients. Discussion paper 628/2,

Personal Social Services Research Unit. University of Kent, Canterbury.

Tyner P. and Malone S. (1992): Psychiatry without hospital beds: A review of treatment strategies. In Recent Advances in Clinical psychiatry number seven. Granville-Grossman K., Churchill Livingstone, Edinburgh, London, Melbourne, New York and Tokyo.

National Institute on Drug Abuse (1988): National Household Survey on Drug Abuse, 1989.

Kaplan H.I. and Sadock B.J. (1991): Synopsis of psychiatry. Sixth edition. Williams and Wilkins. Baltimore, Hong Kong, London, Munich, San Francisco, Sydney, Tokyo. Page 150-152.

Diagnostic and statistical Manual of Mental disorder ed. revised (1987): The American Psychiatric Association, Washington D.C.

Data collected from Fakhry Hospital - Al Kohbar and King Faysal Hospital in Saudi Arabia.

AUTHORS

Lotaief F.

Professor of Psychiatry, Faculty of Medicine, Ain Shams University.

Ghanem M.

Assistant professor of Psychiatry, Faculty of Medicine, Ain Shams University.

El Mahalawy N.

Assistant professor of Psychiatry, Faculty of Medicine, Ain Shams University.

Asaad T.

Lecturer of Psychiatry, Faculty of Medicine, Ain Shams University.

Kahala H.

Fakhry Hospital, AL-Khobar, Saudi Arabia

ADDRESS OF CORRESPONDENCE:

Prof. F. Lotaief

Institute of Psychiatry, Ain Shams University, Abbassia, Cairo, Egypt

دراسه مقارنه إكلينيكية للخدمة الطبفسية للأقسام الداخلية فى مستشفى جامعية ومشفى خاصة فى بيئة عربية

تقارن الدراسة الحالية بين طبيعة الخدمة الطبفسية المقدمة للمرضى فى مستشفين مختلفتين من بيئة عربية (المملكة العربية السعودية) إحداهما مستشفى خاصة حيث يقوم المريض بتحمل تكاليف علاجه والأخرى مستشفى جامعية حيث لا يتحمل المريض تكاليف علاجه، وتدخل أغراض التدريس فى الإعتبار.

لقد أظهرت النتائج وجود عدد أكبر من المرضى فى المستشفى الخاصة خلال سنة الدراسة مع وجود نسبة أكبر من الذكور وصغار السن. ولوحظ أيضا فى المستشفى الخاصة مكوث المرضى بالمستشفيات لفترة أقصر، كما لوحظ أيضا وجود اختلافات فى طبيعة المرضى من ناحية التشخيص الإكلينيكى. وأعقبت الدراسة مناقشة للأسباب المحتملة للاختلاف مع عرض لنظام العمل بالمستشفين.