

The Dexamethasone Suppression Test Findings in Psychiatric Inpatients[†]

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Abstract—83 subjects (20 normal controls, 15 neurotics, 19 major depressives and 29 paranoid schizophrenics) underwent the dexamethasone suppression test (DST) to test the applicability of the test for the diagnosis of major depression. Pre- and post-dexamethasone serum cortisol concentrations were determined in duplicate by radioimmunoassay at 8:00 a.m., 4:00 p.m. and 11:30 p.m. Significantly decreased suppressibility of serum cortisol levels were found in the major depressives at 8:00 a.m. and 4:00 p.m. in the DST and the cortisol suppression index (CSI), compared with those in the normal controls. No significant differences were found in the other comparisons. The nonsuppression rate in the major depressives was 47%. And due to the significant nonsuppression rates (14%-22%) in the other groups, the specificity of the nonsuppression in the DST needs to be further clarified before its significance is established. In the meantime, it is recommended that multiple chronological DSTs be performed during the clinical course and other intervening variables should be considered for meaningful interpretation.

Key words: *Cortisol, Cortisol suppression index (CSI), Dexamethasone, Dexamethasone suppression test (DST), Major depression, Neurosis, Paranoid schizophrenia*

INTRODUCTION

Recent adoption of the dexamethasone suppression test (DST), originally developed for diagnosing Cushing's syndrome (Liddle 1960), in the diagnosis of major depression has followed the studies (Carroll *et al.* 1968; Carroll *et al.* 1976; Carroll *et al.* 1980) on the hypothalamo-pituitary-adrenal (HPA) functions associated with major depression. It has been maintained that major depression is frequently associated with increased activity of the HPA axis, indicated by the elevated basal plasma cortisol levels and disrupted 24 hour diurnal rhythm of cortisol secretion (Rubin and Mandell 1966; Sachar *et al.* 1973). Further investigation on these matters proved that the DST in some depressive patients is

characterized by nonsuppression and early escape (Carroll *et al.* 1968; Nuller and Ostroumova 1980; Sachar 1980). These findings are claimed not to be found in other mental disorders such as non-endogenous depression (Carroll *et al.* 1968; Brown *et al.* 1979; Schlessler *et al.* 1980; Carroll *et al.* 1980; Carroll *et al.* 1981). Thus, there are many who are trying to make use of the DST in classifying the depressives into subtypes (Brown and Shuey 1980), for evaluating the treatment outcome and predicting the prognosis (Brown *et al.* 1979). On the other hand, there are those who have done subsequent studies on DST, which showed inconsistent results with considerable nonsuppression in chronic schizophrenia, bipolar disorders, dementia, neurosis, alcoholism and other psychiatric disorders (Amsterdam *et al.* 1982; Dewan *et al.* 1982; Meltzer *et al.* 1982; Raskind *et al.* 1982; Spar and Gerner 1982; Swartz and Dunner 1982; Balldin *et al.* 1983; Castro *et al.* 1983; Coppen *et al.* 1983;

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Gwirtsman *et al.* 1983; Krishnan *et al.* 1983; Sheehan *et al.* 1983; Targum 1983; Lindy *et al.* 1985). Thus, the specificity of the DST in diagnosing major depression is subject to challenge.

In this paper, we present our experience with the DSTs that have been done to evaluate the diagnostic implication of the DST in psychiatric inpatients.

MATERIALS AND METHODS

63 patients consecutively admitted to the Department of Neuropsychiatry of Seoul National University Hospital and 20 healthy controls were evaluated. There were 19 major depressives, 29 paranoid schizophrenics and 15 neurotics (conversion disorder, somatization disorder, dysthymic disorder, adjustment disorder and obsessive compulsive disorder). Diagnoses were made according to the DSM-III (American Psychiatric Association 1980) criteria and without knowing the results of pre- and post-dexamethasone cortisol measurements. Agreement was made between the authors as to the diagnosis before accepting a patient as a study subject. Patients were excluded from the study if they had a significant medical, neurological, or endocrinological disorder. They were also excluded if they received any of the drugs influencing the result of the DST such as phenytoin, barbiturates, carbamazepine, thyroid hormones, narcotics, and high dose of benzodiazepines. Any patient having recently received electroconvulsive therapy was excluded. The age of these 63 patients (23 males and 40 females) ranged from 16 to 72 years.

The control group consisted of 20 medical and nursing students who had no proven physical and mental illnesses. The age range of the control group (10 males and 10 females) was from 21 to 25 years.

Blood samples for pre-dexamethasone serum

cortisol level were obtained by venipuncture at 8:00 a.m., 4:00 p.m. and 11:30 p.m. Then, 1 mg of oral dexamethasone was administered at 11:31 p.m. On the following day, blood was obtained for assay of post-dexamethasone serum cortisol at 8:00 a.m., 4:00 p.m. and 11:30 p.m. All the measurements were done in duplicate by radioimmunoassay technique (Amerlex^R).

A failure to suppress (nonsuppression) in the DST was defined by the presence of a cortisol level greater than or equal to 5.0 $\mu\text{g}/100\text{ ml}$ at one or more of the three time points. Cortisol suppression index (CSI) was defined as the ratio of a pre-dexamethasone cortisol level divided by the post-dexamethasone level measured at the same time of day (Bernstein *et al.* 1982).

Statistical analysis of the data was done using Student's t-test (two-tailed), Chi-square test, analysis of variance, and analysis of covariance.

RESULTS

The characteristics of the study subjects are as seen in Table 1. There was no significant difference in sex ($\chi^2 = 4.3$, d.f. = 3, $p > 0.05$) and weight ($F = 0.35$, d.f. = 3,73, $p > 0.05$, by analysis of variance) distribution among the 4 groups. The age distribution was significantly different among the 4 groups ($F = 6.6$, d.f. = 3,77, $p < 0.01$, by analysis of variance).

The distribution of nonsuppression rates among the study groups was as seen in Fig. 1. Of the 20 subjects in the control group, 4 failed to suppress on the DST. Of the 14 neurotics (a patient with missing post-dexamethasone data excluded), 2 failed to suppress. Of the 19 major depressives, 9 failed to suppress. Of the 27 paranoid schizophrenics (2 patients with missing data excluded), 6 failed to suppress. If failure to suppress on the DST was

Table 1. Demographic characteristics of the subjects

Demographic Characteristics	Normal	Neurosis	Major depression	Paranoid schizophrenia
No.	20	15	19	29
(M/F)	(10/10)	(4/11)	(5/14)	(14/15)
Age* (Mean \pm S.D.)	22.2 \pm 1.1	32.6 \pm 13.9	37.0 \pm 16.0	28.6 \pm 8.1
Weight (Mean \pm S.D.)	56.1 \pm 11.0	55.3 \pm 8.2	54.2 \pm 10.7	57.3 \pm 9.5

*significant difference among the 4 groups
($F = 6.6$, d.f. = 3,73, $p < 0.01$, by analysis of variance)

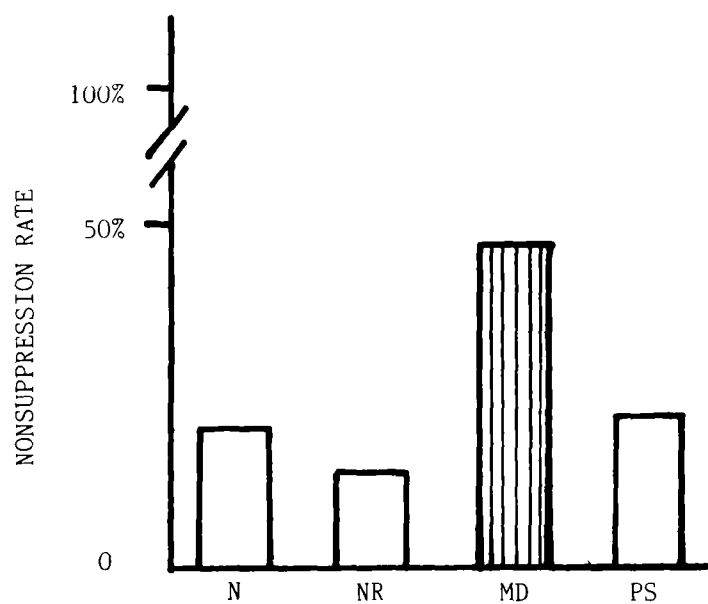


Fig. 1. Distribution of nonsuppression rates among the study groups.
 N : Normal controls
 NR: Neurosis
 MD: Major depression
 PS: Paranoid schizophrenia

used as a diagnostic marker for major depression in the patients studied here, the sensitivity would be 47% in the major depressives. The false positive rate of the DST in normal controls, neurotics and paranoid schizophrenics were 20%, 14% and 22% respectively (Fig. 1).

Pre- and post-dexamethasone cortisol levels were as seen in Table 2. Mean cortisol levels were apparently the highest in the depressed patients at each post-dexamethasone time point. However, there were no significant differences among groups

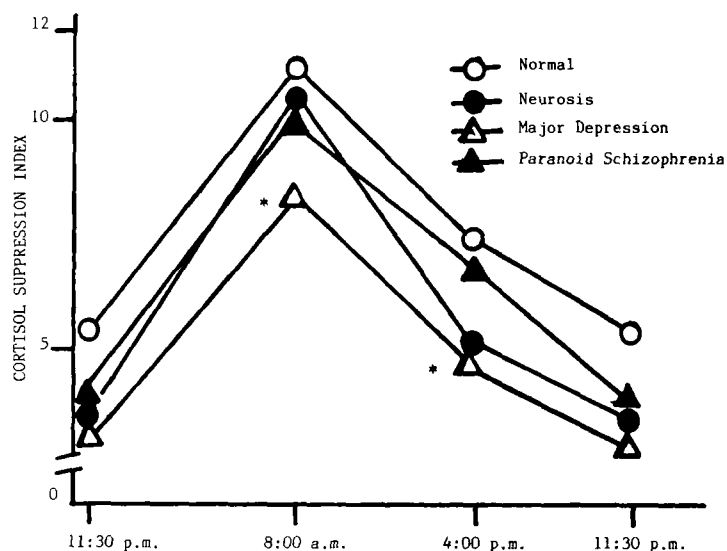


Fig. 2. Distribution of cortisol suppression indices (CSIs),
 *: statistically significant difference between normal controls and major depressives at the indicated time point ($p < 0.05$, by Student's t-test)

except the normal control subjects at 8:00 a.m. and 4:00 p.m. compared with the depressed ones (by Student's t-test, two-tailed). In order to eliminate the possible influence of the varying pre-dexamethasone cortisol levels as seen in Table 2 upon those after dexamethasone administration, analysis of covariance was done between each two groups and statistically significant differences were found between the normal controls and the major depressives at 8:00 a.m. and 4:00 p.m. No significant differences were found in the other comparisons (Table 2).

In multiple comparison of mean CSIs (Fig. 2), sig-

Table 2. Pre- and post-dexamethasone serum cortisol concentrations

Groups	No.	8:00 a.m.		4:00 p.m.		11:30 p.m.	
		Pre-D.	Post-D.	Pre-D.	Post-D.	Pre-D.	Post-D.
Normal	20	15.4 ± 4.3	1.5 ± 0.4 ^{d, †}	8.4 ± 2.6	1.6 ± 1.8 ^{e, *}	6.5 ± 3.6 ^c	1.9 ± 1.9
Neurosis	15	14.4 ± 6.5	1.5 ± 0.4	6.7 ± 3.0 ^{a, b}	2.3 ± 2.7	4.7 ± 5.1	1.8 ± 2.1
Major depression	19	18.5 ± 5.1	3.8 ± 4.4 ^{d, †}	10.6 ± 5.1 ^a	4.0 ± 3.8 ^{e, *}	5.7 ± 3.2	2.7 ± 2.3
Paranoid schizophrenia	29	17.7 ± 5.3	3.5 ± 5.2	9.6 ± 3.8 ^b	3.4 ± 4.3	4.5 ± 2.6 ^c	1.6 ± 1.5

a, b, c, d, e: each letter means statistically significant difference between the indicated (with the same letters) two groups ($p < 0.05$, by Student's t-test, two-tailed)

^{†, *}: each means statistically significant difference at the indicated time point between normal controls and major depressives ($p < 0.05$, by analysis of covariance)

Pre-D.: pre-dexamethasone Post-D.: post-dexamethasone

Table 3. Cortisol suppression indices(CSIs) in normal controls and psychiatric inpatients

Groups	N	8:00 a.m.	4:00 p.m.	11:30 p.m.
Normal	20	11.0±3.3 ^a	7.4±2.9 ^b	5.2±4.1
Neurosis	15	10.4±4.2	5.1±4.2	3.3±2.5
Major Depression	19	8.2±5.0 ^a	4.9±3.8 ^b	3.1±2.4
Paranoid Schizophrenia	29	9.9±4.6	6.7±4.5	3.8±3.1

a,b: each letter designates statistically significant difference between normal controls and major depressives at each indicated time point ($p < 0.05$, by Student's t-test, two-tailed)

nificant differences were found at 8:00 a.m. and 4:00 p.m. between the normal control and the major depressives(Table 3). This finding was consistent with the results calculated by the analysis of covariance(Table 2).

DISCUSSION

The possibility of neuroendocrine abnormality underlying depression has long been the subject of investigation. Evidence of cortisol hypersecretion reflecting the increased activity of the HPA axis has been demonstrated in depressive disorders by measuring plasma cortisol, urinary free cortisol, and urinary cortisol metabolites(Gibbons 1964; Sachar *et al.* 1970; Carroll *et al.* 1976; Ettigi and Brown 1977; Brown *et al.* 1979). One of the HPA abnormalities in depressed patients is the abnormal response to dexamethasone which is claimed to have high specificity(Carroll *et al.* 1981). Over the past 10 years there has been much enthusiasm about the utilization of the DST for the diagnosis of major depression. The practical value of the DST as a diagnostic tool in psychiatric practice is being emphasized. However, the clinical application of the research findings still has much to make clear. One of them would be that no relationship between severity of depression and resistance to dexamethasone suppression has been found(Carroll and Davies 1970). Caution should be used in interpreting those findings because there exist many intervening or confounding variables to be controlled before accepting those results blindly. In that sense our study results offer much to think about rather than stating a solid conclusion.

The effect of age on the DST deserves comment in this study because there is a statistically significant age difference among the groups. It seems that age is not likely to have exerted a significant

effect on the test result. All the subjects were below 50 years of age except 7 patients. The finding that advanced age did not appear to affect the overnight dexamethasone suppression in healthy humans(Tourigny-Rivard *et al.* 1981) may be supportive to the DST results of the non-age matched normal controls in this study.

Statistically significant differences of pre-dexamethasone cortisol level between some of the groups at 4:00 p.m. and 11:30 p.m. are not so simple as to be explainable. It needs further investigation. One of possibilities is that there may exist different way of cortisol secretion in each group. The significant differences of mean post-dexamethasone cortisol concentrations between the normal controls and the major depressives at 8:00 a.m. and 4:00 p.m. are consistent with the nonsuppression tendency in major depression. However, in our data the major depressives did not further distinguish themselves from the paranoid schizophrenics and the neurotics. Before taking them as a whole and coming to a conclusion that the DST is not a valid tool for diagnosing major depression, we should consider some possible influencing factors on the DST. We should look into the individual patient and see which factor in the patient might have affected the test results. Acute weight loss (Berger *et al.* 1983; Edelstein *et al.* 1983) or drugs could have influenced the results (Burch *et al.* 1986). Also, the diagnostic sensitivity of the cortisol measuring tool should not be taken for granted (Meltzer and Fang 1983; Carroll 1986). The non-suppression rate in the normal controls is 20% and it is higher than the 15.1% in Amsterdam and associates' study(1982). It may be due to the fact that we adopted one more time point than they and it might have contributed to the increased rate of nonsuppression. It could also have been a function of chronic stress prevalent in medical and nursing

students. The 22% of nonsuppression in paranoid schizophrenics is contrary to the findings of Rothschild *et al.* (1982) and Arana *et al.* (1983) that schizophrenic patients have not been shown to demonstrate similarly high rates of nonsuppression. It seems to imply that the abnormal DSTs in some of these patients may have been due to some external factors influencing cortisol regulation such as acute involuntary hospitalization or their psychopathologically peculiar alertness. Another factor could be some impairment of the liver's metabolic capacity by the chronic use of antipsychotics, prolonging the cortisol half life and thus elevating cortisol levels. Also, the diagnosis of paranoid schizophrenia, although done by strict criteria in DSM-III and intentionally chosen to rule out the affective component, may have been associated with some primary or secondary depressive symptoms. Depressive symptoms in schizophrenics are difficult to diagnose because of the characteristics of the schizophrenic symptoms and signs. Besides, numbers of environmentally related factors such as sleeping and eating pattern can affect cortisol regulation (Szwartz 1982).

The nonsuppression rate in major depression was 47% and it was the highest one among the 4 groups. The nonsuppression rate per se is comparable to those in other studies (Shapiro *et al.* 1983), but considering the significant nonsuppression rates in the other groups, especially in the paranoid schizophrenics, it raises questions about the potential utility of the DST as a diagnostic tool in the general psychiatric population as well as in the major depressives. The apparently high nonsuppression rate calculated in the neurotic patients deserves comment. Among the 14 patients, excluding the one without post-dexamethasone cortisol data, 2 patients showed nonsuppression. One had depressive symptoms and the other was a 16-year-old adolescent girl. Considering those factors, the 14% nonsuppression rate in the neurotic group is somewhat interpretable.

The CSI was originally adopted for making the DST more convenient and feasible especially for outpatients because it is enough for us to know only pre- and post-dexamethasone cortisol concentrations of one time point for calculating CSI. Blood sampling at 11:30 p.m. or 8:00 a.m. is impossible in outpatient service. It is a burdensome event even to an inpatient. The CSI findings in this study is consistent with those in Bernstein and associates' (1982) in that they discriminate the ma-

ajor depressives from the normal controls. However, its clinical application needs repeated replication in further studies.

The results of this study is never straightforward. It does suggest the presence of many variables intrinsic or extrinsic to the neuroendocrine research. The variables are subject to further clarification. The somewhat ambiguous results may reflect the overall present perspective of the DST in the diagnosis of major depressive disorder. Further research needs to be carried out with larger series of patients, additional age-matched normal controls and more strict control of the subjects and the study conditions in order to clarify the findings and hypotheses raised here. Thus, we may be able to define the true prevalence of DST abnormalities in adults with major depression and other psychiatric problems. It will further enable us to investigate the relationship between nonsuppression on the DST and pathophysiology of major depression, offering data on the prognosis and response to treatment modalities including antidepressant treatment.

In the mean time, care should be taken in administering and interpreting the DST in the clinical psychiatry and it is recommended that multiple DSTs be performed in a patient along the clinical course.

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= 국문초록 =

정신과질환에서의 Dexamethasone억제검사소견에 관한 연구

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정신과 질환중 주요우울병의 진단에 상당한 가치가 있다고 주장되고 있는 dexamethasone 억제검사를 그 진단적 유용성을 평가하기 위하여 63명의 정신과입원환자(주요우울병, 망상형 정신분열병, 신경증)와 20명의 정상인을 대상으로 시행하였다. 혈중 cortisol 기초치 및 억제 검사 후의 cortisol치를 방사면역법을 사용하여 오전 8:00시, 오후 4:00시 및 11:30시에 각각 측정하였으며 그 결과로부터 비억제율과 cortisol 억제지수를 산출하여 각 집단간을 비교분석 하였다. 정상대조군과 주요우울병 사이에는 억제검사소견 및 억제지수에서 오전 8:00시 및 오후 4:00시에 유의한 차이를 볼 수 있었으나 정신질환 각 집단별 비교를 비롯한 다른 비교에서는 상호 유의한 차이를 찾아볼 수 없었다. 또한 주요우울병에서의 비억제율은 47%로서 타 정신질환에서의 14%-22%보다는 높았으나 비억제에 의한 주요우울병 진단의 특이성은 검토의 여지가 있겠다. 따라서 dexamethasone억제검사를 주요우울병 진단에 사용하기 위해서는 1회 검사를 지양하고 병의 경과에 따른 반복검사가 필요한 것으로 생각되며, 혈중 cortisol치에 영향을 줄 수 있는 여러 변인들도 고려되어야 하겠다.