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## Psychiatric Morbidity in End Stage Renal Disease Patients on Haemodialysis: A Case Series Study

Rohan Kalra<sup>1</sup>, Govind S. Bhogale<sup>2</sup>, Narayan R. Mutualik<sup>3</sup>, Vinod A.<sup>4</sup>

### Abstract

**Background:** End-stage kidney or renal disease (ESRD) is often termed the last stage of chronic kidney dysfunction where in the kidneys are no longer in a position to function good enough to meet the needs of daily life. The usual treatments for ESRD are dialysis or kidney transplant. These patients are prone for various psychological stressors leading to various psychiatric illnesses. Present study was planned to find the psychiatric morbidity and the type of morbidity in patients with ESRD undergoing haemodialysis (HD).

**Methods:** A total of 40 consecutive patients diagnosed as having ESRD in dialysis unit of S.N. Medical College and HSK Hospital Research Centre, Bagalkot were evaluated using M.I.N.I. PLUS. Fischer's exact and Chi-square tests were used to check the association between two variables. Statistical analysis was done to get the results.

**Results:** 42.5% of patients with ESRD had psychiatric morbidity where major depressive episode and dysthymia constituted 32.5% and 10% of the sample respectively. Study participants were in the age group of 18 to 65 years, 70% were males and 30% were females. Post haemodialysis complication of infection was most common in 55% cases. But, none of the results were statistically significant.

**Conclusion:** Majority of ESRD patients do have some psychiatric morbidity commonest being major depressive disorder followed dysthymia. A multidisciplinary approach is essential necessary which will definitely improve prognosis of these unfortunate patients.

**Keywords:** End-Stage Renal Disease; Chronic Kidney Disease; Haemodialysis; Depression.

### Introduction

End-stage renal disease (ESRD) refers to a stage of chronic renal dysfunction in which uremic syndrome develops due to the accumulation of fluids, toxins and electrolytes which are normally excreted through kidneys. This may lead to death if the toxins are not eliminated by renal replacement therapy, using either the dialysis or renal transplantation [1]. The term chronic kidney failure represents the process of persisting significant irreversible decrease in the number of nephrons and typically corresponds to third till fifth stages in chronic kidney disease [2].

Haemodialysis (HD) is a physicochemical process involving single pass of blood and dialysis solution (dialysate) across a semi-permeable membrane. The solutes move across the membrane by diffusion, but water moves by ultra-filtration to reach a state of equilibrium [3]. Those membranes act as molecular size-selective filters and the size threshold is dependent on the nature of the membrane.<sup>4</sup> It is an artificial way of filtering blood which targets removal of toxins and excess fluids from the blood where in the blood is purified by an external artificial kidney machine. Patients will have to spend 3 to 4 hours for one haemodialysis session and needs 2-3 such

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sessions per week. Patients are often asked whether they perceive any reduction in the energy levels, any stressors leading to decrease in self-confidence levels, doubtfulness about their future, guilt towards other people in the family [5]. Levy introduced the term "psychonephrology", for such kind of mental health problems associated with patients having chronic kidney disease (CKD) with special reference to psychiatric problems in patients suffering from kidney disease, particularly those with kidney failure undergoing maintenance dialysis or renal transplantation [6].

For the sake of collecting the data to document and characterize the patterns of CKD, a registry had been set up in 2005 by the Indian Society of Nephrology. The most common cause was diabetic nephropathy followed by CKD of undetermined aetiology and chronic glomerulonephritis in the Indian Registry [7]. Chronic kidney disease (CKD) is emerging to be an important chronic disease globally possibly due to the rapidly increasing incidence of diabetes and hypertension worldwide [8].

A study by Rajapurkar M, reported the incidence of ESRD as 181 per million population in 2005 in central India and prevalence of CKD ranging from 0.79% to 1.4% [9]. According to GK Modi et al. average crude and age-adjusted incidence rates of End stage renal disease was 150 per million population in 2002, 143 per million population in 2003, 149 per million population in 2004 and 163 per million population in 2005 [10]. Of late, haemodialysis facilities are made available at many district hospitals in India with the help of many state governments. In addition all government hospitals in big cities, metros and even private hospitals are having such facilities. Therefore, it may be important to study the psychiatric morbidity in end stage renal disease cases on haemodialysis. The department of medicine in our kumareshwar hospital, Bagalkot, is having hemodialysis facilities having 5 beds, from 2005 onwards.

Psychiatric disorders are common among patients with chronic kidney disease (CKD) and these include depression, dementia, delirium, psychosis, anxiety, personality disorders and substance abuse. The commonest psychiatric disorder in patients with end-stage renal disease (ESRD) is major depressive disorder. The prevalence of depression in CKD patients has varied widely in different studies and different populations, using different assessment tools [11].

Patients on HD are prone to emotional problems possibly due to the chronic stress in relation to the burden of the disease, restrictions in food intake, financial limitations, impaired quality of life (QOL),

co-morbid chronic medical illnesses, medication induced side effects, and fear of dying [12,13]. Haemodialysis is a life sustaining treatment for patients with ESRD. This feeling of artificial life totally depending on every haemodialysis session could be acting as tremendous stress factor for patients with ESRD. Sleep related disorders among these patients are often related to disability, pain, duration of dialysis treatment sessions, increased creatinine and/or urea levels and somatic complaints like itching [14].

Chronic kidney disease patients on long term haemodialysis are under enormous stress. Nature of the stress could be physical, psychosocial, emotional and/or economical. These patients have the knowledge that they are on artificial life support, which itself might lead to persistent fear of dependency and disability. The investigator felt that psychiatric co-morbidity, sometimes hidden behind an array of vague symptoms could be affecting the outcome of treatment modalities in these patients. It is necessary therefore, to carefully assess the patients undergoing haemodialysis and treat them holistically to improve prognosis and quality of life in these unfortunate patients. With this background in mind the present study was undertaken.

#### *Aims and Objectives*

1. To find the psychiatric morbidity in patients with ESRD undergoing HD.
2. To study the type of psychiatric morbidity among ESRD patients undergoing HD.

#### **Materials and Methods**

This study is on Psychiatric morbidity in end-stage renal disease cases on haemodialysis who attended the dialysis unit of S.N. medical college and HSK hospital and research centre, Bagalkot.

#### *Study Design*

Descriptive cross sectional study.

#### *Sampling*

Patients with ESRD who are on haemodialysis.

#### *Sample Size*

M Rai et al. who conducted a study at state-run tertiary care hospital in New Delhi, found the

prevalence of depression in their study population to be 47.8% [15].

Sample size calculation was done by Open Epi trial version (2.3.1) software.

P = 47.8%

Absolute precision = 16%

Design effect = 1

At 95% confidence level

**Sample size**  $n = [\text{DEFF} * Np(1-p)] / [(d^2 / Z_{1-\alpha/2}^2) * (N-1) + p^2(1-p)]$

Sample size calculated = 38. So, a sample size of 40 was decided to be taken. All patients undergoing hemodialysis and who satisfied the Inclusion and Exclusion criteria formed the sample of the study.

#### *Inclusion Criteria*

- (1) Admitted for haemodialysis in the dialysis unit.
- (2) Above 18 years and below 65 years of age.
- (3) Undergoing dialysis more than 3 months.
- (4) Who gave written informed consent.

#### *Exclusion Criteria*

1. Patients who refused to give written informed consent.
2. Critically ill.
3. Unconscious / Altered consciousness patients.
4. Uncooperative patients.

#### *Procedure*

The institutional ethical committee clearance was obtained. The design and nature of the clinical study was explained to the patients. Informed consent was obtained from each patient who were included in the study, after satisfying inclusion and exclusion criteria. All the patients visiting the Dialysis centre in the hospital during the period from 1<sup>st</sup> Jan 2014 to 31<sup>st</sup> July 2015 formed the sample of this study.

We used a specially prepared proforma which included present history, past history, family history, personal history, demographic details, illnesses details and dialysis details. Later on general physical examination and mental status examination was conducted and relevant findings were noted. Then each patient was given MINI scale by the investigator to know type of illnesses if any. Further each patient was examined independently by experienced consultant psychiatrist to know psychiatric

diagnosis clinically. All the findings were tabulated and results were obtained. Results of psychiatric diagnoses on MINI scale examination were tabulated. Statistical evaluation was done using Chi-square test and Fisher's exact test wherever appropriate.

#### *Tools for Assessment*

**M.I.N.I PLUS** (Mini International Neuropsychiatric Interview Scale): The Mini-international neuropsychiatric interview is a short structured clinical interview used by many researchers for diagnosing psychiatric disorders according to DSM-IV. It was designed for epidemiological studies and multicenter clinical trials. It is very convenient for administering and it requires less time than any other diagnostic interviews like the Schedules for Clinical Assessment in Neuropsychiatry (SCAN), the Composite International Diagnostic Interview (CIDI) or the Structured Clinical Interview for DSM-IV disorders (SCID). MINI being a relatively brief scale, is divided into different modules which correspond to diagnostic categories such as major depressive episode, dysthymia, suicide, mania/hypomania, panic disorders, social phobia, post traumatic stress disorder, alcohol and other psychoactive substance, psychotic disorders, anorexia nervosa, somatisation disorder, hypochondriasis and other anxiety spectrum disorders including obsessive compulsive disorder. It does not assess the personality disorders. One can finish administering this instrument in just 15 minutes [16].

#### *Statistical Analysis*

Data was collected and tabulated using Microsoft excel. Frequency and percentages calculated for all quantitative measures. Mean and standard deviation were calculated for qualitative measures. Chi-square test and Fisher's exact test were used to analyse categorical values and check the association between two variables. P value of <0.05 is considered as statistically significant. SPSS 11 was used to process the data.

#### **Results**

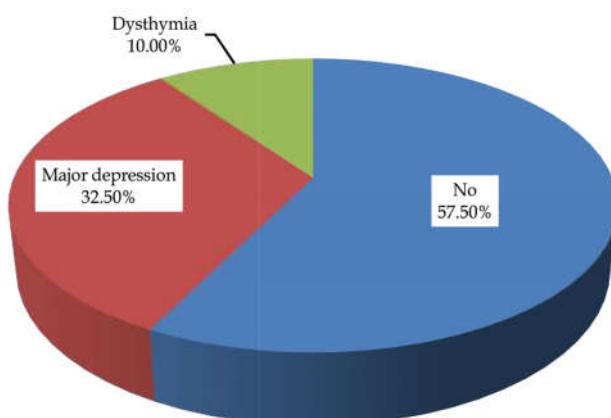
Total of the participants were 40 and males were 28 and females were 12 (Table 1). A large majority i.e. 35 (87.50%) patients belonged to married group. Out of this only 14 (35.00%) had psychiatric morbidity. As shown in Table 2 and Figure 1, in the present study, 17 (42.50%) cases had some type of psychiatric

morbidity. Only 23 (57.50%) cases were free of any psychiatric problems as shown in Table 1. Major Depressive episode (32.50%) was the major morbidity followed by Dysthymia (10.00%). These results also correlated with the clinical psychiatric diagnosis as per the ICD-10. Even though 17 (42.50%) of cases did suffer from psychiatric illness, all were having depression related disorder and other type of psychiatric disorders were absent. Socio-economic status of the cases was assessed by applying

Modified B.G. Prasad 2014 classification. Table number 2 reveals that majority of the cases belonged to social class-II numbering 12 (30.00%) and social class-I numbering 11 (27.50%). Thus totally 23 (57.50%) cases were from higher income group. ESRD treatment of continuous long term dialysis being quite costly was perhaps not affordable to lower income group of social class-IV and social class-V. Hence only 10 (25.00%) cases were from these groups.

**Table 1:** Sociodemographic details

Variables		(N-40)
		Numbers (%)
Gender	Male	28(70%)
	Female	12(30%)
Age(in years)	<=25	01(2.5%)
	26-35	12(30%)
	36-45	5(12.5%)
	46-55	10(25%)
	56-65	12(30%)
Education Status	No Education	07(17.5%)
	Primary Education	05(12.5%)
	Secondary Education	13(32.5%)
	College Education	08(20%)
	Graduation and above	07(17.5%)
Family History of Psychiatric Illness	Present	1(2.5%)
	Absent	39(97.5%)
Family History of Major medical illness	Present	2(5%)
	Absent	38(95%)
Socioeconomic status	I	11(27.5%)
	II	12(30%)
	III	07(17.5%)
	IV	09(22.5%)
	V	01(2.5%)
Duration of Chronic Kidney Disease	<=60 (months)	38(95%)
	61-120 (months)	1(2.5%)
	241+ (months)	1(2.5%)
Marital Status	Single	2(5%)
	Married	35(87.5%)
	Divorced	1(2.5%)
	Widow	2(5%)
Past History of Diabetes Mellitus	Present	13(33%)
	Absent	27(67%)
Past History of Hypertension	Present	25(63%)
	Absent	15(37%)



**Fig. 1:** Distribution of study participants according to Psychiatric morbidity

**Table 2:** Distribution of study participants according to Psychiatric morbidity

Psychiatric Morbidity	Psychiatric Morbidity		Percentage
	Number		
Major Depressive episode	13		32.50%
Dysthymia	4		10.00%
Nil	23		57.50%
Total	40		100.00%

**Table 3:** Distribution of psychiatric morbidity in study participants according different variables

Variables		Psychiatric Morbidity			Chi Square Value	P-Value
		Major Depression	Dysthymia	None		
Gender	Male	9(32.14%)	4(14.28%)	15(53.58%)	$\chi^2=1.968$	$p=0.374$
	Female	4(33.33%)	0(0.00%)	8(66.67%)		
Age	<=25	0(0.00%)	0(0.00%)	1(100.00%)	$\chi^2=8.048$	$p=0.429$
	26-35	4(33.34%)	3(25.00%)	5(41.66%)		
Socioeconomic status	36-45	2(40.00%)	1(20.00%)	2(40.00%)	$\chi^2=3.208$	$p=0.921$
	46-55	4(40.00%)	0(0.00%)	6(60.00%)		
Educational Status	56-65	3(25.00%)	0(0.00%)	9(75.00%)	$\chi^2=8.327$	$p=0.402$
	I	3(27.22%)	1(9.09%)	7(63.69%)		
Duration of Kidney Disease in Months	II	4(33.34%)	2(16.66%)	6(50.00%)	$\chi^2=1.556$	$p=0.817$
	III	2(28.58%)	0(0.00%)	5(71.42%)		
Marital Status	IV	4(44.40%)	1(11.20%)	4(44.40%)	$\chi^2=6.476$	$p=0.372$
	V	0(0.00%)	0(0.00%)	1(100.00%)		
Educational Status	No	2(28.58%)	0(0.00%)	5(71.42%)	$\chi^2=8.327$	$p=0.402$
	Primary	0(0.00%)	1(20.00%)	4(80.00%)		
Duration of Kidney Disease in Months	Secondary	7(53.84%)	1(7.69%)	5(38.47%)	$\chi^2=1.556$	$p=0.817$
	College	1(12.50%)	1(12.50%)	6(75.00%)		
Marital Status	Graduation and above	3(42.86%)	1(14.28%)	3(42.86%)	$\chi^2=6.476$	$p=0.372$
	<=60	13(34.22%)	4(10.52%)	21(55.26%)		
Duration of Kidney Disease in Months	61-120	0(0.00%)	0(0.00%)	1(100.00%)	$\chi^2=1.556$	$p=0.817$
	241+	0(0.00%)	0(0.00%)	1(100.00%)		
Marital Status	Single	0(0.00%)	1(50.00%)	1(50.00%)	$\chi^2=6.476$	$p=0.372$
	Married	11(31.42%)	3(8.58%)	21(60.00%)		
Duration of Kidney Disease in Months	Divorced	1(100.00%)	0(0.00%)	0(0.00%)	$\chi^2=6.476$	$p=0.372$
	Widow	1(50.00%)	0(0.00%)	1(50.00%)		

## Discussion

Prevalence of depression in ESRD cases has varied widely according to different studies in the world. Prevalence of Depression according to various studies is as follows Watnik S -30%, O Amira-23.7%, Aghanwa-25%, and it is reported much higher according to studies by Zeb Saeed-70% and by Pramiladevi- 72.7% [17-21]. If one takes dysthymia as one of the chronic low intensity depressive disorder then present study has got prevalence of depressive disorders to be 42.50% (Major Depressive episode 32.50% and dysthymia 10.00%). There was no statistical significance. This is broadly similar to the finding of M Rai et al. of 47.8% [15]. This was an Indian study from New Delhi. The prevalence of depression in CKD patient has varied widely in different studies and different populations, using different assessment tools as mentioned by Kimmel

PL and Peterson RA in the year 2006 [11]. These cases of ESRD are basically from Nephrology department. Hence few studies might have been conducted by Nephrologists or Physicians and not by Psychiatrists. This could be another reason for wide variation in the prevalence of depressive disorders in ESRD patients. Surprisingly, there was no other type of psychiatric illness in this sample. Even though, anxiety disorders were expected in these cases because ESRD cases are chronic and there are many stress factors like "dependency feeling" on dialysis and its nature of artificial life support, constant needs of physical and psychological support by relatives and economical costs involved in each session must be putting tremendous amount of stress on these patients which could lead to anxiety and related disorders. However, this study did not find a single anxiety disorder case in the sample. An Indian study by S.Kohli reported 87% of patients having anxiety state [22]. Another study by Carmen M

Perales Montilla et al. reported that anxiety state was seen in 24.9% of patients [23]. In this present study, anxiety symptoms or high suicidal ideas or history of suicidal attempt and gross OCD symptoms were not found in any of the subjects. However, study by Macaron G et al. reported 37% of cases having suicide ideas [24] and as per the study by Epameinodas Lyros et al., obsessive-compulsive symptoms were important aspect of their patients [25]. Absence of any other psychiatric illness except depressive disorder in this sample could be because of less number of sample size in this study.

Majority of these cases were between the ages of 26 yrs to 55 years. But, youngest age group i.e.  $\leq 25$  yrs was only one patient which is expected in chronic kidney disease (Table 1). Except this age group all other age groups had almost equal distribution of major depression. It may be that the youngest age group patient is less mature to understand the gravity of CKD and after the age of 55 years patients learn to accept the reality of CKD and had less of depression as reaction to their major medical problem of ESRD. There was no statistical significance (Table 3). ESRD is a chronic disease. It certainly has poorer prognosis. Patients require continuous dialysis and they are on artificial life support. Patients require constant medical attention, caregivers' cooperation and support. Above all it is costly treatment over long period of time. Thus ESRD is also one of those chronic medical conditions where prognosis is poor like myocardial infarction, various types of cancer, especially of oropharynx and pancreatic, Parkinson's disease, HIV positive individuals, diabetes mellitus, hypothyroidism and ESRD as mentioned in textbook of medicine. Between 20% to 30% such patients do have depression [26]. Present study found major depression in many of the cases which is almost similar to other chronic medical disorders.

The gender association on the type of psychiatric morbidity affected was not statistically significant as  $\chi^2(2)=1.968$ ,  $p=0.374$ . In other studies done by Muhammed Anees and Pramiladevi also reported more number of males as compared to females in their samples [21,27]. In India usually males are given more preference in giving continuous treatment of any illness. This is more true of socially backward areas like Bagalkot district where this study was conducted. In addition, chronic renal disease may be more common in males. These factors may explain the preponderance of male patients in this sample.

We did not find any statistically significant association between the educational status, socioeconomic status, marital status, duration of illness of the patients suffering from chronic kidney

disease and the psychiatric morbidity (Table 3). However, one can safely say that psychiatric morbidity in CKD must be starting early in the period of ESRD. Future studies with more details of subdivision of duration of CKD and presence of psychiatric morbidity would throw light on this aspect.

Marital status perhaps protects from the psychiatric morbidity if the married partner gives a lot of support to the patient. It could also be argued that having married increase stressors in terms of marital responsibilities which may add to psychiatric problem. Therefore, a definite comment cannot be made from this study specially when the finding are not statistically significant. In another study by Anees M et al. there were also large majority of patients numbering 77 (86.50%) who were married which is similar to present study's figure of 35 (87.50%) [27].

One study by Jha V et.al, states that diabetic nephropathy was commonest cause of CKD in India [7]. Another study by Gupta R et.al, argued that CKD is emerging to be important chronic disease globally because of rapidly increasing worldwide incidence of Diabetes and Hypertension [8]. Both these conditions are known to increase psychiatric morbidity and are also related to CKD. However, definite comments cannot be made because there is no statistical significance in this study regarding these factors. Infections as a Post Haemodialysis complications were present in 22 (55.00%) of the cases studied. Out of these 11 (27.50%) had Psychiatric morbidity and 11 (27.50%) had Nil Psychiatric morbidity. Other complications were absent. The results were not statistically significant with  $\chi^2(3)=1.125$ ,  $p=0.29$ . Past history or family history of either major medical illness or any psychiatric illness did not have any association with psychiatric morbidity. Still, 5 out of 13 cases of diabetes i.e. 38.50% of diabetics with ESRD had psychiatric morbidity of major depression. This little higher figure in this study could be because these patients were having both ESRD and diabetes leading to increased psychiatric morbidity.

#### Limitations

The cross-sectional psychiatric assessment of the patients limits explanation of causal relation between psychiatric disease and end-stage renal disease. This is hospital based study having small number of cases as the study was limited to the patients attending the dialysis unit of S.N Medical College and H S K Hospital and Research center, Bagalkot. Results of this hospital based study cannot be generalized to entire population.

## Conclusion

ESRD is commonly said as the final stage of the chronic kidney disease. It is not an uncommon scenario where in a psychiatrist opinion is sought for these patients especially on haemodialysis. Patients are usually questioned about the restrictions in the intake of fluids and meals, about symptoms like itching, decreased energy, psychosocial stressors like as loss of self-esteem, worthlessness, hopelessness, guilty feelings towards members in the family and any issues in the society. Present study was planned with the objectives to know the psychiatric disorders in patients suffering from end stage renal disease. Present study is on small number of subjects. There was no statistical significance in any association studied. Hence definite conclusions cannot be drawn. However, majority (42.5%) of ESRD patients do have some psychiatric morbidity, commonest being major depression found in 32.50% of cases. However, prospective studies and population based studies on this topic would be highly useful. Though there was no statistical significance this study suggested that there is a trend of Psychiatric morbidity to be associated with higher education and higher social class along with presence of diabetes/hypertension in these cases. It indicates a need for more studies in multiple centres on large number of patients, findings of which may guide clinicians to treat patients more effectively. A multidisciplinary approach may be necessary in this regard. It will definitely improve prognosis of these unfortunate patients.

### Abbreviations

ESRD-End stage renal disease, HD-Haemodialysis, SPSS-Statistical Package for Social Studies, CKD-Chronic kidney disease.

### Acknowledgement

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### References

- Bargman MJ, Skorecki K. Chronic Kidney Disease. In: Kasper DL, Hauser DL, Jameson JL, Fauci AS, Longo DL, Loscalzo J. Harrison's Principles of Internal Medicine. 19<sup>th</sup> ed. Vol 2. New York: McGraw Hill; 2015.p.1811.

2. Longo DL, Kasper DL, Jameson JL, Fauci AS, Hauser SL, Loscalzo J, editors. Harrison's principles of internal medicine. 18<sup>th</sup> ed. Vol 2. New York: McGraw Hill; 2012.p.2308.
3. Shah SN, Anand MP, Acharya VN, Bichile SK, Karnad DR, Kamath SA, et al, editors. API Textbook of MEDICINE. 7th ed. Mumbai: The Associations of Physicians of India; 2003.p.69.
4. Warrell DA, Cox TM, Firth JD. Oxford textbook of medicine. 5th ed. Vol 3. New York: Oxford University Press; 2010.p.3932.
5. Kaptein AA, Dijk SV, Broadbent E, Falzon L, Thong M, Dekker FW. Behavioural research in patients with end-stage renal disease: A review and research agenda. *Patient Educ Couns* 2010 Oct; 81(1):23-9.
6. Levy NB. What is psychonephrology? *J Nephrol* 2008; 21(1)Supp13:s51-3.
7. Jha V. Current status of end-stage renal disease care in India and Pakistan. *Kidney Int Suppl* 2013;3: 157-60.
8. Gupta R. Trends in hypertension epidemiology in India. *J Hum Hypertens* 2004; 18(2):73-8.
9. Rajapurkar M, Dabhi M. Burden of disease-Prevalence and incidence of renal disease in India. *Clin Nephrol* 2010; 74(1):9-12.
10. Modi GK, Jha V. The incidence of end-stage renal disease in India. *Kidney Int* 2006; 70:2131-3.
11. Kimmel PL, Peterson RA. Depression in patients with end-stage renal disease treated with dialysis: Has the time to treat arrived ? *Clin J Am Soc Nephrol* 2006; 1:349-52.
12. Son YJ, Choi KS, Park YR, Bae JS, Lee JB. Depression, symptoms and the quality of life in patients on Hemodialysis for End Stage Renal Disease. *Am J Nephrol* 2009; 29:36-42.
13. O'Donnell K, Chung JY. The diagnosis of major depression in end-stage renal disease. *Psychother Psychosom* 1997; 66:38-43.
14. Williams SW, Tell GS, Zheng B, Shumaker S, Rocco MV, Sevick MA. Correlates of sleep behaviour among hemodialysis patients, The Kidneys Outcomes Prediction and Evaluation (KOPE) Study. *Am J Nephrol* 2002; 22:18-28.
15. Rai M, Rustagi T, Rustagi S, Kohli R. Depression, insomnia and sleep apnea in patients on maintenance hemodialysis. *Indian J Nephrol*. 2011; 21(4):223-9.
16. Sheehan DV, LeCrubier Y, Sheehan KH, Amorim P, Janavas J, Weiller E, et al. The Mini-International Neuropsychiatric Interview (M.I.N.I): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and

ICD-10. J Clin psychiatry 1998; 59(20):22-33.

17. Watnick S, Kirwin P, Mahnensmith R, Concato J. The prevalence and treatment of depression among patients starting dialysis. Am J Kidney Dis 2003; 41:105-10.
18. O Amira. Prevalence of symptoms of depression among patients with chronic kidney disease. Niger J Clin Pract 2011; 14(4):460-3.
19. Aghanwa HS, Morakinyo O. Psychiatry complications of haemodialysis at a kidney centre in Nigeria. J psychosom Res 1997; 42:445-51.
20. Saeed Z et al. Depression in patients on Haemodialysis and their caregivers. Saudi J Kidney Dis Transpl 2012; 23(5):946-52.
21. Pramiladevi R., Goornavar SM, Kora S. Depression in patients on Haemodialysis in Bagalkot. Medica Innovatica 2012; 1(2):5-11.
22. S.Kohli, P Batra, HK Agarwal. Anxiety, locus of control and Coping Strategies among end-stage Renal disease patients Undergoing Maintenance Haemodialysis. Indian J Nephrol 2011; 21(3): 177-81.
23. Montilla CM, Duscheck S, Reyesdelpaso GA, Reyes-Step G. Influence of emotional factors on the report somatic symptoms in patients on chronic haemodialysis: Relevance of anxiety. Nefrologia 2013; 33:816-25.
24. Macaron G, Fahed M, Matar D, Kazour F, Richa S, Bou-Khalil K et al. Anxiety, Depression and Suicidal Ideation in Lebanese patients Undergoing Haemodialysis. Community Ment Health J 2014 Feb; 50:235-8.
25. Lyros E, Messinis L, Dendias G, Siavelis C, Aggeliki T, Papathanasopoulos P. Increased Self-Report of Obsessive-Compulsive Behaviours Among Hemodialysis Patients. A Case -Control Study. Prim Care Companion J Clin Psychiatry 2010; 12(3).
26. Reus VI. Mental disorders. In: Longo DL, Kasper DL, Fauci AS editors. Harrison's principles of internal medicine. 18<sup>th</sup>ed. Vol 2. New York: McGraw Hill; 2012.p.3536.
27. Anees M, Barki H, Masood M, Ibrahim M, Mumtaz A. Depression in Haemodialysis Patients. Pak J Med Sci 2008; 24:560-5.

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## Gender Differences in Locus of Control and Sensation Seeking among Late Adolescents: A College Based Cross-Sectional Study

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### Abstract

The Sensation seeking behavior is very common among adolescents as they most often desire for varied, novel, complex and intense sensations and experience, and are willing to take physical, social, legal and financial risks for the sake of such experience. The locus of control deals with the perception or belief about the degree of control people have over the events occurring in their lives. The objective of this research study is to identify the locus of control and sensation seeking behaviour among late adolescents. The sample comprised of 100 students, 50 girls and 50 boys each, belonging to the age group of 16 -18 years from urban locality. The tools used to assess locus of control and sensation seeking were Leven son's Locus of Control scale and Zukerman's Sensation Seeking Scale respectively. The statistical analysis was done using Independent Sample t-test. The Results indicated that there is no significant gender difference in the sensation seeking behavior among late adolescents but there were significant gender differences with findings that boys were found to have external locus of control.

**Keywords:** Locus of Control; Sensation Seeking; Late Adolescents.

### Introduction

It was the social learning theorist Julian Rotter who developed the concept of locus of control in 1966. During this time, the dominant perspective in clinical psychology was Freud's Psychoanalysis, which focused on people's deep-seated instinctual motives of childhood as determining activities. Rotter however, believed that reinforcement helped to modify the behavior [1]. He exposed through reinforcements individuals got to know about the causes of their actions, and these beliefs then lead them what type of attitudes and actions they accept in the future [2].

Locus of control is an incorporated in both the Expectancy-Value Theory (1970) which was developed by Martin Fishbein, and the Social Learning Theory (1954), which was developed by

Rotter. Both theories claim that reinforcements act to make stronger the expectancy that an exact actions or events will be followed by that same reinforcement in the future [1]. On the other hand, once a relationship is established between a behavior and reinforcement, the absence of the reinforcement will reduce or extinguish the expectancy. Expectancies from exact situations to situations were generalized that are perceived as similar or related. These general beliefs and expectancies can influence a variety of behavioral choices in many dissimilar life situations [10].

"Sensation seeking" is defined as "the need for varied, novel and complex sensations and experiences and willingness to take physical and social risks for the sake of such experiences". Findings of new studies on hazardous behaviors have shown a consistent link between high sensation-seeking behavior and engaging in risk-taking actions during

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adolescence [11-12]. An rising number of adolescents and young people are fascinated by intense stimuli and strong sensations [13]. Many youngsters have proved that they have developed a sort of selfishness to get gratification in everyday life events. The threshold of gratification becomes increasingly higher; the low capacity for pleasure makes many young people apathetic, bored or incapable to be able to defer the achievement of the desires.

Sensation-seekers are characterized by low sensitivity to stimuli and therefore in need of high levels of stimulation to maintain an optimal state of arousal. To the extent that individuals need sensations, they engage in behavior that increases the amount of stimulation they experience, and they are likely to take risks to that end. Data have indeed highlighted that high sensation-seekers report significant levels of violent behavior, abuse of illegal substance, alcohol use and risky sexual behaviors [14-16].

In recent years, sensation seeking is considered as a personality trait, and it has led to the series of studies on the physiological and temperamental characteristics of sensation seekers. In this sense, this personality trait has been described by Zuckerman in his work on bio-psychological personality research and is often related to biochemical reactions in the brain. Within this conceptual framework, recent studies have underlined that changes in sensation seeking during adolescent development have been hypothesized to be due to maturational changes in the adolescent brain [17-19]. A survey of public and private college students (aged 16-19 years) reveals that there is significant relation between personality and sensation seeking. In addition, risk-taking is not found to be correlated to personality and sensation seeking. Taking all these into consideration, this study was planned.

## Methodology

### Aim

- To assess the gender differences in the locus of control and sensation seeking behavior among adolescent population

### Hypothesis

1. There is no significant difference between boys and girls in locus of control.
2. There is no significant difference between boys and girls in their level of sensation seeking.

### Methods

It is a cross-sectional study conducted at a tertiary care teaching medical college and hospital in our city. It was carried out during October 2016. The study involved the distribution of study questionnaire to the participants which included the adolescents studying in 2<sup>nd</sup> year Pre-University Course in the Basaveshwar Science College, Bagalkot.

### Participants

The participants for the present study were selected using purposive sampling method from the urban area in Bagalkot. Selection criteria for participants required that they should fulfill the inclusion and exclusion criteria.

### Inclusion Criteria

1. Age between 15-18 years.
2. Those who give written informed consent.

### Exclusion Criteria

1. Not giving informed consent.

### Instruments

1. *Socio-demographic data sheet*: It consisted of the subject's details like name, age, gender, year of education, place.
2. *Levenson's Scale for Locus of Control*: Locus of control scale was developed by Levenson. There are 24 statements pertain to general life outcomes. Each statement has five point scale (Strongly Agree - Strongly Disagree) [21].
3. *Sensation Seeking Scale*: Sensation Seeking Scale was developed by Zukerman. Here in this study, Zukerman's Sensation Seeking Scale form V is used. The scale consists of 40 items, which is made up of the four subscales (disinhibition, Boredom Susceptibility, Thrill and Adventure Seeking, Experience Seeking) of a maximum of 10 points for each of the four subscales and it takes a participant 12 to 25 minutes to complete. Each question has two responses and participants should mark the response relevant to their opinion [22].

### Ethical Considerations

Permission was obtained from the institutional ethical committee where the study was conducted.

Participants were informed of the study's aims and procedures so that they could decide if they were willing to participate. The investigator explained explicitly that students' responses would have no influence on their further course of the study. Written informed consent was taken from all study subjects, before enrolment in the study. After taking the consent, the principal investigator gave them the questionnaires. Data collection tools contained no identifying information and thus kept individual responses confidential.

#### Procedure

The participants were personally met and one of the authors verbally explained the aims and method of the research and how to complete the questionnaires. After taking the consent to participate in the study, socio-demographic data sheet was distributed. Later, both the Levenson's Scale for Locus of Control and Sensation Seeking Scale were distributed to the subjects individually at various times. The subjects who were willing to participate in the study completed both questionnaires. All the instructions were given in simple language. After completion of the assessment the tools were collected back and the participants were thanked for their

participation. The scoring of each response sheet was done as mentioned in the respective manual.

#### Statistical Analysis

The data were tabulated in Microsoft excel and analyzed using SPSS software version 13. Results were presented in narratives and tables. The obtained results were analyzed using descriptive statistic and independent sample t-test. Statistical significance was assumed at  $p < 0.05$ .

#### Results

The questionnaires were given to 110 students but six girls and four boys did not give consent. Hence, to fulfill the sample size, 100 students were included. Sample included fifty girls and fifty boys. All were from 12<sup>th</sup> standard (PUC-II) as per our study criteria and were belonging to urban domicile. Mean age of the students was 16.8 years (SD-1.25). Table 1 explains the mean, standard deviation (SD) and the minimum and maximum values on the individual parameters on both the scales used.

**Table 1:** Descriptive Statistics (N-100)

Variables	Mean	SD	Minimum Score	Maximum Score
Disinhibition	4.99	1.474	1	8
Thrill & Adventure	6.33	1.741	2	10
Boredom Susceptibility	3.85	1.459	1	8
Experience Seeking	4.65	1.395	2	8
Powerful to Others	27.07	4.632	13	36
Chance control	26.28	4.557	16	37
Internal control	29.66	5.578	14	41

**Table 2:** Gender differences in Locus of control

Locus of Control	Gender	N	Mean	SD	t - value	Sig.( p-value)
Powerful Others	Girls	50	25.78	4.409	-2.886	0.005
	Boys	50	28.36	4.530		
Chance Control	Girls	50	25.60	4.634	-1.502	0.136
	Boys	50	26.96	4.421		
Internal Control	Girls	50	29.64	6.197	-0.036	0.972
	Boys	50	29.68	4.946		

As in Table 2, it shows that the girls and boys do not differ significantly on chance control and internal in locus of control scale as the p-value for chance control and internal control are statistically insignificant ( $p\text{-value} > 0.05$ ). We retained the null hypothesis. But as seen in powerful others in locus

of control as the t-value of -2.886, which is statistically significant with  $p\text{-value} < 0.05$ . Hence the null hypothesis is rejected and alternative hypothesis is accepted that there is a significant gender difference (boys > girls) with respect to powerful others in locus of control scale.

**Table 3:** Gender differences in Sensation Seeking

Sensation Seeking	Gender	N	Mean	SD	t - value	Sig.(p-value)
Disinhibition	Girls	50	4.82	1.561	1.156	0.251
	Boys	50	5.16	1.376		
Thrill & Adventure	Girls	50	6.14	1.412	1.092	0.277
	Boys	50	6.82	1.483		
Boredom Susceptibility	Girls	50	4.08	1.738	-1.588	0.115
	Boys	50	3.62	1.741		
Experience Seeking	Girls	50	4.48	1.403	1.221	0.225
	Boys	50	4.82	1.380		

Our hypothesis was stating that there is no significant difference between boys and girls in their level of sensation seeking. Now, Table 3 depicts that t-values for disinhibition, thrill and adventure, boredom susceptibility and experience seeking are 1.156, 1.092, -1.588 and 1.221 respectively and p-value is not significant for any of them. This shows that there are no significant gender differences with respect to disinhibition, thrill and adventure, boredom susceptibility and experience seeking in sensation seeking scale. Hence the null hypothesis is accepted.

## Discussion

Adolescence is a period of transition between childhood and adulthood that involves biological, cognitive and socio-emotional changes. Social Scientists who study adolescence usually divided the adolescence into three stages such as early adolescence, which covers period from about age 14 through 13; middle adolescence from about age 14 through age 18; and late adolescence from about 19 through age 22. A key task of the adolescence is preparation for the adulthood.

Locus of Control refers to individual's very common and cross-sectional belief about what it determines whether or not, the yare reinforced in lifetime. Individual can be classified from internal to external personality trait [1,2]. Previous studies have suggested that affective execution is related to the locus of control. Many studies have revealed that the peripheral expectancies for locus of control are positively associated to depressive tendencies. results show that an external locus of control appear to be positively related to a feeling of powerlessness, alienation from self and work, and a tendency to avoid challenge. Locus of control also seems evidently associated to physical and psychological health problems. People who had more internal locus of

control indicated less mental and physical health problems and expressed less stress than those who had a more external locus of control [3-8].

Previous studies reported of significant gender differences with respect to powerful others with evidence bearing on the division of control, in both the sexes ( $M=16.65$ ,  $t=12.41$ ,  $p<0.001$ ), in the chance control ( $M=13.94$ ,  $t=13.28$ ,  $p<0.001$ ), and on individual control on the scale ( $M = 14.64$ ;  $F = 4.86$ ,  $p< 0.05$ ) [23]. These above results are partially contrast to our study stating there is no significant difference between males and females with respect to two areas on the locus of control scale but our study also say there is gender difference in powerful others. In addition to that comparison with earlier studies by using locus of control scale on psychiatric patients, neurotic males had higher internal scale scores than neurotic females. Paranoid males scored higher on the powerful others scale than paranoid females [24].

As per the study done by Levenson [25], analyses of scores on the internal and chance scales indicate that neither the effect of activism nor that of the activism plus ideology interaction is significant. However, the effect of ideology approaches significance. Compared with the liberals, the conservatives tended to score higher on the internal scale and lower on the chance scale. The factorial analysis of scores on powerful others scale show there are no main effects, but the predicted interaction is significant with  $p < 0.05$  [25].

The theory of individual differences in responses to the experimental situation of sensory deprivation, and the consistent work of early 1960's is based on the idea of Zuckerman's sensation seeking which showed that individual differences in optimal level of stimulation and arousal and the difference was measured using Zuckerman questionnaire, which developed to examine the traits of sensation seeking by using sensation seeking scale (SSS). The four sub factors in this scale were thrill and adventure seeking

: aspiration to engage in games or activities involving in physical danger or risk, experience seeking: aspiration to look for new experiences through the mind and senses by living in a out of the ordinary life style, boredom susceptibility: an hatred towards repetitive experience of any kind, everyday work, or even dull or predictable individuals etc, disinhibition: wish to disinhibit and individual's behavior in the society by drinking, partying and seeking variety in sexual partners etc [20].

As per previous studies regarding the SSS scale, in order to determine how each sample group differed in the Sensation-Seeking Scale V (SSS-V), it is theorized that the students should measure higher regarding sensation-seeking given their interest in high-risk recreation. Based on the results, there is a significant difference between the two sample groups on each of the sub-scales of the SSS-V [26].

But, in the present study, we did not find any significant gender differences with respect to disinhibition, thrill and adventure, boredom susceptibility and experience seeking. Studies conducted in the Europe, USA, China and Australia have all reported higher average scores in males than females on three of the four subscales on sensation-seeking scale, namely, Disinhibition (Dis; favorable attitudes to uninhibited social interactions), Thrill and Adventure Seeking (TAS; interest in physically challenging activities) and Boredom Susceptibility (BS; dislike for repetition and predictability), but not difference was found on Experience Seeking (ES; interest in low-risk, novel experiences) [28,29]. Males also have higher average scores than females on few measures of risk-taking that could lead to damaging or undesirable outcomes [30].

A meta-analysis by Cross et.al., investigated whether gender differences in sensation-seeking have changed over the past years. They found that gender differences in total SSS-V scores have remainconstant across years, as have gender differences in Boredom Susceptibility and disinhibition.

Whereas the gender difference in Thrill and Adventure Seeking has declined attributing this to outdated questions on this sub-scale or changes in social norms. Their study results supported the view that male and female differ in their propensity to report sensation-seeking characteristics, while behavioral manifestations of sensation seeking change over a period. Gender differences in sensation-seeking could imitatehereditarily predisposed interacting with socially transmitted information [31].

## Conclusion

The present study aimed to determine whether male adolescents involve in a considerably level of high-risk recreational activities, would differ with regards to sensation-seeking. Our study found significant result. As per our study hypothesis, the findings of this study state that there are no such gender differences in chance control and internal control but in powerful others on locus of control scale, there is significant gender differences with boys showing more of external locus of control. Most of the findings in the literature are in favor of this finding. In modern society, men and women are still viewed as different but equal. Consequently, the corroboration for gender differences in LOC may no longer be pertinent, so that gender differences in LOC might no longer become visible in a modern people or may now be moderated by an unnoticed variable [27]. The results of the study show that the boys are more external and influenced by others than girls in powerful to others compare to chance control and internal control. However, it was highly limited in its ability to accurately represent, or be representative of, the general population. This warrants studies on larger sample size.

## Acknowledgements

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*Conflict of Interest:* None declared

*Source of Support:* Self

## Abbreviations

PUC:Pre-University College, SSS-V: Sensation-Seeking Scale V, LOC-Locus of Control.

## References

1. Mearns J. The social learning theory of Julian B. Rotter. url: <http://psych.fullerton.edu/jmearns/rotter.htm>. 2004.
2. Rotter JB. Generalized expectancies for internal versus external control of reinforcement. Psychological monographs: General and applied. 1966; 80(1):1.

3. Benassi VA, Sweeney PD, Dufour CL. Is there a relation between locus of control orientation and depression? *Journal of abnormal psychology* 1988; 97:357-67.
4. Brown JD. Staying fit and staying well: Physical fitness as a moderator of life stress. *Journal of Personality and Social Psychology*. 1991 Apr; 60(4):555-61.
5. Burger JM. Desire for control, locus of control, and proneness to depression. *Journal of personality*. 1984 Mar 1; 52(1):71-89.
6. Ganellen RJ, Blaney PH. Stress, externality, and depression. *Journal of Personality*. 1984 Dec 1; 52(4):326-37.
7. Kirkcaldy BD, Cooper CL, Furnham A, Brown JI. Personality, job satisfaction and well-being among public sector (police) managers. *European Review of Applied Psychology/Revue Européenne de Psychologie Appliquée*. 1993; 43:241-8.
8. Kobasa SC, Maddi SR, Kahn S. Hardiness and health: a prospective study. *Journal of personality and social psychology*. 1982 Jan; 42(1):168-77.
9. Martin G, Richardson AS, Bergen HA, Roeger L, Allison S. Perceived academic performance, self-esteem and locus of control as indicators of need for assessment of adolescent suicide risk: implications for teachers. *Journal of adolescence*. 2005 Feb 28; 28(1):75-87.
10. Rotter JB. Generalized expectancies for internal versus external control of reinforcement. *Psychological monographs: General and applied*. 1966; 80(1):185-90.
11. Zuckerman, M. *Sensation seeking and risky behavior*. American Psychological Association, Washington 2007.
12. Zuckerman M. Behavioral expressions and biosocial bases of sensation seeking. Cambridge university press; 1994 June 24.
13. Eurispes. *Rapporto Nazionale sulla Condizione dell'Infanzia e dell'Adolescenza* (National report on infancy and adolescence). XIII ed. Rome 2012.
14. Lynne-Landsman SD, Gruber JA, Nichols TR, Botvin GJ. Is sensation seeking a stable trait or does it change over time? *Journal of youth and adolescence*. 2011 Jan 1; 40(1):48-58.
15. Pace U, Schimmenti A, Zappulla C, Di Maggio R. Psychological variables characterizing different types of adolescent gamblers: A discriminant function analysis. *Clinical Neuropsychiatry*. 2013 Dec 1; 10(6):253-60.
16. Baiocco R, Laghi F, D'Alessio M. Decision-making style among adolescents: Relationship with sensation seeking and locus of control. *Journal of Adolescence*. 2009 Aug 31; 32(4):963-76.
17. Sijtsema JJ, Veenstra R, Lindenberg S, van Roon AM, Verhulst FC, Ormel J, Riese H. Mediation of sensation seeking and behavioral inhibition on the relationship between heart rate and antisocial behavior: The TRAILS study. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2010 May 31; 49(5):493-502.
18. Steinberg L. A social neuroscience perspective on adolescent risk-taking. *Developmental review*. 2008 Mar 31; 28(1):78-106.
19. Somerville LH, Jones RM, Casey BJ. A time of change: behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. *Brain and cognition*. 2010 Feb 28; 72(1):124-33.
20. Zuckerman M, Eysenck SB, Eysenck HJ. Sensation seeking in England and America: Cross-cultural, age, and sex comparisons. *Journal of consulting and clinical psychology*. 1978 Feb; 46(1):139-49.
21. Levenson, H. Differentiating among internality, powerful others, and chance. In H. M. Lefcourt (Ed.), *Research with the locus of control construct*. New York: Academic Press. 1981; 1:15-63
22. Zuckerman M. Item revisions in the sensation seeking scale form V (SSS-V). *Personality and Individual Differences*. 1996 Apr 1; 20(4):515.
23. Levenson H. Activism and powerful others: Distinctions within the concept of internal-external control. *Journal of personality assessment*. 1974 Aug 1; 38(4):377-83.
24. Levenson H. Multidimensional locus of control in psychiatric patients. *Journal of consulting and clinical psychology*. 1973 Dec; 41(3):397.
25. Levenson H, Miller J. Multidimensional locus of control in sociopolitical activists of conservative and liberal ideologies. *Journal of personality and social psychology*. 1976 Feb; 33(2):199.
26. Le Roux HN. *Sensation-seeking, locus of control and self-efficacy correlates of adventure-based trainees: A comparative study* (Doctoral dissertation, Stellenbosch: Stellenbosch University). 2014.
27. Christopher Seemann. *Sex Differences in Locus of Control, Coping, and the Relationship between Locus of Control and Coping*. Boston University 2008.
28. Wang W, Wu YX, Peng ZG, Lu SW, Yu L, Wang GP, Fu XM, Wang YH. *Test of sensation seeking in a Chinese sample*. *Pers Indiv Diff*. 2000; 28(1):169-79.
29. Zuckerman M, Buchsbaum MS, Murphy DL. *Sensation seeking and its biological correlates*. *Psychological Bulletin*. 1980 Jul; 88(1):187-214.
30. Byrnes JP, Miller DC, Schafer WD. *Gender differences in risk taking: A meta-analysis*. *Psych Bull*, 1999; 125:367-83.
31. Cross CP, Cyrenne DL, Brown GR. *Sex differences in sensation-seeking: a meta-analysis*. *Sci Rep*, 2013; 3:2486.

## Behaviour Management in a Workplace: An Overview

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### Abstract

Human behaviour is the response of the individual to various stimuli. Work behaviour is the formal behaviour one uses in the place of work. Behaviour can range from normal or eccentric to abnormal types depending on the situations and circumstances. Employees often come across various types of abnormal work behaviour like counterproductive work behaviour, sexual harassment in the workplace and verbal abuse. These not only produce a decrease in work efficiency but also create a high level of anxiety and both mental as well as physical stress among the employees. They may even feel depressed, resign from the job or commit suicide. Intervention is needed when the behaviour is creating some problem within the individual or to others. Management depends on the individual matters as well as the severity of the abnormal behaviour. All these issues have been discussed in detail including their practical management.

**Keywords:** Human Behaviour; Workplace; Management.

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Behaviour is the range of activities and gestures made by an individual that change its relationship to its environment. It is the response of the individual to various stimuli either from the within or from the external environment or both [1,2]. The nervous system and the endocrine system are believed to be responsible for human behaviour [3].

### Work Behaviour

Work behaviour is the behaviour one exhibits in the place of work or employment and is usually more formal than the behaviour at other places. This generally depends on the job profile and varies from profession to profession. For instance, a photographer would usually have more flexibility in the work behaviour than a doctor. People usually remain more careful about how they behave among their colleagues as many actions made carelessly can

be perceived by others as serious or inappropriate and create displeasure in the work environment [4,5].

#### *Counterproductive Work Behaviour*

Counterproductive work behaviour is a type of work behaviour. These are the acts that employees have against the organisations that do harm or violate the work production. Even individuals do not recognise this behaviour and seem normal to them. Few examples of counterproductive behaviour are passive actions such as not working to meet date line or faking incompetence [6].

#### *Violence by Colleagues and Superiors:*

Sometimes employees are harassed by colleagues and superiors while working. A variety of abusive behaviours is demonstrated against victims to interrupt their work, get their work done and stay in

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their current employment. The interferences that the perpetrators employ are: stocking, harassment and sabotaging the victim so that they cannot get to work [7].

#### *Boredom*

Jobs that require individuals to do the same task on a daily basis can lead to boredom on the work. It could result in unfavourable work practices such as frequently missing work, lack of concentration or withdrawal from the task that the person was hired to do leading to a decrease in work efficiency [8].

#### *Other Counterproductive Behaviours*

Often people come across various other forms of counterproductive behaviours in their workplace. These are: ignoring people at work, working slowly when the work needs to be done fast, refusing to help their colleagues, refusing to accept a task, showing less interest in their work, showing destructive behaviour against their colleagues and not appreciating their colleague's success [9].

#### *Sexual Harassment in the Workplace*

Sexual harassment occurs when one individual either a male or a female takes a sexual interest in the other person while at work and try to exploit him or her. It could lead to the feeling of insecurities and pressures to leave the organisation. Studies also showed that sexual harassment could lead to people feel depressed, result in high level of anxiety and mental as well as physical stress [10].

#### *Verbal Abuse*

Verbal abuse indicates some form of mistreatment using an oral expression [11]. Verbal abuse includes the following: anger with abusive words, accusing and blaming, countering, judging and criticising, name calling, ordering, threatening, age discrimination, bullying, emotional abuse, hate speech, social rejection and so on.<sup>12</sup> Verbal abuse creates emotional pain and anguish. The person abused verbally for long period may succumb to any stress related illness.

The victims may even develop clinical depression, post-traumatic stress disorder and other forms of anxiety disorders. It can reduce the productivity in the workplace affecting the quality of work, turnovers, and even may lead to the resignation of the employee. Despite being the most common form of abuse, verbal abuse is generally not taken seriously

as there is no visible proof [13].

So naturally, the question arises what is *normal behaviour* then? In general, 'normal' refers to a lack of significant deviation from the average. It not only varies from person-to-person [14] but also depends on time, place, and situation – it may change along with changing societal standards and norms. On the individual level, persons who suffer from psychiatric disorders may present with behavioural abnormalities. In severe cases, people who violate social norms, such as criminals, invite a punishment from others in the society [15,16]. At the same time nobody wants to be labelled as sick or abnormal. Most people want to be normal so that they can relate to society at large [17]. Since being normal is generally considered an ideal, there is often pressure from external sources as well as from people's intrinsic desire to feel included.

Before labelling somebody *abnormal eccentricity should be ruled out*. In contrast to normal behaviour, eccentricity is not the quite common ways which individuals in society follow to solve given problems and pursue certain priorities in everyday life. Eccentricity is often associated with genius, intellectual giftedness or creativity. Eccentric behaviour may be the outward expression of their unique intelligence or creative impulse [18].

#### *Management*

##### *Corrective Measures & Lifestyle Issues*

After assessing the relationship between behaviours and health outcomes, studies have established their role in both morbidity and mortality of individuals [19,20]. These studies have indicated towards seven features of lifestyle which were often associated with lower risk for diseases and thereby extending active lifespan [21]. These are avoiding snacks, eating breakfast regularly, exercising on a regular basis, maintaining a desirable body weight, moderate alcohol intake, not smoking, sleeping 7–8 hours per night. The quality of life should be improved by promoting good health and avoiding all types of addictions.

##### *Promoting Positive Mental Health*

At least one counsellor should be there in the workplace or office who will deal with day to day issues where a personalised approach is required. Presentations should be arranged on a regular basis on various topics like time management techniques, how to cope with stress in workplace, yoga and

meditation practices [22]. To promote positive physical and mental health the importance of recreational activities cannot be ignored. There should be space and arrangement for various recreational activities in the office or workplace. Employees should be taught to keep aside some time daily for his/her personal purpose only. All these are required to create a relaxed working environment for the employees [23].

### *Recognition*

Recognising positive and productive behaviour at the workplace can be done using job analysis. Feedback may be given according to the performance. This method gives others a better understanding and evaluation of a typical duty they are looking for. Rewarding the productive behaviour increases the performance further [24].

### *Counselling*

Personal conflict leads to disappointment and dissatisfaction in work leading to lowering of effectiveness. Counselling may be definitely helpful in these cases. If organizations cannot afford to appoint counsellors, staff can be trained by professional counsellors to manage and deal with personal issues effectively with a non-directive approach. To perform the role of a counsellor the person should be a good listener who will listen to with the understanding of the problem [25]. Simply being able to communicate one's feelings to a concerned and active listener is enough to relieve mental disturbances and may facilitate a person to go forward towards a problem-solving frame of mind. Ventilation may help upset employees and workers to cope with their problems [26].

Counselling by professional counsellors with expressing sympathy and empathy to the person concerned increases its effectiveness. While sympathy is an expression of understanding and care for someone else's suffering, empathy is the ability to share someone else's feelings or experiences by putting oneself in the shoes of another.

### *Conflict Resolution*

Conflict resolution at work is important to resolve any issue that arises at work among team members. Conflict resolution or reconciliation is the process and the technique used in facilitating the peaceful ending of conflict and grievance. Few group members may attempt to resolve group conflicts by actively communicating information about their conflicting

motives or ideologies to the rest of the group and by engaging in collective negotiation [27]. Handling these issues appropriately helps decrease harmful influences of all types of conflicts by bringing back integrity, building success in the workplace and restoring efficiency. Resolving conflict allows all disagreements to be fixed in a way that is beneficial to two or more individuals or the group [28].

### *Psychiatric Consultation*

Abnormality in behaviour may occur due to various psychiatric disorders [29]. Whenever it becomes difficult to identify the cause of a particular odd behaviour, psychiatric consultation may be taken. If the psychiatrist diagnoses a disorder and advises any treatment for that, it should be followed appropriately to control the abnormal behaviour pattern.

### **References**

1. Minton EA, Kahle LR.. Belief systems, religion, and behavioral economics : marketing in multicultural environments. New York: Business Expert Press; 2014.
2. Dusenberry, DB. Living at micro scale : the unexpected physics of being small. Cambridge, Mass: Harvard University Press; 2009.
3. Nelson RJ. An introduction to behavioral endocrinology, Sunderland, MA : Sinauer Associates; 2005.
4. Robertson I, Callinan M. Personality and Work Behaviour. European Journal of Work and Organizational Psychology.1998; 7(3):321-340.
5. Balducci C, Schaufeli WB, Fraccaroli F. The job demands-resources model and counterproductive work behaviour: The role of job-related affect. European Journal of Work and Organizational Psychology. 2011; 20(4):467-496.
6. Maria Rotundo & Jia Lin Xie. Understanding the domain of counterproductive work behaviour in China. The International Journal of Human Resource Management. 2008; 19(5):856-877.
7. Fox S, Spector PE, Goh A, Bruursema K, Kessler SR. The deviant citizen: Measuring potential positive relations between counterproductive work behaviour and organizational citizenship behaviour. Journal of Occupational and Organizational Psychology. 2012; 85:199-220.
8. Bruursema K, Kessler SR, Spector PE. Bored employees misbehaving: The relationship between boredom and counterproductive work behaviour. Work & Stress. 2011; 25(2):93-107.

9. Neff, WS. *Work and human behavior*. New York: Aldine Pub. Co; 1985.
10. McDonald P, Charlesworth S. Workplace sexual harassment at the margins. *Work, Employment & Society*. 2015; 30(1):118-134.
11. Oweis A, Diabat KM. Jordanian nurses perception of physicians' verbal abuse: findings from a questionnaire survey. *International Journal of Nursing Studies*. 2005; 42(8):881-888.
12. Bosch K. When Words Are Used As Weapons: The Signs of Verbal Abuse (Part 2 of a four part series) 2004. Historical Materials from University of Nebraska-Lincoln Extension. Paper 545.
13. Evans P. *The Verbally Abusive Relationship: How to recognize it and how to respond*. Avon, Massachusetts: Adams Media; 2010.
14. Hur T, Roese NJ, Namkoong J. Regrets in the East and West: Role of intrapersonal versus interpersonal norms. *Asian Journal of Social Psychology*. 2009; 12(2):151-156.
15. Durkheim É. *Rules of Sociological Method*. New York: Free Press; 1982.
16. Jones RA, Durkheim E: *An Introduction to Four Major Works*. Beverly Hills, CA: Sage Publications; 1986.
17. Bridgens R. Disability and Being 'Normal': A Response to McLaughlin and Goodley: Response. *Sociology*. 2009; 43(4):753-761.
18. Weeks DJ, James J. *Eccentrics : A study of sanity and strangeness*. New York: Villard Books; 1996.
19. Kooiker S, Christiansen T. Inequalities in health: the interaction of circumstances and health related behaviour. *Sociology of Health & Illness*. 1995; 17 (4):495-524.
20. Blaxter M. *Health and Lifestyles*. London: Routledge;1990.
21. Belloc NB, Breslow L. Relationship of physical health status and health practices. *Prev Med*. 1972; 1(3): 409-421.
22. Feuerstein G. *Textbook of Yoga*. London : Rider & Co; 1975.
23. Hariharan M, Rath R. *Coping with Life Stress: The Indian Experience*. New Delhi : SAGE Publications Pvt. Ltd; 2008.
24. Davis K. *Human behavior at work : human relations and organizational behaviour*. New York: McGraw-Hill; 1972.
25. Knowles HP, Saxberg BO. *Personality and Leadership Behavior*. Reading, MA: Addison-Wesley Publishing Co; 1971.
26. Johnson RA. *Management, Systems, and Society: An Introduction*. Pacific Palisades, Calif.: Goodyear Pub. Co; 1976.
27. Forsyth DR. *Group Dynamics*. Boston, MA: Wadsworth; 2009.
28. Behfar KJ, Peterson RS, Mannix EA, Trochim WMK.. The critical role of conflict resolution in teams: A close look at the links between conflict type, conflict management strategies, and team outcomes: Correction. *Journal of Applied Psychology*. 2008; 93(2):462.
29. World Health Organization. *The ICD-10 classification of Mental and Behavioral Disorders : Clinical Descriptions and Diagnostic Guidelines*. Geneva: WHO; 1992.

## Neurological Soft Signs in Schizophrenia

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### Abstract

Neurological Soft Signs (NSS) are postulated to reflect functional disorders in selective areas of the brain. Studies have reported that during the first schizophrenia episode higher total rates of NSS and motor problems are already present. Schizophrenia patients and first degree relatives have more NSS compared to patients with other psychiatric disorders and healthy controls. Whether NSS have a developmental origin or result from some early acquired lesion remains to be decided. Genetic cause of NSS is suggested by studies that indicate that NSS is increased in patients with a positive family history as well as in unaffected first degree relatives of schizophrenia patients. A relationship between NSS and different subtypes of schizophrenia has been observed. Studies indicate that NSS are associated with a more serious clinical course, escalated cognitive dysfunction and unsatisfactory psychosocial outcome has been reported.

**Keywords:** Neurological Soft Signs; Schizophrenia; Brain Disorder; Genes.

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### Introduction

“Neurological Soft Signs (NSS)” or “Soft Neurological Signs (SNS)” in schizophrenia have been reported since the 19th century [1]. NSS probably indicate the presence of functional disorders in some brain areas and not diffuse brain dysfunction [2]. The term “Soft Neurological Signs”(SNS) was introduced by Bender who conceptualized it as a “developmental lag”, akin to organic signs found in psychiatric disorder in the aged, and defined SNS as follows: SNS is not due to any postnatal neurological insult that may leave residual neurological signs e.g. severe head injury, intoxication, infection or tumor. Grouping of SNS found in an individual should not have a pathognomonic pattern of a kind that would indicate one or more clearly localized structural lesions, generalized encephalopathy or CNS involvement [3]. Subsequent studies supported the

idea that NSS may be related to a specific deficit in the function or anatomical regions of the brain[4,5]. NSS are mild, non-localizing, neurological signs that are identified from deficits in performance in domains such as sensory integration, motor coordination, and motor sequencing. At the beginning of the initial episode of schizophrenia both medicated and treatment-naïve patients exhibit elevated rates of NSS and motor abnormalities [6,7]. In addition individuals at high risk for schizophrenia have elevated rates of NSS as compared to controls. [8] NSS have also been detected in other psychiatric disorders like OCD, though rates of NSS are significantly higher in patients with schizophrenia [9].

Neurological abnormalities are traditionally classified as “hard signs” (impairments in basic sensory, motor and reflex behaviors not seen in schizophrenia) and “soft signs”. (complex phenomena of aberrations in motor activity,

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integrative sensory functions, sensorimotor integration, and cerebral laterality) [10,11]. Soft signs are traditionally organized into seven categories including: 1) Integrative sensory dysfunction; 2) Motor incoordination; 3) Impaired sequencing of complex motor tasks; 4) Frontal release signs; 5) Abnormal eye movements; 6) Memory impairments; and 7) Cerebral dominance [12].

Over the last five decades numerous studies have reported that patients with schizophrenia and their first degree relatives have significantly more NSS than healthy controls and patients with other psychiatric disorders [13-16]. Prevalence of NSS in schizophrenia range from 50% to 73%, compared with 5% in controls. Positive symptoms tend not to be related to NSS, whereas negative symptoms have been related to soft signs that reflect frontal (motor function) and parietal (sensory integration) functions [13]. Cognitive performance is partially linked with NSS, but is also influenced in a way that soft signs are not by sociodemographic variables such as age, education, sex, and socioeconomic status [13]. NSS have been associated with multiple clinical features of schizophrenia, have been conceptualized as a vulnerability marker for schizophrenia, and may represent a phenotype useful in genetic studies. Although NSS are held to have little localizing value, this is not entirely true; for example, motor perseveration is associated with damage to the dorsolateral prefrontal cortex [17,18] grasp reflex localizes to the frontal lobes, and soft signs have identifiable functional neuroimaging correlates [16,19-20]. However, their value lies more in that their presence indicates dysfunction within the distributed neural networks that underlie complex behaviors [11]. Thus, while most of the primitive reflexes (e.g. palmomental, snout, and glabellar reflexes) are not localizable, they do indicate cortical deterioration or diffuse cerebral dysfunction in patients diagnosed with schizophrenia [21]. NSS may provide valuable prognostic information, as they may be associated with greater psychopathology, [22] more severe cognitive dysfunction, [23] poorer treatment response [24] and a high risk for the occurrence of tardive dyskinesia [21].

#### *Developmental Aspects*

Pediatric neurologists widely consider NSS as having a developmental origin. It is supported by the evidence that a higher prevalence of Neurological Abnormalities (NAs) in younger than older children) [25]. These signs follow a maturational curve, which reaches adult level at approximately 8

years in normal individuals [26]. The increase in NAs with age up to 8 years was thought to be the consequence of the maturation of the CNS, as dysfunction can be assessed reliably only when the structure involved has become functionally active. The maturation complete by 9 years [27]. Persistence of these neurological signs after 9 years of age indicates that there has been a "developmental lag" in the process of developing complex integrative function. Testing this hypothesis showed that when children were divided into 2 groups above and below 8 years, proportionately greater number of signs was found in the younger age group [26]. But all children previously shown to be having neurological signs still had neurological signs of one sort or other at follow up [28]. Further, the rate of neurological signs still increased beyond the age of 9 yrs mainly in boys [27].

Another possibility for the origin of NAs is that it results from some early acquired lesion i.e. it represents a feature of brain damage [29]. One study observed a significant excess of low birth weight for gestation among males with NAs at the age of 7 [30]. Another study reported that schizophrenia patients but not their siblings showed significantly more obstetric complications compared to their respective neonatal controls. However there was a lack of significant relationship between NAs and obstetric complications in the patient group, indicating that besides perinatal events there are other determinants of NAs in schizophrenia [31].

A third possibility is that NAs may be a heritable individual difference based on reports of significantly more NAs in the siblings [32] and first degree relatives [15] of schizophrenia patients. These studies indicate a higher rate of NAs characterizing a portion of the offspring's and relatives of schizophrenia patients who are at a higher genetic risk of developing schizophrenia. This is compatible with the view that NSS reflect a familial transmitted alteration in neurological process that constitutes a vulnerability or diathesis to subsequent schizophrenia.

A number of neurological signs have been described as occurring more frequently in children with psychiatric disorders than in normal controls [33-36]. Almost all studies have shown a relationship between presence of these signs and age and I.Q., and for a given age and I.Q. occur more frequently in boys. Many of these signs fail to discriminate between problem and non problem children when such factors are taken into account. However, even after taking account of age, sex, and I.Q. both dysdiadochokinesia and dysgraphaesthesia are more frequently observed in disturbed children [33].

### *Prevalence of NSS in Psychiatric Illnesses*

Hertzog and Birch found that a high population of psychotic patients had abnormal "soft signs". These studies were uncontrolled and did not take account of current drug intake, raters were not blinded, included patients with frank neurological disease, and also listed hyperkinetic behavior as a soft sign [22].

An excess of soft signs was found among patients with schizophrenia and personality disorder, but not in patients with affective disorders in a study of 65 random admissions to 3 adult psychiatric units, with drug free period of 48 hours with 20 staff controls [37]. Another study involved 298 consecutive admissions under age 50, none of whom had organic neurological disease or had been treated previously with ECT, and were drug free for at least 10 days prior to examination.

It was found both the schizophrenia groups, viz. with Premorbid Asociality (adult schizophrenia who had experienced marked personality difficulty in childhood, characteristically being friendless, having academic difficulties and narrow interests) and Emotionally Unstable Character Disorder (characteristically antisocially impulsive, who had frequent but brief non-reactive mood swings), differed from other diagnostic categories in having more of dysdiadochokinesia, agraphaesthesia, mirror movements, finger apraxia, disturbances of speech and gait [38].

Primitive neurological soft signs like grasp, snout and palmonental reflexes are present in a considerable number of older people and in organic and functional psychosis [39]. Higher frequency of neurological signs have been reported in studies in schizophrenic subjects as compared to other psychiatric and non-psychiatric subjects [40,41]. A review reported an average prevalence of 50-60 % neurological abnormalities in schizophrenia patients [13].

### *Genetic Basis of NSS*

An early study showed that schizophrenics with a positive family history manifested significantly more neurological abnormalities than normal controls, and the schizophrenics with no family history were not significantly different from normal or psychiatric controls on any of the measures. The authors hypothesize that schizophrenics with a positive family history constitute a distinct subgroup with genetic contribution greater than the patients without a family history [42].

Schizophrenia patients without positive family history had an excess of primary signs (dysfunction identified by a standard neurological examination, lateralizing limb pyramidal signs and frontal release signs) compared to normal controls. Both the schizophrenic group with positive family history and their first degree relatives showed an increase in integrative signs (depends on integration within the motor and sensory systems or between the motor and sensory systems). These findings indicate that different mechanisms produce the brain dysfunction in familial and sporadic schizophrenia [43].

In 24 schizophrenia patients, 21 of their non-schizophrenic first degree relatives and 29 normal controls, the prevalence of neurologic abnormalities in relatives was congruous to that among schizophrenia subjects but significantly greater than in controls. Based on signs of localizing motor system abnormalities a marked difference was noted between relatives and controls. On the basis of these results the authors suggest that overt schizophrenia may result from the combined operation of two independent familial factors, firstly "psychopathologic" and secondly "neurologic" [32].

NSS was assessed in 58 DSM III schizophrenia patients, their 31 healthy first degree relatives and 38 normal controls by two assessors blind to the diagnoses using a standardized neurological assessment procedure. Schizophrenia patients and their first degree relatives showed more severe NSS than the normal controls indicating that these signs may be the result of a family related pathophysiological process [44]. The lack of family history data for other first degree relatives makes the control of information variance due to illness heterogeneity in terms of genetic loading difficult and the possible influence of medication on NSS cannot be completely ruled out.

### *Neuroanatomical and Neurotransmitter Abnormalities and NSS*

The "cognitive dysmetria" theory explained the diversity of symptoms in schizophrenia by scattered disturbance in the cortico-cerebellar-thalamic-cortical circuit, [45] which may also be related to NSS abnormalities[46]. Though the exact anatomical localization of NSS is yet to be determined, the "network inhibition hypothesis" posits a central role to the interconnections among basal ganglia, cerebellum, cerebral cortex and dopamine neurotransmission which inhibit voluntary movements [47]. 22q11 Deletion syndrome (22q11DS) occurs due to hemizygous microdeletion

on the long arm of chromosome 22. The increased prevalence of NSS in this syndrome is believed to be due to catechol-O-methyltransferase COMT haploinsufficiency, dopamine dysfunction, and white matter abnormalities [48].

#### *Demographic Variables and NSS*

**Age:** Contradictory results have been reported by studies of NSS and age varying from no correlation[49], a negative correlation[50], while one study reported a positive correlation with age [51]. However, most studies fail to give the age range of their patient populations, and it may be the case that the range is too narrow to adequately evaluate or detect an age effect [12].

**Sex:** Conflicting results are reported by studies of the relationship of sex with NSS. Few studies observed no differences related to gender [49,51] while others reported slightly more [12,22,52] or significantly more [37] neurological impairment in males.

**Education:** A significant correlation between educational level and soft neurological signs in schizophrenia was demonstrated [44]even after controlling for age and sex [53]. A possible explanation for this relation may be that NSS may be evidence of an early cerebral insult as the resultant CNS dysfunction may lead to poor educational attainment even before the illness. Another argument is that lower education is an index of poor socio-economic status which is more likely to expose these patients to infections and deficiencies in early childhood [53].

**Race and Ethnicity:** Caucasian patients and controls have lower prevalence of neurological impairment [12,23, 54], and cognitive/perceptual neurological abnormalities [55] compared to African-American patients and controls.

#### *Temporal Stability of NSS*

There has been very little attempt to assess the temporal stability of NA across time, with few exceptions, especially in child psychiatry literature. Examination of a group of children for NSS after a few years revealed a reduction in the frequency of NSS in older children, which was attributed to neuronal maturation. However, the number of children with two or more NSS did not differ significantly at the two time points. Stability of positive findings was highest for speech, followed by coordination and double simultaneous stimulation [56]. Since the consistency of signs was

directly related to chronological age and maturity, the instability in neurological signs over short term was more likely to occur in the most immature and psychiatrically impaired children [26]. In sub-chronic and chronic schizophrenia patients retested after 2-10 months, 4 out of 6 patients initially equivocal for the presence of neurological signs had demonstrable signs, while 20 out of 24 patients initially positive for presence of neurological signs remained so. However, in acute group, 3 out of 4 patients who had had equivocal signs had no signs of neurological impairment at retesting [57]. Similar findings have been reported by others [21].

No difference in NSS was found in inpatients and outpatients with schizophrenia, groups that would presumably differ in clinical states [52]. A five year follow up study of schizophrenia patients [58] observed a number of neurological abnormalities in patients with a non-remitting course of disease and in patients with genetic predisposition (with obstetric complications). In another study moderate stability of neurological signs from childhood to adolescence has been demonstrated in the offspring of schizophrenic patients [59]. A prospective study comprising of first-episode schizophrenia patients revealed that improvement in motor-related and cortical neurological soft signs at six months, was associated with improvement in psychopathology. However, harder T signs tended to deteriorate [60].

#### *Relation of NSS to Psychiatric Symptoms in Schizophrenia*

Till date, several lines of research indicate a relationship between NSS and the pathophysiology of schizophrenia. The base rate of NSS in schizophrenia patients is approximately 60% [12]. As compared to controls higher rates of NSS have been observed in first-episode, [6] as well as both treatment-naïve and medicated schizophrenia patients [7] as well as individuals at high risk for schizophrenia (e.g., schizotypal personality disorder)[61]. Furthermore, there is evidence for a genetic component to NSS, as family members of schizophrenia subjects exhibit elevated levels of NSS than matched control subjects [62].

Numerous studies have investigated the relationship between NSS and schizophrenia symptoms with ambiguous results. A relationship of NSS with positive symptoms was reported by one study [63] but not by others [49,64]. Similarly, an association of negative symptoms and NSS was observed in some studies [15,65], but not others. [66-67].

### *Relationship between NSS and Scores on Rating Scales*

Partial correlations, controlling for duration of illness, were used to test the relationships between Baseline NSS ratings and Baseline BPRS subscale scores, and subscale scores after treatment. At Baseline, there were significant positive correlations between levels of NSS and BPRS Positive and BPRS Negative ratings, and a significant negative correlation between NSS and the BPRS Psychological Discomfort subscale scores. The pattern of results after treatment was similar to that at Baseline: there was a significant positive correlation between NSS and BPRS Positive ratings and a significant negative correlation between NSS scores and scores on the BPRS Psychological Discomfort subscale but BPRS Negative Symptoms were not significantly related to NSS [8].

### *Relation of NSS and Clinical Variables at Different Stages of the Illness*

At initial presentation, no significant correlation was found between the levels of neurological soft signs and the levels of positive and negative symptoms, affective symptoms and obsessive compulsive symptoms, or measures of extra-pyramidal signs. The picture is similar upon clinical stabilization following medication. At the end of the first year, a moderate correlation with negative symptoms emerged. A significant correlation of motor coordination NSS with a wider range of negative symptoms was evident by the end of the second year. There were also modest correlations with the global HEN (The High Royd Evaluation of Negativity Scale) [68] score and the negative symptoms subscale scores of the PANSS. By year three, HEN subscales of thought and affect showed significant correlations [69].

### *Relationship between NSS and the Psychopathology and Course of Schizophrenia*

Earlier studies observed a relationship between NSS and different subtypes of schizophrenia, such as chronic v. acute schizophrenia [57] and disorganized v. nondisorganized schizophrenia [70]. NSS were also associated with total number of psychiatric symptoms, [71] thought disorder, [51] negative symptoms [15] and emotional stability [38]. In contrast some studies found no association between NSS and positive symptoms, [49] or paranoid/non-paranoid schizophrenia [52]. Other studies have also characterized similar conflicting results on relationship between NSS and psychopathology in first episode psychosis. Few studies reported an association between NSS and

total symptom severity and positive symptoms [63] whereas others found no association with global measures of psychopathology [72] or with positive and negative dimensions of schizophrenia [64]. The discrepant findings could be due to use of different scales for detecting NSS. It has been suggested that the correlation between total NSS and positive symptoms may reflect attentional deficits secondary to untreated symptoms [63].

The association between NSS and a more severe and chronic form of schizophrenia has also been investigated by examining patients at different stages of the illness. This has been supported by the association of NSS with young age at onset, a more chronic course [57], longer index hospitalization [37] and impaired premorbid functioning [38,49]. However, some studies failed to demonstrate an association of NSS with age at onset, poor premorbid functioning, number of hospitalizations in a 3-year follow-up, and lifetime hospitalizations [49,57,71]. A majority of first episode studies have reported no correlation between NSS and age at onset, [58] duration of untreated psychosis [58,63], global assessment of functioning [72], and occupational outcome [73]. It is possible that factors such as global assessment of functioning and occupational outcome are worse in more advanced phases of the illness, and are therefore not associated with neurological dysfunction in the early stages. On the other hand, few studies have demonstrated an association between NSS and both poorer premorbid social adjustment [63] and duration of hospitalization [73]. The above mentioned associations may probably be related to the fact that higher rates of NSS are part of a more severe clinical picture, which probably could explain the longer period of hospitalization; it is also possible that this is reflected in longer pharmacological therapy which may give rise to more NSS.

In conclusion, the studies reviewed confirm that an excess of NSS is already evident in patients suffering their first episode of schizophrenia and in high-risk subjects without psychosis. Neurological performance is worse in sensory integration, motor coordination and sequencing, and in developmental reflexes. These NSS are associated with male gender, lower education, and a more severe clinical picture. The NSS are not a consequence of antipsychotic drug use, although first-episode schizophrenia patients on antipsychotic treatment obtain higher NSS scores.

### *Neurological Soft Signs in Non-Psychotic First-Degree Relatives*

In schizophrenia patients, NA are frequently noted but their pathophysiological importance remains

elusive [4,55]. Family studies have consistently demonstrated that nonschizophrenic relatives of probands including parents [44,64], siblings [40], and offspring [59] exhibit increased rates of NAs. A high degree of correlation is seen within families and it has been claimed that the degree of genetic loading for schizophrenia within the family may be a determining factor [40,43,74].

Previous studies of MZ twins discordant for schizophrenia [75-77] indicate that the non-schizophrenic co-twins lie midway between probands and healthy controls in the extent of NAs detected. An association between obstetric complications and NAs in both the probands and their well co-twins [75], and non twin relatives [31] has been reported. Based on the above it was postulated that NAs in schizophrenia reflect the impact of environmental agents in genetically sensitized individuals, and that patients from MZ discordant pairs are subject to greater environmental effect than MZ twins from pairs concordant for schizophrenia [31].

NAs have been divided into primary and integrative subscales. Primary NAs include cranial nerve signs, eye movement abnormalities, lateralizing limb pyramidal neurology and frontal release signs, which are caused by dysfunction that can be detected at routine neurological examination. Integrative NAs reflected dysfunction in the integration of activity within and between the sensory and motor systems, and include dysdiadokokinesia, and the sequencing of complex motor acts, such as the fist-edge-palm test. Primary NAs were elevated in nonfamilial cases of schizophrenia, but not in their well relatives.

In contrast to this, integrative NAs were elevated both in probands and unaffected relatives in families in which multiple members suffered from schizophrenia. In view of the above findings it is believed that environmentally induced neurological damage results in primary NAs, while genetic loading for schizophrenia is the cause of integrative NAs [43].

In accord, most studies have demonstrated that biological relatives of schizophrenia patients have elevated NSS than individuals without an immediate family history [77-79] but some studies reported opposite findings [80]. Additionally NSS severity appears to be graded, with patients showing the most, unaffected controls showing the least, and first degree relatives falling in between[44]. The above findings indicate that the origin of NSS is partly genetic, and that such abnormalities may be intermediate phenotypes, or endophenotypes [81].

#### *Assessment of NSS*

The clinical utility of direct examinations is a function of useful information yielded per time spent on the examination. There have been efforts made towards optimizing clinical utility by improving reliability and validity. Undoubtedly, the use of reliable and valid neurologic test items will ensure that the results of a specific test would inform individual cases. Since individual tests have insufficient power, aggregations of items have to be used [82]. However, selecting item aggregations has proved difficult. Few studies treat the neurologic examination as unidimensional, working only if a single summary score documenting the presence or absence of soft signs[83-84]. Others problematically divide items into "hard" and soft) categories [67]. Still others group items based on the effects of lesions acquired after normal brain development. None of these approaches are entirely satisfactory or well supported. Different instruments devised for assessment of NSS are as under:

1. Woods scale [85],
2. Condensed Neurological Examination (CNE) [44],
3. Modified Quantified Neurological Scale (MQNS) [86],
4. Heidelberg Scale [71],
5. Cambridge Neurological Inventory (CNI) [87],
6. Neurological Soft Sign Scale [38],
7. Brief Motor Scale [88],
8. Neurological Evaluation Scale (NES) [4],
9. Extended Standard Neurological Assessment Instrument [40],
10. Short NeurologicalEvaluation Scale (S-NES) [89].

A number of studies have used the NES in samples of patients of schizophrenia. Scores obtained by patients with schizophrenia and their siblings was higher than those of normal controls on the Soft Signs Total, as well as the Sensory Integration and Motor Functioning subscales [40]. Additionally, schizophrenia patients reported higher scores compared to at risk patients, who in turn scored higher than controls on the Soft Signs Total, Sensory Integration, and Other Soft Signs[90]. The Other Soft Signs subscale of the NES correctly classified the maximum number of patients and controls to their true group [23] indicating that the Other Soft Signs subscale is particularly sensitive in identifying individuals with schizophrenia or a proneness to it. The S-NES comprise of 12 empirically determined items of the

NES that showed high agreement with the 26 items in the original NES (sensitivity=96.3%, specificity=100%) [89].

## Conclusion

Much debate has centered on the relative contribution of genetic and environmental factors to the etiology of schizophrenia. Review of the literature suggests that NSS are not a consequence of antipsychotic treatment but may imply cerebral dysfunction that needs further investigation, mainly to understand the effect of genetic-environmental contribution to neuro dysfunction in schizophrenia. The base rate of NSS in schizophrenia patients is approximately 60% [12]. NSS in relatives of schizophrenia patients increases with the potential genetic loading [43]. Further, the Total Soft Signs score could be used to distinguish relatives with a genetic vulnerability to schizophrenia from those who were not [63]. The above suggests that the NSS may identify a subject of being a "gene-carrier" for psychosis. In contrast, the results of another study suggests that NSS are not an indicator of genetic risk specifically for psychosis [90]. Other causes of NSS may be low birth weight [91] and obstetric complications [31].

NSS have been associated with a worse clinical course of psychosis, poor psychosocial performance, and cognitive dysfunction [38,49,72,92], suggesting a subgroup of patients characterized by a more dire pathophysiological process. Comparison of the prevalence of NSS in patients with psychosis across various studies is confounded by the fact that different scales (some of which may not be published or validated) have been used for assessment of NSS.

Studies conducted till date have several limitations. The presence of significant effect variability in few studies indicate that the average effect sizes may not represent adequately the underlying populations, which may include important subsets of patients. The relationships between NSS and potential moderators like age and duration of illness will need further detailed analysis. NSS are detected in a majority of patients with schizophrenia and are similar to or may even exceed the cognitive, psychophysiological and neuro-anatomic findings as indicators of schizophrenia. Additionally important queries about the illness remain regarding prevalence of specific soft signs, the sources of heterogeneity and effect variability.

## References

1. Schröder J, Heuser M. Neurological Soft Signs in First -Episode Schizophrenia. *Directions in Psychiatry* 2008; 28:243-257.
2. Praharaj SK, Ram D, Arora M. Neurological Abnormalities in Drug-free and Drug-treated Patients with Bipolar Affective Disorder. *Hong Kong Journal of Psychiatry* 2005; 15:82.
3. Bender L. *Psychopathology of children with organic brain disorders*. Springfield, IL, US: Charles C Thomas Publisher. 1956.
4. Buchanan RW, Heinrichs DW. The Neurological Evaluation Scale (NES): a structured instrument for the assessment of neurological signs in schizophrenia. *Psychiatry Res* 1989; 27(3):335-350.
5. Cox SM, Ludwig AM. Neurological soft signs and psychopathology: Findings in schizophrenia. *J Nerv Ment Dis* 1979; 167:161-165.
6. Dazzan P, Murray RM. Neurological soft signs in first-episode psychosis: a systematic review. *Br J Psychiatry* 2002; 43:50-57.
7. Venkatasubramanian G, Latha V, Gangadhar B, Janakiramaiah N, Subbakrishna DK, Jayakumar PN, Keshavan MS. Neurological soft signs in never-treated schizophrenia. *Acta Psychiatr Scand* 2003; 108(2):144-146.
8. Mittal VA, Hasenkamp W, Sanfilipo M, Wieland S, Angrist B, Rotrosen J, Duncan EJ. Relation of neurological soft signs to psychiatric symptoms in schizophrenia. *Schizophrenia Res* 2007; 94:37-44.
9. Jaafari N, Baup N, Bourdel MC, Olié JP, Rotge JY, Wassouf I, Sharov I, Millet B, Krebs MO. Neurological soft signs in OCD patients with early age at onset, versus patients with schizophrenia and healthy subjects. *J Neuropsychiatry Clin Neurosci*. 2011; 23(4):409-16.
10. Boks MP, Russo S, Knegtering R, van den Bosch RJ. The specificity of neurological signs in schizophrenia: a review. *Schizophrenia Res* 2000; 43(2-3):109-116.
11. Ovsiew F. Bedside neuropsychiatry: eliciting the clinical phenomena of neuropsychiatric illness. In *Textbook of Neuropsychiatry*. Eds Yudofsky et al 5th ed .American Psychiatric Press, Washington, DC. 2008. pp 137-188.
12. Heinrichs DW, Buchanan RW. Significance and meaning of neurological signs in schizophrenia. *Am J Psychiatry*, 1988; 145(1):11-18.
13. Bombin I, Arango C, Buchanan RW. Significance and meaning of neurological signs in schizophrenia: two decades later. *Schizophrenia Bull* 2005; 31(4):962-977.
14. Chan RC, Xu T, Heinrichs RW, Yu Y, Gong QY. Neurological soft signs in non-psychotic first-

degree relatives of patients with schizophrenia: A systematic review and meta-analysis. *Neurosci Biobehav Rev* 2009; 34:889-96.

15. Hembram M, Simlai J, Chaudhury S, Biswas P. First Rank Symptoms and Neurological Soft Signs in Schizophrenia. *Psychiatry Journal* 2014, Article ID 931014, 11 pages. doi:10.1155/2014/931014.
16. Gunasekaran V, Venkatesh VM, Asokan TV. A study of soft neurological signs and its correlates in drug-naïve patients with first episode psychosis. *Indian J Psychol Med*. 2016; 38(5):408-413.
17. Luria AR. Two kinds of motor perseveration in massive injury of the frontal lobes. *Brain* 1965; 88: 1-10.
18. Milner B. Some effects of frontal lobectomy in man. In: Warren, J and Akert, K, Eds. *The Frontal Granular Cortex and Behavior*. New York: McGraw-Hill. 1964
19. Rao H, Di X, Chan R, Ding Y, Ye B, Gao D. A regulation role of the prefrontal cortex in the fist-edge-palm task: evidence from functional connectivity analysis. *Neuroimage* 2008; 41:1345-1351.
20. Schroder J, Wenz F, Schad LR, Baudendistel K, Knopp MV. Sensorimotor cortex and supplementary motor area changes in schizophrenia. A study with functional magnetic resonance imaging. *Br J Psychiatry* 1995; 167(2):197-201.
21. King DJ, Wilson A, Cooper SJ, Waddington JL. The clinical correlates of neurological soft signs in chronic schizophrenia. *Br J Psychiatry* 1991; 158: 770-775.
22. Hertzig M E, Birch HG. Neurologic organization in psychiatrically disturbed adolescent girls. *Arch Gen Psychiatry* 1966; 15(6):590-598.
23. Arango C, Bartko JJ, Gold JM, Buchannan RW. Prediction of neuropsychological performance by neurological signs in schizophrenia. *Am J Psychiatry* 1999; 156(9):1349-1357.
24. Smith RC, Hussain MI, Chowdhury SA, Stearns A. Stability of neurological soft signs in chronically hospitalized schizophrenic patients. *J Neuropsychiatry Clin Neurosci* 1999; 11(1):91-96.
25. Shaffer D, Schonfeld IS, O'Connor PA, Tokman C, Trautman P, Shafer S, Ng S. Neurological soft signs and their relationship to psychiatric disorder and intelligence in childhood and adolescents. *Arch Gen Psychiatry* 1985; 42:342-351.
26. Shapiro T, Burkes L, Petti TA, Panz J. Consistency of "nonfocal" neurological signs. *J Am Acad Child Psychiatry* 1978; 17:70-79.
27. Lunsing RJ, Hadders-Algra M, Huisjes HJ, Touwen BCL. Minor neurological dysfunction from birth to 12 years. II. Puberty is related to decreased dysfunction. *Developmental Medicine and Child Neurology* 1992; 34:404-409.
28. Hertzig MD. Stability and change in nonfocal neurological signs. *J Am Acad Child Psychiatry*. 1982; 21:231-236.
29. Precht HFR, Stemmer CH. The choreiform syndrome in children. *Developmental Medicine and Child Neurology* 1962; 4:665-674.
30. Shaffer D, O'Connor PA, Shaffer SQ. Neurological soft signs: Their origin and significance for behavior. In Rutter M (Ed.) *Developmental Neuropsychiatry*. Churchill-Livingstone, London. 1984. pp 144-173.
31. Cantor-Graae E, Ismail B, McNeil TF. Are neurological abnormalities in schizophrenic patients and their siblings the result of perinatal trauma? *Acta Psychiatr Scand* 2000; 101:142-147.
32. Kinney DK, Woods BT, Yurgelun-Todd D. Neurological abnormalities in schizophrenic patients and their families: II. Neurologic and psychiatric findings in relatives. *Arch Gen Psychiatry* 1986; 43:665-668.
33. Adams RM, Kocsis JJ, Estes RE. Soft neurological signs in learning disabled children and controls. *Am J Dis Child* 1974; 128:614-618.
34. Mandelbaum DE, Stevens M, Rosenberg E, Wiznitzer M, Steinschneider M, Filipek P, et al. Sensorimotor performance in school-age children with autism, developmental language disorder, or low IQ. *Dev Med Child Neurol* 2006; 48:33-9.
35. Dickstein DP, Garvey M, Pradella AG, Greenstein DK, Sharp WS, Castellanos FX, et al. Neurologic examination abnormalities in children with bipolar disorder or attention deficit/hyperactivity disorder. *Biol Psychiatry* 2005; 58:517-24.
36. Patankar VC, Sangle JP, Shah HR, Dave M, Kamath RV. Neurological soft signs in children with attention deficit hyperactivity disorder. *Indian J Psychiatry* 2012; 54(2):159-165.
37. Rockford JM, Detre T, Tucker G J, Harrow M. Neuropsychological impairment in functional psychiatric disease. *Arch Gen Psychiatry* 1970; 22: 114-119.
38. Quitkin F, Rifkin A, Klein DF. Neurologic soft signs in schizophrenia and character disorders. Organicity in schizophrenia with premorbid asociality and emotionally unstable character disorders. *Arch Gen Psychiatry* 1976; 33(7):845-853.
39. Keshavan MS, Vikram K, Channabasavanna SM. A critical evaluation of infantile reflexes in neuropsychiatric diagnoses. *Ind J Psychiatry* 1979; 21:267-270.
40. Ismail B, Cantor-Graae E, Cordenal S, McNeil TF. Neurological abnormalities in schizophrenia: clinical, etiological and demographic correlates. *Schizophrenia Res* 1998; 30:229- 238.
41. Malla AK, Norman RMG, Aguilor O, Cortese L. Relationship between neurological 'soft signs' and syndromes of schizophrenia. *Acta Psychiatr Scand* 1997; 96:274-280.
42. Walker E, Shaye J. Familial schizophrenia: a

predictor of neuromotor and attentional abnormalities in schizophrenia. *Arch Gen Psychiatry* 1982; 39:1152-1156.

43. Griffiths TD, Sigmundsson T, Takei N, Rowe D, Murray RM. Neurological abnormalities in familial and sporadic schizophrenia. *Brain* 1998; 121:191-203.
44. Rossi A, De Cataldo S, Di Michele V, Manna V, Ceccoli S, Stratta P, Casacchia M. Neurological soft signs in schizophrenia. *Br J Psychiatry* 1990; 157:735-739.
45. Andreasen NC, Paradiso S, O'Leary DS. "Cognitive dysmetria" as an integrative theory of schizophrenia: a dysfunction in cortical-subcortical-cerebellar circuitry? *Schizophr Bull*. 1998; 24:203-218.
46. Mouchet-Mages S, Rodrigo S, Cachia A, Mouaffak F, Olie JP, Meder JF, Oppenheim C, Krebs MO. Correlations of cerebello-thalamo-prefrontal structure and neurological soft signs in patients with first-episode psychosis. *Acta Psychiatr Scand* 2011; 123(6):451-8.
47. Pasini A, D'agati E. Pathophysiology of NSS in ADHD. *World J Biol Psychiatry* 2009; 10(4 Pt 2): 495-502.
48. Casarelli L, Minnei M, Pitzianti M, Armando M, Pontillo M, Vicari S, Pasini A. Dopamine dysfunction in 22q11 deletion syndrome: possible cause of motor symptoms. *Psychiatr Genet*. 2016; 26(5):187-92.
49. Kolakowska T, Williams A, Jambor K, Ardern M. Schizophrenia with good and poor outcome: III. Neurological "soft" signs, cognitive impairment and their clinical significance. *Br J Psychiatry* 1985; 146:348-357.
50. Peters JE, Roming JS, Dykman RA. A special neurological examination of children with learning disabilities. *Developmental Medicine and Child Neurology* 1975; 175:63-75.
51. Tucker GJ, Campion EW, Silberfarb PM. Sensorimotor functions and cognitive disturbance in psychiatric patients. *Am J Psychiatry* 1975; 132: 17-21.
52. Manschreck TC, Ames D. Neurologic features and psychopathology in schizophrenic disorders. *Biol Psychiatry* 1984; 19(5):703-719.
53. Shaji KS, Richard J, Verghese A. Neurologic abnormalities in schizophrenic patients and their relatives. *Ind J Psychiatry* 1990; 32(3):223-228.
54. Chen EY, Kwok CL, Au JW, Chen RY, Lau BS. Progressive deterioration of soft neurological signs in chronic schizophrenic patients. *Acta Psychiatr Scand* 2000; 102(5):342-349.
55. Keshavan MS, Sanders RD, Sweeney JA, Diwadkar VA, Goldstein G, Pettegrew JW, Schooler NR. Diagnostic specificity and neuroanatomical validity of neurological abnormalities in first-episode psychoses. *Am J Psychiatry* 2003; 160:1298-1304.
56. Hertzig M A, Birch HG. Stability and change in nonfocal neurologic signs. *J Am Acad Child Psychiatry* 1982; 21:231-236.
57. Torrey EF. Neurological abnormalities in schizophrenic patients. *Biol Psychiatry* 1980; 15: 381-388.
58. Madsen AL, Vorstrup S, Rubin P, Larsen JK, Hemmingsen R. Neurological abnormalities in schizophrenic patients: a prospective follow-up study 5 years after first admission. *Acta Psychiatr Scand* 1999; 100:119-125.
59. Marcus J, Hans SL, Lewow E, Wilkinson L, Burack CM. Neurological findings in high-risk children: childhood assessment and 5-year followup. *Schizophrenia Bull* 1985; 11:85-100.
60. Whitty P, Clarke M, Browne S, McTigue O, Kamali M, Feeney L, Lane A, Kinsella A, Waddington JL, Larkin C, O'Callaghan E. Prospective evaluation of neurological soft signs in first-episode schizophrenia in relation to psychopathology: state versus trait phenomena. *Psychol Med* 2003; 33: 1479-1484.
61. Mittal, V.A., Tessner, K.D., McMillan, A.L., Delawalla Z, Trottman H, Walker E. Gesture Behavior in Unmedicated Schizotypal Adolescents. *Journal of Abnormal Psychology*, 2006; 115(2):351-358.
62. Gourion D, Goldberger C, Olie J, Loo H, Krebs MO. Neurological and morphological anomalies and the genetic liability to schizophrenia: a composite phenotype. *Schizophrenia Res* 2004; 67(1):23-31.
63. Browne S, Clarke M, Gervin M, Lane A, Waddington JL, Larkin C, O'Callaghan E. Determinants of neurological dysfunction in first episode schizophrenia. *Psychol Med* 2000; 30(6):1433-1441.
64. Flyckt L, Sydow O, Björkenstedt L. Neurological signs and psychomotor performance in patients with schizophrenia, their relatives and healthy controls. *Psychiatry Res* 1999; 86:113-129.
65. Ho BC, Mola C, Andreasen NC. Cerebellar Dysfunction in Neuroleptic Naïve Schizophrenia Patients. Clinical, Cognitive, and Neuroanatomic Correlates of Cerebellar Neurologic Signs. *Biol Psychiatry* 2004; 55(12):1146-1153.
66. Mohr F, Hubmann W, Albus M, Franz U, Hecht S, Scherer J, Binder J, Sobizack N. Neurological soft signs and neuropsychological performance in patients with first episode schizophrenia. *Psychiatry Research* 2003; 121(1):21-30.
67. Ohaeri JU; Otote DI. Subtypes and factors of schizophrenia in an acutely ill Nigerian sample. *Psychopathology* 2003; 36(4):181-189.
68. Mortimer AM, McKenna PJ, Lund CE, Mannuzza S. Rating of negative symptoms using the High Royds Evaluation of Negativity (HEN) scale. *Br J Psychiatry Suppl*. 1989; (7):89-92.

69. Chen EY, Hui CL, Chan RC, Dunn EL, Miao MY, Yeung WS, Wong CK, Chan WF, Tang WN. A 3-year prospective study of neurological soft signs in first-episode schizophrenia. *Schizophrenia Res* 2005; 75:45-54.

70. Schroder J, Niethammer R, Geider FJ, Reitz C, Binkert M, Jauss M, Sauer H. Neurological soft signs in schizophrenia. *Schizophrenia Res* 1991; 6(1): 25-30.

71. Tucker G J, Silberfarb PM. Neurologic dysfunction in schizophrenia: significance for diagnostic practice. In *Psychiatric Diagnosis: Exploration of Biological Predictors*. H. Akiskal & W. Webb eds. New York: Spektrum. 1978. pp. 453-462.

72. Sanders RD, Keshavan MS, Schooler NR. Neurological examination abnormalities in neuroleptic-naïve patients with first-break schizophrenia: preliminary results. *Am J Psychiatry* 1994; 151:1231-1233.

73. Johnstone EC, Macmillan J F, Frith C D, Benn DK, Crow TJ. Further investigation of the predictors of outcome following first schizophrenic episodes. *Br J Psychiatry* 1990; 157:182-189.

74. Yazici, A.H., Demir, B., Yazici, K.M. and Gogus, A. Neurological soft signs in schizophrenic patients and their nonpsychotic siblings. *Schizophrenia Research*, 2002; 58:241-246.

75. Cantor-Grae E, McNeil TF, Rickler KC, Sjostrom K, Rawlings R, Higgins ES, Hyde TM. Are neurological abnormalities in well discordant monozygotic co-twins of schizophrenic subjects the result of perinatal trauma? *Am J Psychiatry* 1994; 151:1194-1199.

76. Mosher LR, Pollin W, Stabenau JR. Identical twins discordant for schizophrenia. *Arch Gen Psychiatry* 1971; 24:422-430.

77. Niethammer R, Weisbrod M, Schiesser S, Grothe J, Maier S, Peter U, Kaufmann C, Schröder J, Sauer H. Genetic influence on laterality in schizophrenia? A twin study of neurological soft signs. *Am J Psychiatry* 2000; 157:272-274.

78. Egan MF, Hyde TM, Bonomo JB, Mattay VS, Bigelow LB, Goldberg TE, Weinberger DR. Relative risk of neurological signs in siblings of patients with schizophrenia. *Am J Psychiatry* 2001; 158:1827-1834.

79. McCreadie RG, Thara R, Srinivasan TN, Padmavathi R. Spontaneous dyskinesia in first-degree relatives of chronically ill, never-treated people with schizophrenia. *Br J Psychiatry* 2003; 183:45-49.

80. Tarbox SI, Pogue-Geile MF. Spontaneous dyskinesia and familial liability to schizophrenia. *Schizophrenia Res* 2006; 81:125-137.

81. Cannon TD. The inheritance of intermediate phenotypes for schizophrenia. *Current Opinion in Psychiatry* 2005; 18:135-140.

82. Sanders RD, Keshavan MS, Forman SD, Pieri JN, McLaughlin N, Allen DN, van Kammen DP, Goldstein G. Factor structure of neurologic examination abnormalities in unmedicated schizophrenia. *Psychiat Res* 2000; 95:237-243.

83. Flashman LA, Flaum M, Gupta S, Andreasen NC. Soft signs and neuropsychological performance in schizophrenia. *Am J Psychiatry*. 1996; 153:526-532.

84. Shibre T, Kebede D, Alem A, Kebreab S, Melaku Z, Deyassa N, Negash A, Fekadu A, Fekadu D, Medhin G, Negeri C, Jacobsson L, Kullgren G. Neurological soft signs (NSS) in 200 treatment-naïve cases with schizophrenia: a community-based study in a rural setting. *Nord J Psychiatry* 2002; 56(6):425-31.

85. Woods BT, Kinney DK, Yurgelun-Todd D. Neurologic abnormalities in schizophrenic patients and their families. I. Comparison of schizophrenic, bipolar, and substance abuse patients and normal controls. *Arch Gen Psychiatry* 1986; 43(7):657-663.

86. Convit A, Jaeger J, Lin SP, Meisner M, Volavka J. Predicting assaultiveness in psychiatric inpatients: a pilot study. *Hosp. Community Psychiatry* 1988; 39(4):429-434.

87. Chen EY, Shapleske J, Luque R, McKenna PJ, Hodges JR, Calloway SP, Hymas NF, Dening TR, Berrios GE. The Cambridge Neurological Inventory: a clinical instrument for assessment of soft neurological signs in psychiatric patients. *Psychiatry Res* 1995; 56(2):183-204.

88. Jahn T, Cohen R, Hubmann WW, Mohr F, Köhler I, Schlenker R, Niethammer R, Schröder J. The Brief Motor Scale (BMS) for the assessment of motor soft signs in schizophrenic psychoses and other psychiatric disorders. *Psychiatry Res* 2006; 142(2-3): 177-189.

89. Ojagbemi A, Emsley R, Gureje O. Proposing the short Neurological Evaluation Scale Acta Neuropsychiatr 1-8. 2016 Epub 2016 Oct 24. <http://dx.doi.org/10.1017/neu.2016.55>

90. Lawrie SM, Byrne M, Miller P, Hodges A, Clafferty RA, Cunningham Owens DG, Johnstone EC. Neurodevelopmental indices and the development of psychotic symptoms in subjects at high risk of schizophrenia. *Br J Psychiatry* 2001; 178:524-530.

91. Tandon A, Kumari S, Ramiji S, Malik A, Singh S, Nigam VR. Intellectual psycho-education and function status of low birth weight survivors beyond five years of age. *Ind J Pediatrics* 2000; 67: 91-96.

92. Wong AH, Voruganti L, Heslegrave R J, Awad AG. Neurologic abnormalities in schizophrenic patients and their families. I. Comparison of schizophrenic, bipolar, and substance abuse patients and normal controls. *Arch Gen Psychiatry* 1997; 43:657-663.

## Neuropsychological Assessment

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### Abstract

Neuropsychological assessment is perhaps the most important domain in neuropsychology. It allows for an in-depth and comprehensive evaluation of various cognitive domains, development of strategies for remediation and methods of management of neurobehavioral disorders. There has been constant advancement in the tools, techniques, and area under study. Purpose of this article is to discuss issues related to neuropsychological assessment approaches, tools, methodologies etc in this context, outlining the development of tests, role of computer, and limitations. This paper examines the major body of empirical research on neuropsychological assessment. It also provides recommendation for future research.

**Keywords:** Neuropsychological Assessment; Approaches; Reliability Validity.

### Introduction

The discipline of neuropsychology, the applied science concerned with the behavioral expression of brain dysfunction, came from the parent disciplines of Psychology and Neurology. Neuropsychological assessment is a performance-based method to assess cognitive functioning. This method is used to examine the cognitive consequences of brain damage, brain disease, and severe mental illness [1]. The importance of these tests depends on the fact that they often identify abnormality in the absence of positive findings from sophisticated diagnostic techniques like CT or MRI.

#### *Approaches to Neuropsychological Assessment*

There are three common modes of assessments in neuropsychology. They are 1) behavioral neurology, 2) neuropsychological battery approach and 3)

individual centered normative approach [2,3]. The behavioral neurology approach is quantitative, individualized and non-structured. Often it seems time consuming and comparison or generation of main data is often difficult. The battery approach is a structured approach to quantify cognitive functions that can be subjected to both cross sectional and longitudinal comparisons and offers the possibility of generating mass data e.g. Halstead-Reitan Battery (HRB)[4]. The individual centered normative approach is a path between the other two approaches. In clinical neuropsychology these data is often needed on a large number of subjects and frequent comparisons is impracticable in most situations. However, the battery approach is modified to suit the requirements of the disease condition under study. This led to the division such as fixed and flexible approaches in the field of neuropsychological assessment. Fixed or standard batteries have been advocated by many neuropsychologists. Halstead Reitan Neuropsychological Test Battery [4]

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Cambridge Neuropsychological Test Automated Battery (CANTAB) [5] and Luria Nebraska neuropsychological Battery (LNNB) [6] are few known fixed batteries. However, in view of the limitations of these tests, some neuropsychologists are attracted towards flexible batteries [7,8].

#### *Goals of Neuropsychological Assessment*

Goals of any neuropsychological assessment include 1) detection of neuropsychological impairment, 2) monitoring changes and 3) identification of specific disabilities. Ascertaining a clinical diagnosis for a sub threshold level of impairment may be difficult without the help of a detailed neuropsychological assessment. Sometimes neuropsychological assessments are very fruitful in making a differential diagnosis. Once a baseline data obtained which will serve two purposes such as keeping it as a baseline data and the observations obtained from the assessment can be disseminated to the caregiver regarding the strength and weakness of the index patient.

#### *Developments in Neuropsychological Tests*

Though the older tests are in practice widely, an understanding regarding the newer tests and instruments is unavoidable. Here we provide a brief description regarding the newer tests or about revisions that has been made to the older one.

**Intelligence:** The status of Wechsler Adult Intelligence Test (WAIS-III) and its other versions (such as WPPSI-III, WISC-IV) are considered as gold standard measure for intelligence [9-11]. But a comprehensive assessment of intelligence only with the help of WAIS-III has been criticized [12-14], and led to the development of six factor model from the WAIS-III and WMS-III, which includes verbal, perceptual, processing speed, working memory, auditory memory, and visual memory constructs [14]. In order to tackle the time constraints the use of shorter forms of IQ tests such as Wechsler Abbreviated Scale of Intelligence (WASI) are in use [15,16]. Test of Nonverbal Intelligence (TONY) [17] is considered as a substitute for Raven's Progressive Matrices [18]. As the construct of intelligence is difficult to assess in children under the age of two years, Bayley Scale of Infant Development (BSID) is often used to understand the development of children. BSID was revised (BSID-II) by adding new items and dropping old one [19].

**Executive Functioning:** Executive functioning is composed to four functions 1) volition; 2) planning;

3) purposive action and 4) effective performance [8]. The WCST is one of the unavoidable tests in the assessment of executive functioning. Recently a modified abbreviated form of administration by giving only first set of 64 cards, known as WCST-64, has been validated [20,21]. Due to some practical problems in terms of test cards and patient factors Nelson modified the card sorting test (Modified Card Sorting Test) [22]. Though it is a time saving the test it is not comparable to WCST [23,24].

Another important test is Cambridge Neuropsychological Test Automated Battery (CANTAB). It assesses the subject's visual memory, visual attention, working memory, and planning. The test consists of one screening test and 12 principal tests [25]. The test suffers from methodological issues such as inadequate norms and its effects on certain variables such as age, IQ and gender [26,27]. Eclipse 6 is the latest version of Cambridge Cognition's leading cognitive testing platform for academic research, used to assess cognitive functioning over 30 CNS disorders such as Alzheimer's disease, Parkinson's disease, ADHD, schizophrenia and depression and cited in over 1200 peer-reviewed publications.

**Attention and Concentration:** Comprehensive Trail Making Test is used to measure attention, visual scanning and executive functions, developed primarily to address the shortcomings of original TMT as it was lacking proper norms and increased sensitivity to detection of separate cognitive components [28]. Another test Paced Auditory Serial Addition Test (PASAT) and its children versions (CHIPASAT) measures working memory, divided attention and information processing speed. Children's version is also available as computer software. Trail Making Test can also be used to measure attention speed and mental flexibility. An oral version of the TMT is recommended for people having problems in drawing [29,31].

**Memory:** California Verbal Learning Test (CVLT) is used to measure verbal learning and memory which was developed according to process model [32]. Its revised form CVLT assesses both recall and recognition of two word lists over immediate and delayed memory trials. It has got children's version too known as CVLT-C. The test Doors and People was developed to assess visual and verbal episodic memory, including both recall and recognition [34]. Rey Auditory Verbal Learning Test (RAVLT) also known as Auditory Verbal Learning Test (AVLT) is used to measure learning and memory. Recently it is translated to various foreign languages [35]. The revised form of Rivermead Behavioral Memory Test

was originally designed to detect impairment in every day memory functioning due to neurological impairment [36-38]. A lot of other tests also developed or modified from its older version like WMS-III, [14] and Ruff Light Trail Learning Test (RULIT) [39].

In conditions like mild cognitive impairment (MCI) Clinical Dementia Rating (CDR) [40] and Dementia Rating Scale (DRS) [41] can be administered. Among these tests the subjects with MCI may get a score of 3 on GDS and 0.5 on CDR [42]. Where as in conditions like traumatic brain injury (TBI) scales like Galvenston Orientation and Amnesia Test are considered to be the right to begin with after detailed case history. There is a dearth of indigenously developed neuropsychological test battery that is suitable for Indian older adults to assess cognitive function as well as decline, [43] and the need for developing and validating cognitive measures has been highlighted by several researchers [44]. NIMHANS Neuropsychological Battery for Elderly (NNB-E) has been developed and standardized on Indian population[45]. Hindi mental-status examination (HMSE) is a modified version of MMSE and is validated for Indian population [46].

#### *Use of Computers in Neuropsychological Assessment*

Computer assisted psychological examinations are increasingly applied in health care. A major advantage of use of computers in the assessment are it is less time consuming and potentially more cost effective, once it is standardized it assures reliability and validity. In addition to these it enables precise time, quality monitoring and analysis of results. Though in many cases, validity and reliability of many tests remains a matter of controversy and more researches are needed. The following are some of the tests that available as computer programs or software: The Auditory Verbal Learning Test, The Five Point Test, Trail Making Test (TMT), The Computer Assisted Stroop Test, Complex Figure, The Poreh Picture Naming Test, The Controlled Oral Word Association Test (COWA), The Cleveland Card Sorting Sorting tests, The QPSS Continuous Performance Test.

#### *Factors Affecting the Neuropsychological Assessment*

**Culture-** Ethnicity and cultural background are important factors in interpreting test performance. [47-48] Minority members may score lower on some tasks due to unfamiliarity with test-taking and dissimilarities in cultural experiences and expectations.

**Education-** Education has a marked effect on neuropsychological test performance. For example, the cutoff for "normal" performance on the Mini-Mental State Examination may vary by as much as eight points depending on the individual's educational level[49]. The clock-drawing test, widely used in clinical screening, is also affected by low education. Education-specific normative data are not available for most tests, and interpretation of test results in individuals with unusually high or low levels of education must be done with caution. It has an important influence on cognitive test performance[50,51] An effect of education on non-verbal tests has also been reported in children [52].

**Gender -** Gender has consistent but minor effects on neuropsychological assessment. Women tend to perform better on tests of verbal memory than men[53] and men evidence more decline than women on most neuropsychological tests in the course of normal aging [54] Gender effects are of modest magnitude compared with the influences of age and education on neuropsychological test performance [55].

**Age-** Aging affects several domains of neuropsychological function, including fluid intellectual abilities, complex attentional processes, some aspects of memory, psychomotor speed, accessing word knowledge, visuospatial skills, some forms of abstract reasoning, and complex problem-solving [56,57]. Age stratified norms exist for most widely used instruments, although patients in the extremes of old age (above age 75) are often underrepresented in the normative sample, making interpretation of test results in very old patients more difficult.

**Psychiatric disorders-** Neuropsychiatric disorders like anxiety, depression, psychosis, apathy, and irritability all have an impact on the patient's ability to cooperate with testing and may directly affect cognition. Anxiety and depression impair performance on effort-demanding tests and have less effect on tests of over-learned skills. Memory complaints are common manifestations of depressive disorders, and severe depression is commonly accompanied by psychomotor slowing, impaired attention, decreased cognitive flexibility, and poor retrieval memory [58].

**Substance abuse-** Chronic alcoholism is associated with deterioration in abstraction, visuospatial skills, and problem-solving abilities [59]. A history of excessive substance use must be sought and integrated the interpretation of neuropsychological test data. This is particularly important in the evaluation of patients with histories of head trauma because trauma is more common among those with

substance abuse [60].

*Flynn effect-* Performance on mental ability tests rising from one generation to the next is called "Flynn effect". The phenomenon has important implications for clinical utilization of IQ tests [61]. Flynn effect does apply to the Wechsler and Stanford-Binet tests, as well as tests such as the Otis Intermediate Test of Mental Ability and the California Test of Mental Maturity [62]. Not only does the Flynn effect causes published norms for Full Scale IQ to become progressively less appropriate over time, but it also causes different subtest norms to change at different rates. Clinical neuropsychologists who use old versions of IQ tests not only will overestimate IQ but also will risk misinterpreting indicators such as Verbal-Performance disparities and subtest profiles [61].

*Neuropsychological Methods Vs Other Methods of Assessment: A Co-relational View:*

Neuropsychology and neuroimaging both provide information about the relationship between brain structure and function, and thus attempts to understand if the neural basis of cognition should benefit from converging results obtained across the two methods. However, serious attempts to integrate the two methodologies face several challenges, such as differences in basic paradigm designs [63]. Many attempts have been made to evaluate the benefits that can be derived from in-depth comparisons of neuroimaging or functional neuroimaging and neuropsychology, or focusing on the strengths and weaknesses of the two methods [64]. Studies conducted with this type of interdisciplinary objective in mind are likely to be extremely valuable, because they should provide new constraints that motivate revisions and refinement to theoretical models of human cognition in neuropsychological assessment using event-related brain potentials [65].

*Electrophysiological Methods:* Two studies examined ERP responses to the computerized version of the Peabody Picture Vocabulary Test - Revised (PPVT-R) [66] in 10-year-old children [67] and young adults [68] and concluded that the results from the PPVT-R supported the use of ERPs in the assessment of receptive vocabulary levels. Historically, clinicians have utilized evoked potentials for evaluating sensory functions and neuropsychological testing for evaluating cognitive functions, however, the clinical implementation of it remains to be developed fully. Language functions have also been assessed using neuropsychological tests that are formatted for computer presentation with simultaneous ERP

recordings and significantly correlating the two measures [69].

*Neuroimaging Methods:* Neuropsychological testing and structural imaging with X-ray computed tomography (CT) or magnetic resonance imaging (MRI) in major depression shows evidence of slowing in motor and cognitive domains with additional prominent effects on mnemonic function most marked in the elderly [70]. Recently, functional neuroimaging techniques, such as PET [71], fMRI [72], and Magnetoencephalography (MEG) [73], have provided a more direct link between cerebral and cognitive aging [74]. Studies of cognitive recovery after acquired brain damage in adults using functional neuroimaging and neuropsychological tests found males performing better on block design and visual reproduction, and females performing better on the California Verbal Learning Test (CVLT). Across both sexes, block design scores correlated significantly with right hemisphere M100 location, with more anterior source locations associated with better performance. CVLT scores were negatively correlated with right hemisphere M100 source locations, suggesting that MEG-based measures of interhemispheric asymmetry may be related to specific neuropsychological test performance measures [75]. Comparative study of the neuropsychological and neuroimaging evaluations in children with dyslexia indicated significant correlation between the two measures [76]. In yet another study, neuropsychological test battery sensitive to fronto-striatal system dysfunction and CT scans were applied on patients with schizophrenia indicating structural frontal abnormalities in them [77].

*Methodological Considerations of Neuropsychological Assessment*

Low reliability not only limits the sensitivity of any individual test when it is used on its own for clinical diagnosis or as a research tools but also sets limits to the extent to which it can be expected to correlate with other measures and its sensitivity when it is repeatedly administered to detect changes in ability [78]. Many of tests in both Cambridge Neuropsychological Test Automated Battery (CANTAB) and the International Study of Post-Operative Cognitive Dysfunction (ISPOCD) batteries seem to have test/re-test correlations that are unacceptably low for use as research tools [78]. In a review of eight common neuropsychological instruments, most measures showed significant

practice effects [79]. The instruments were Trail Making Test, Stroop Color/Word Interference Test, Grooved Pegboard, California Computerized Assessment Package (CalCAP), Digit Span, Symbol Digit Modalities Test, Rey-Osterrieth Complex Figure Test (RCFT), Controlled Oral Word Association Test (COWAT). However, small effect sizes on some measures raises doubt on their clinical significance. The greatest effect sizes were found on the RCFT recall trials. Most other measures that had significant changes had effect sizes within the .20 to .32 range. The measures that failed to show significant practice effects, including the direct copy of the RCFT Copy trial, Word Reading trial of the Stroop, and the CalCAP Choice Reaction Time 1 trial. All of the measures that required some cognitive engagement such as memory, strategy, speeded processing, or divided attention showed some practice effects, while the relatively straightforward overlearned tasks did not. In sum, practice effects appeared to occur on motor, psychomotor, attention, processing speed, nonverbal memory, and executive functioning measures. Test-retest correlations were generally good in these instruments. In contrast to the above, another study [80] reported better reliability with Grooved Pegboard as their sample was somewhat more heterogeneous. It has been observed that sensitivity to diffuse neuropsychological changes and ease/speed of administration make the Rey Auditory Verbal Test a valuable tool in medical rehabilitation settings [81]. Cancellation task have traditionally been used to measure visuospatial neglect but recently, such tests have also been viewed as potential measures of executive function with good convergent validity for all three executive organization measures (mean inter-target distance, path intersection rate, and a quantification of overall path uniformity) [82]. The Brown Location Test (BLT) designed to specifically measure visual memory for location of identical objects (dots) has good internal and alternate form reliabilities [83]. Factor analysis of a brief test battery confirmed that BLT performance is generally independent of verbal memory and global intellectual abilities. BLT performance declined with age, but there was no association between performance and gender, education, or intellectual functioning. In view of the favorable psychometric properties observed during preliminary studies, additional normative and validation studies in healthy and patient populations are warranted.

In the future, neuropsychological assessment, neuroimaging and electrophysiological methods should be used in convergence in designing rehabilitation programs and in monitoring the effectiveness of such programs, assessing outcomes

and the success of rehabilitation treatments. New guidelines, Specific training are needed as in disability; ecological validity of tests; academic programs for students with disabilities; specific cognitive-behavioral interventions, government assistance programs for individuals with disabilities.

## Conclusion

Neuropsychology is one of those avenues where the contribution from the mental health professional is pivotal. Understanding and updating information is unavoidable to a psychologist to excel .and this ability to adapt to changes in techniques, ideas, and patients served accomplishes its potential to be of service to humanity.

## References

1. Harvey PD. Clinical applications of neuropsychological assessment. *Dialogues in Clinical Neuroscience* 2012; 14(1): 91-9.
2. Beaumont JG. *Introduction to Neuropsychology*. 2nd edition. New York: The Guilford Press;2008.
3. Walsh KW. *Understanding Brain Damage*. 2nd ed. Edinburgh: Churchill Livingston; 1991.
4. Reitan RM, Wolfson D. *The Halstead-Reitan Neuropsychological Test Battery*.2nd ed. Tucson, AZ: Neuropsychology Press;1993.
5. Robbins TW, James M, Owen AM, Sahakian BJ, McInnes L, Rabbitt P. Cambridge Neuropsychological Test Automated Battery (CANTAB): A Factor Analytic Study of a Large Sample of Normal Elderly Volunteers. *Dement Geriatr Cogn Disord* 1994; 5:266-81.
6. Devaraju-Backhaus S, Espe-Pfeifer P, Mahrou M L, Golden C J. Correlation of the LNNB-III with the Wais-III in a Mixed Psychiatric and Brain-Injured Population. *International Journal of Neuroscience*. 2001;111 (3-4): 235-40.
7. Benton AL, Sivan AB, Hamsher KD, Varney NR, Spreen O. *Contributions to neuropsychological assessment: a clinical manual*. 2nd ed. New York: Oxford University Press;1994.
8. Lezak, M.D., Howieson, D.B., Bigler, E.D., & Tranel, D. *Neuropsychological Assessment*, Fifth Edition. New York: Oxford University Press; 2012.
9. Ivnik R, Malec JF, Smith GE, Tangalos EG, Petersen RC, Kokmen E, Kurkland LT. Mayo's Older American Normative Studies : WAIS-R norms for ages 56 to 97. *Clinical Neuropsychologist*, 1992; 6 Suppl:1-30.

10. Camara W J, Nathan JS, Puente A E. Psychological test usage: Implication in professional psychology: Research and Practice 2000; 31:141-54.
11. Rabin LA, Barr WB, Burton LA. Assessment practices of clinical neuropsychologist in United States and Canada: A survey of INS, NAN, and APA Division 40 members . Archives of Clinical Neuropsychology. 2005; 20(1):33-65.
12. Flanagan D P, McGrew KS, Ortiz S O. The Wechsler Intelligence Scales and Gf- Gc theory: A contemporary approach to interpretation. Boston: McGraw-Hill; 2000.
13. Larrabee GJ. A review of clinical interpretation of WAIS-III and WMS-III: Where do we go from here and should we do with WAIS-IV and WMS-IV? J Clin Exptl Neuropsychol 2004; 24:707-17.
14. Tulsky DS, Saklofske DS, Ricker J. Historical overview of intelligence and memory: Factor influencing Wechsler scales. In D.S. Tulsky, D.H Saklofske, GJ Chelune (eds) Clinical interpretation of WAIS III and WMS III. San Diego: Academic Press; 2003. p7-41.
15. Holdnack HA. Wechsler Test of Adult Reading Manual. San Antonio, TX: The Psychological Corporation;2001.
16. Canivez GL, Konold T R, Collins J M, Wilson G. Construct validity of the Wechsler Abbreviated Scale of Intelligence and Wide Range Intelligence Test: Convergent and structural validity. School Psychology Quarterly 2009; 24(4): 252-65.
17. Brown DJ, Kerr S, Wilson JR. Virtual environments in special-needs education. Comm ACM 1997; 40(8): 72-5.
18. Raven J, Raven J. Raven Progressive Matrices. Handbook of Nonverbal Assessment R. Steve McCallum (ed) Kluwer Academic/Plenum Publishers, New York; 2003. p223-37.
19. Bayley N. Bayley Scales of Infant Development 3rd edition (Bayley III). San Antonio: Harcourt Assessment Inc; 2006.
20. Greve KW. The WCST-64: a standardized short-form of the Wisconsin Card Sorting Test. Clin Neuropsychol. 2001;15(2):228-34.
21. Greve K W, Stickle T R, Love J M, Bianchini K J, Stanford MS. Latent structure of the Wisconsin Card Sorting Test: A confirmatory factor analytic study. Archives of Clinical Neuropsychology, 2005; 20(3): 355-64.
22. Nelson HE. A modified card sorting test sensitive to frontal lobe defects. Cortex 1976; 12(4): 313-24.
23. De Zubizaray G, Ashton R. Nelson's Modified Cards Sorting Test: A review. The Clinical Neuropsychologist 1996;10:245-54.
24. Van Gorp WG, Kalechstein AD, Moore LH, Hinkin CH, Mahler ME, et al. A clinical comparison of two forms of the cards sorting test. The Clinical Neuropsychologist 1997; 11:155-60.
25. Fray PJ, Robins TW, Sahakian B J. Neuropsychiatric application of CANTAB. Int J Geriatr Psychiatry 1996; 11: 329-36.
26. De Luca CR, Wood SJ, Anderson V, Buchanan JA, Proffitt TM, Mahony K et al. Normative data from the CANTAB I. Development of executive function over the life span. J Clin Exp Neuropsychol 2003;25:242-54.
27. Luciana N, Nelson CA. Assessment of Neuropsychological functioning through use of the Cambridge Neuropsychological Testing Automated Battery; Performance in 4- to 12 - years-old children. Dev Neuropsychol 2002 ; 22 : 595-624.
28. Reynolds CR. Comprehensive Trail making Test. Austin, TX:PRO-ET Inc; 2002.
29. Abraham E, Axelrod BN, Ricker JH. Application of the oral Trail Making Test to a mix clinical sample. Arch Clin Neuropsychol 1996; 11:697-701.
30. Ricker JH, Axelrod BN. Analysis of an oral paradigm for the Trail Making Test. Assessment 1994; 1:47-51.
31. Ricker JH, Axelrod BN, Houtler BD. Clinical validation of the oral Trail Making Test. Neuropsychiatry Neuropsychol Behav Neurol 1996; 9: 50-3.
32. Delis DC, Kramer JH, Kaplan E, Ober BA. The California Verbal Learning Test. San Antonio: the Psychological Corporation; 1987.
33. Delis DC, Kramer JH, Kaplan E, Ober BA. California Verbal Learning Test-Second Edition, Adult Version. San Antonio Tex: The Psychological Corporation; 2000.
34. Baddeley A, Emslie H, Nimmo-Smith I. Doors and People. Bury St. Edmund, England: Thames Valley Test Company; 1994.
35. Lee TMC. Normative data: Neuropsychological measures for Hong Kong Chinese. Neuropsychology Laboratory. Hong Kong :The University of Hong Kong; 2003.
36. Wilson B, Cockburn J, Baddeley A. The Rivermead Behavioural Memory Test, 2nd ed. Suffolk:Thames Valley Test Company; 1991.
37. Cockburn J, Keene J. Are changes in everyday memory over time in autopsy-confirmed Alzheimer's disease related changes in reported behavior? Neuropsychol rehabil 2001;11: 201-71.
38. Wilson BA. Memory Rehabilitation: Integrating Theory and Practice. New York:Guilford Press; 2009.p231-73.
39. Strauss E, Sherman EMS, Spreen O. A Compendium of Neuropsychological Tests: Administration, Norms, and Commentary. New York; Oxford University Press; 2006.p 851-4.
40. Morris JC. The Clinical Dementia Rating (CDR): current version and scoring rules. Neurology 1993;

43(11):2412-4.

41. Strauss E, Sherman EMS, Spreen O. *A Compendium of Neuropsychological Tests: Administration, Norms, and Commentary*. New York: Oxford University Press; 2006. p144-58.
42. Petersen RC, Smith GE, Waring SC, Ivnik RJ, Tangalos EG, Kokmen E. Mild cognitive impairment: clinical characterization and outcome. *Arch Neurol* 1999;56(3):303-8.
43. Shaji KS, Jotheeswaran AT, Girish N, Bharath S, Dias A, Pattabiraman M, Varghese M. The Dementia India Report: prevalence, impact, costs and services for Dementia. New Delhi: ARDSI; 2010.
44. Chan AS, Shum D, Cheung RW. Recent development of cognitive and neuropsychological assessment in Asian countries. *Psychol Assess* 2003; 15: 257-67.
45. Tripathi R. Development and standardization of neuropsychological test battery for older adults. Unpublished Ph.D. Thesis, Department of Clinical Psychology. Bangalore: NIMHANS (Deemed University); 2012.
46. Ganguli M, Ratcliff G, Chandra V, Sharma S, Gilby J, Pandav R, et al. A Hindi version of the MMSE: The development of a cognitive screening instrument for a largely illiterate rural elderly population in India. *Int J Geriatr Psychiatry* 1995;10:367-77.
47. Oberg G, Ramírez M. Cross-linguistic meta-analysis of phonological fluency: Normal performance across cultures. *Int J Psychol* 2006; 41 (5): 342-7.
48. Rosselli M, Ardila A. The impact of culture and education on non-verbal neuropsychological measurements: A critical review. *Brain Cogn* 2003; 52: 326-33.
49. Crum RM, Anthony JC, Bassett SS, Folstein MF. Population-based norms for the Mini-Mental State Examination by age and educational level. *JAMA* 1993; 269: 2386-3291.
50. Eviatar Z. Culture and brain organization. *Brain Cogn* 2000; 42: 50-2.
51. Solís OF, Lozano A. Digit Span: Effect of education and culture. *Int J Psychol* 2006; 41(5) 333 - 41.
52. Klenberg L, Korkman M, Lahti-Nuutila P. Differential development of attention and executive functions in 3- to 12 year old finnish children. *Dev Neuropsychol* 2001; 20: 330-407.
53. Delis D, Kramer J, Kaplan E, Ober B. *California Verbal Learning Test*. San Antonio, TX: The Psychological Corporation; 1987.
54. Ganguli M, Ratcliff G, Huff J, Belle S, Kancel MJ, Fischer L, et al. Effects of age, gender, and education on cognitive tests in a rural elderly community sample: norms for the Monongahela Valley Independent Elders Survey. *Neuroepidemiology* 1991; 10:42-52.
55. Niemeier JP, Marwitz JH, Lesser K, Walker WC, Bushnik T. Gender differences in executive functions following traumatic brain injury. *Neuropsychol Rehabil* 2007; 17(3):293-313.
56. Raymond DP, Radel M, Ray MJ, Hinton-Bayre AD, Marsh NA. Investigation of factors relating to neuropsychological change following cardiac surgery. *Perfusion* 2007; 22(1), 27-33.
57. Rabbitt P M A, McInnes L, Diggle P. The University of Manchester Longitudinal study of cognition in normal healthy old age, 1983 through 2003. *Aging Neuropsychol C* 2004;11: 245-79.
58. Nelson DV, Harper RG, Kotik-Harper D, Kirby HB. Brief neuropsychologic differentiation of demented versus depressed elderly patients. *Gen Hosp Psychiatry* 1993; 15:409-16.
59. Parsons OA. Neuropsychological consequences of alcohol abuse: many questions—some answers. In: Parsons OA, Butters N, Nathan PE, eds. *Neuropsychology of alcoholism: implications for diagnosis and treatment*. New York: The Guilford Press;1987. p153-75.
60. Mearns J, Lees-Haley PR. Discriminating neuropsychological sequelae of head injury from alcohol-abuse-induced deficits: a review and analysis. *J Clin Psychol* 1993;49:714-20.
61. Hiscock M. The Flynn effect and its relevance to Neuropsychology. *J Clin Exp Neuropsychol* 2007; 29 (5),514-29.
62. Flynn JR. Massive IQ gains in 14 nations: What IQ tests really measure. *Psychol Bull* 1987; 101:171-91.
63. Fiez JA. Bridging the Gap Between Neuroimaging and Neuropsychology: Using Working Memory as a Case-Study. *J Clin Exp Neuropsychol* 2001; 23(1): 19-31.
64. Kim SG, Ugurbil K. Functional magnetic resonance imaging of the human brain. *J Neurosci Methods* 1997;74: 229-43.
65. Connolly JF, D'Arcy RCN. Innovations in neuropsychological assessment using event-related brain potentials: *Int J Psychophysiol* 1999;37: 31-47.
66. Strauss E, Sherman EMS, Spreen O. *A Compendium of Neuropsychological Tests: Administration, Norms, and Commentary*. New York: Oxford University Press; 2006. 467-71.
67. Byrne JM, Dywan C, Connolly JF. Assessment of children's receptive vocabulary using brain event-related potentials: development of a clinically valid test. *Child Neuropsychol* 1995; 1: 211-23.
68. Connolly JF, Byrne JM, Dywan CA. Eventrelated brain potentials reflect the receptive vocabulary of individuals as measured by the Peabody Picture Vocabulary Test Revised: a study of crossmodal and cross-form priming. *J Clin Exp Neuropsychol* 1995; 17: 548-65.
69. Connolly JF, D'Arcy RCN. International Innovations in neuropsychological assessment using event-

related brain potentials. *J Psychophysiol* 2000; 37: 31-47.

70. Goodwin GM. Neuropsychological and Neuroimaging Evidence for the Involvement of the Frontal Lobes in Depression. *J Psychopharmacol* 1997; 11(2):115-22.

71. Anderson ND, Iidaka T, Cabeza R, Kapur S, McIntosh A R, Craik FI. The effects of divided attention on encoding and retrieval-related brain activity: A PET study of younger and older adults. *J Cognitive Neurosci* 2000; 12(5):775-92.

72. Daselaar SM, Veltman DJ, Rombouts SA, Raaijmakers JG, Jonker C. Neuroanatomical correlates of episodic encoding and retrieval in young and elderly subjects. *Brain* 2003; 126(1): 43-56.

73. Maestú F, Fernández A, Simos PG, Gil-Gregorio P, Amo C, Rodriguez R, Arrazola J, Ortiz T. Spatiotemporal patterns of brain magnetic activity during a memory task in Alzheimer's disease. *NeuroReport* 2001; 12(18):3917-22.

74. Munoz-Cespedes J, Rios-Lago M, Paul N, Maestu F. Functional Neuroimaging Studies of Cognitive Recovery After Acquired Brain Damage in Adults: *Neuropsychol Rev* 2005; 15 (4): 169-83.

75. Reite M, Cullum CM, Stocker J, Teale P, Kozora E. Neuropsychological test performance and MEGbased brain lateralization: Sex differences. *Brain Res Bull* 1993; 32(3): 325-8.

76. Arduni RG, Capellini SA, Ciasca SM. Comparative study of the neuropsychological and neuroimaging evaluations in children with dyslexia. *Arq Neuro-Psiquiatr* 2006; 64, 369-75.

77. Torre JCS, Barrios M, Junqué C. Frontal lobe alterations in schizophrenia Neuroimaging and neuropsychological findings. *Eur Arch Psychiatry Clin Neurosci* 2005; 255: 236-24.

78. Lowe C, Rabbitt P. Test/re-test reliability of the CANTAB and ISPOCD neuropsychological batteries- theoretical and practical issues. *Neuropsychologia* 1998; 36(9):915-23.

79. Levine AJ, Miller EN, Becker JT, Selnnes OA, Cohen BA. Normative Data for Determining Significance of Test-Retest Differences on Eight Common Neuropsychological Instruments. *Clin Neuropsychol* 2004; 18: 373-84.

80. Dikmen S, Heaton R, Grant I, Temkin N. Test-retest reliability and practice effects of Expanded Halstead-Reitan Neuropsychological Test Battery: *J Int Neuropsychol Soc* 1996; 5: 346-56.

81. Callahan CD, Johnstone B. The clinical utility of the Rey Auditory-Verbal Learning Test in medical rehabilitation. *J Clin Psychol Med Settings* 2005; 1(3): 261-8.

82. Woods AJ, Mark VW. Convergent validity of executive organization measures on cancellation: *J Clin Exp Neuropsychol* 2007; 29 (7): 719-23.

83. Brown FC, Roth RM, Saykin AJ, Gibson G B. A New Measure of Visual Location Learning and Memory: Development and Psychometric Properties for the Brown Location Test (BLT). *Clin Neuropsychol* 2007; 21 (5): 811 - 25.

## Non-Suicidal Self Injury and Suicidal Behaviour: From Continuum to Dichotomy

Hemendra Singh

### Abstract

The term non-suicidal self injury (NSSI) and suicidal behaviour are used under a continuum of self harm. NSSI, suicide attempts and suicide are distinct behaviours. NSSI may be considered to be prevalent along this continuum in a place of lesser severity than suicide attempts. NSSI has been included in the *Diagnostic and Statistical Manual of Mental Disorders*- as a condition that requires further examination. Frequency and history of NSSI has correlated with future suicide attempts. While assessing for suicidal risk, history of NSSI and past suicide attempt should also be considered for current and future suicide risk.

**Keywords:** Non-Suicidal Self Injury (NSSI); Suicidal Behaviour; Self Harm.

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Worldwide, suicide is a major cause for concern. According to the WHO: every year, almost one million people die from suicide; a "global" mortality rate of 16 per 100,000, or one death every 40 seconds; in the last 45 years suicide rates have increased by 60% worldwide. Suicide is among the three leading causes of death among those aged 15–44 years in some countries, and the second leading cause of death in the 10–24 years age group; these figures do not include suicide attempts which are up to 20 times more frequent than completed suicide [1].

Research suggests that more than 90% of suicide victims and attempters had at least one current Axis I (mainly untreated) major mental disorder, most frequently major depressive episode (MDE) (56–87%), substance use disorders (26–55%), and schizophrenia (6–13%). Comorbid anxiety and personality disorders and concomitant serious medical disorders are also frequently found, although they are rarely the principal or the only diagnoses [2–6]. However, there are other cases of suicide, who do not meet any criteria of any underlying psychiatric

illnesses. Many times due to genetic vulnerability an individual may develop either suicidal behavior or psychotic breakdown or both may co-occur at a same time.

Suicidal behaviour also meets characteristics diagnostic criteria of like any other disorder, and it had also been argued to be included in classificatory system [7–10]. However, several concerns have also been raised from researchers regarding inclusion of suicidal behaviour as a diagnosis. Such as it could be a symptom of an underlying illness, it may lead to "Medicalization" of behaviours, and it may increase liability for mental health professionals [10].

However, Non – Suicidal self injury (NSSI) has been included in the *Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5)* as a condition that requires further examination [11]. NSSI is defined as intentional damage of one's body tissue without clear suicidal intent, and usually performed to seek immediate relief of psychic distress [12].

Initially NSSI was considered as a symptom of

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Borderline Personality Disorder (BPD), however, current literature supports that NSSI is not a symptom of BPD but it is also seen in individuals who are not diagnosed with BPD and have other Axis I disorders [13].

NSSI and, suicide attempts and suicide are distinct behaviours [14]. NSSI may be considered to be prevalent along a continuum of self-harm in a place of lesser severity than suicide attempts [15]. The most important distinction between NSSI and suicide is that NSSI is intended to injure the body without causing death [16]. Self-injurious behaviour is often resorted to as a means of avoiding suicide [14]. Douglas et al. (2004) suggest that NSSI may at best be taken as a symptom of distress which may lead to suicide if not overcome successfully, since approximately 60% of individuals with a history of self-injury report that they were not considering suicide [14].

Introducing NSSI as a distinct diagnostic category appears as a welcome move in current psychiatric practice. Researchers have found a link between self-injurious behaviour and suicidal ideation and suicide attempts in the future [17-20]. Research suggests that as compared to individuals without a history of non-suicidal self-injury, individuals with a history of non-suicidal self-injury were over nine times more likely to report suicide attempts; seven times more likely to report a suicide gesture; and, nearly six times more likely to report a suicide plan [21]. In patients with psychiatric illnesses, frequency of NSSI was also significantly correlated with number of suicide attempts. Hence, It is clinically relevant to screen an Individual for presence of NSSI in past [22,23].

Many times an individual with NSSI may also have suicide ideations and suicide attempts. Risk factors and methods used for NSSI and suicidal behaviors may not be exclusive. Hence, one can also conceptualize the continuum of suicidal behavior as a new term "**Self Harm Spectrum Disorder (SHSD)**" with various dimensions where NSSI with individual risk factors may be considered as mild form, NSSI with Suicidal ideations with other risk factors as moderate form and suicide attempts due to various risk factors including NSSI as a severe form of SHSD. This term might reduce the stigma attached to suicidal behaviour.

However, one may argue the fact that many suicide attempters might not have history of NSSI. Hence it is clinically relevant while assessing for suicide one should not rely only just presence of NSSI, other multiple factors should be taken into risk assessment and risk formulation. One should be

aware that SHSD of any types, from NSSI, NSSI with suicide ideations or suicide attempts, should be carefully evaluated for current suicide risk or risk for future suicide attempts.

## References

1. WHO: Mental health: suicide prevention (SUPRE). [www.who.int](http://www.who.int).
2. Rihmer Z. Suicide risk in mood disorders. *Curr. Opin. Psychiatry* 2007; 20(1):17-22.
3. Beautrais AL, Joyce PR, Mulder RT, Fergusson DM, Deavoll BJ, Nightingale SK. Prevalence and comorbidity of mental disorders in persons making serious suicide attempts: a case-control study. *Am J Psychiatry* 1996; 153(8):1009-14.
4. Hawton K, Fagg J, Simkin S, Bale E, Bond A. Deliberate self-harm in Oxford, 1985-1995. *J Adolesc* 2000; 23(1):47-55.
5. Rihmer Z, Belso N, Kiss K. Strategies for suicide prevention. *Curr Opin Psychiatry* 2002; 15(1):83-87.
6. Rihmer A, Rihmer Z, Jekkel E, K' arteszi M, Csisze'r N, Farkas A. Psychiatric characteristics of 100 nonviolent suicide attempters in Hungary. *Int J Psychiatry Clin Pract* 2006; 10(1):69-72.
7. Robins E, Guze SB. Establishment of Diagnostic validity in psychiatric illness: its application to schizophrenia. *AM J Psychiatry* 1970; 126:983-7.
8. Oquendo MA, Bacca-Garcia E, Mann JJ et al. Issues for DSM-V: Suicidal behaviour as a separate diagnosis on a separate axis. *Am J Psychiatry* 2008; 13: 83-4.
9. Regier DA, Kuhl EA, Kupfer DJ. The DSM-5: classification and criteria changes. *World Psychiatry* 2013; 12:92-8.
10. Oquendo MA, Bacca-Garcia E. Suicidal Behaviour disorder as a Diagnostic entity in DSM - 5 classificatory system: advantages outweigh limitations. *World Psychiatry* 2014; 13(2):128-30.
11. American Psychiatry Association. Diagnostic and Statistical manual of mental disorders fifth edition (DSSM-5). American Psychiatry Association; 2013, p.783.
12. Klonsky ED, Muehlenkamp JJ, Lewis SP, et al. Nonsuicidal Self Injury. Cambridge, MA: Hogrefe Publishing 2011.
13. Klonsky ED. The functions of deliberate self-injury: A review of the evidence. *Clinical Psychology Review* 2007; 27:226-239.
14. Douglas J, Cooper J, Amos T, Webb R, Guthrie E, Appleby L. "Near-fatal" deliberate self-harm: characteristics, prevention and implications for the prevention of suicide, *J Affect Disord* 2004; 79(1): 263-68.

15. Brausch AM, Gutierrez PM. Differences in non-suicidal self-injury and suicide attempts in adolescents. *J Youth Adolesc* 2010; 39(3):233-42.
16. Nock MK, Mendes WB. Physiological arousal, distress tolerance, and social problem solving deficits among adolescent self-injurers. *J Consult Clin Psychol* 2008; 76(1):28-38.
17. Whitlock JL, Knox K. The relationship between suicide and self-injury in a young adult population. *Arch Pediatr Adolesc Med* 2007; 161(7):634-40.
18. Brown MZ, Comtois KA, Linehan MM. Reasons for suicide attempts and nonsuicidal self-injury in women with borderline personality disorder. *J Abnorm Psychol* 2002; 111(1):198-202.
19. Muehlenkamp, J.J., Gutierrez, P.M. Risk for suicide attempts among adolescents who engage in non-suicidal self-injury. *Arch Suicide Res* 2007; 11(1): 69-82.
20. Nock MK, Joiner TE, Gordon KH, Lloyd-Richardson E, Prinstein MJ. Non-suicidal self-injury among adolescents: diagnostic correlates and relation to suicide attempts. *Psychiatry Res* 2006; 144(1):65-72.
21. Whitlock JL, Eckenrode JE, Silverman D. Self-injurious behavior in a college population. *Pediatrics* 2006; 117(6):1939-48.
22. Andover MS & Gibb BE. Non-suicidal self-injury, attempted suicide, and suicidal intent among psychiatric inpatients. *Psychiatry Research* 2010; 178: 101-05.
23. Singh H, Reddi VSK, Chandra PS. Association of Non-Suicidal Self Injury and Suicide Attempts in Psychiatric Inpatients with High Suicidal Risk. *Suicidol Online* 2016; 7:51-57.

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## Who am I? A Case of Gender Dysphoria

**Kushal Tamuli<sup>1</sup>, Soumik Sengupta<sup>1</sup>, Hemanta Dutta<sup>2</sup>, Baruah Aparajita<sup>3</sup>**

### **Abstract**

The field of sex and gender is extremely controversial and has contributed to a proliferation of terms whose meanings vary over time and within and between studies. The term "gender identity," distinct from the term "sexual orientation," refers to a person's innate, deeply felt psychological identification as a man, woman or some other gender, which may or may not correspond to the sex assigned to them at birth [1]. Stein 1999 stated that, "Someone who has the feeling that they are a different sex than the one that they were born - to some extent" [2]. "Sexual orientation" refers to an individual's physical and/or emotional attraction to the same and/or opposite gender [1].

**Keywords:** Gender Dysphoria; DSM5; Psychotherapy.

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### **Introduction**

Gender dysphoria is a psychological diagnosis recognized by the American Psychiatric Association (APA). This disorder is marked by clinically significant distress caused by a marked difference between the individual's expressed/experienced gender and the gender others would assign him or her. In 2012, the APA announced that the term "gender identity disorder" would be replaced by the more neutral term "gender dysphoria" in the latest version of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-V) [3]. DSM-5 has its own chapter for Gender Dysphoria separated from sexual dysfunction and paraphyllias. The term Gender identity disorder is replaced by Gender Dysphoria.

### **Case History**

A 14 yrs old boy studying in ninth standard from middle class family presented to our Out Patient

Department accompanied by his parents with the chief complaints of withdrawn behavior, irritability, decrease concentration and deterioration in his academic performances for past several months and depressed mood and suicidal ideation for last 2 months and suicide attempt by poisoning 2 weeks back. Since the age of 5 yrs he thinks that he is unfortunately born as male. He had a body of a male, but from inside, he feels like a female. His thinking is of a female character. He likes to play and spent most of the time with the girls, when his elder sister plays with her friends, he also thinks that he is also among them. He preferred to play with toys like dolls, kitchen items, etc. rather than car or truck. He does not love to play outdoor sports like cricket and football among boys. He loves to play indoor games with his elder sister and her friends. He likes to dress and make-up like a girl. He used to dress in frock and a skirt and top like his sister mostly and prefer to keep his hair style like a girl in a playful manner.

Initially there was no discouragement by his family members for his cross gender behavior considering

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it to be a passing phase. But later when he continued insistence on girl type activities and clothing, and his wish to be a girl and to develop like a female bothered his family members. Whenever, his mother or elder sister tried to impose restrictions on these activities, he would become angry and irritable. Later he used to wear the female dress when he was alone.

In the school also he does not want to mix up with the boys, he does not participate in the boys talk. In the leisure time he usually stays in the classroom. He only reveals his feelings to one of his best friend whom he considers to be his boyfriend that he wants to be a girl. He wished to have a long term relationship with this boy but did not express it to him or others and conceal his relationship as he was afraid to be identified as a homosexual by the society.

As he attends his puberty and started developing male secondary sexual characters like growth of facial hairs, body hairs and enlargement of the testis and penis, which was unwelcoming and embarrassing to him. When he gets erection he does not feel good and becomes uncomfortable. He wishes to cut his penis at times. Gradually he started developing depressive symptoms like lack of interest in pleasurable activities, depressed mood, does not want to go school, crying spells, decreased appetite and suicidal wishes. He tried to commit suicide 2-3 times by poisoning. But by luck, he was taken to the doctor in time and recovered.

There is a positive family history of psychiatric disorder. His paternal aunt is having Schizophrenia and elder sister is having OCD. Both are stable and were on regular medicine. The patient had no history of any substance abuse. There is no history of child abuse. Personal history revealed apparently normal developmental milestones with normal motor, social and language development except for the girl type activities and girlish behavior since early childhood. On thorough physical examination, there were no signs suggestive of hormonal dysfunction and intersex on an expert assessment of physician.

On Mental status examination, he is preoccupied with his biological sex and expresses the desire to live like a female. He had depressed affect, suicidal wishes, and contemplation of suicide. On assessment there was no evidence of body delusion, effeminate homosexuality or transvestism. The possibility of Paraphilias and other disorders of sexual preferences were ruled out. His laboratory investigations (endocrine status) and EEG were within normal range. His psychometric assessment was done and his I.Q was found to be 100. Rorschach test revealed depressive symptoms and no other significant finding. Beck Depression Inventory suggests severe

level. He was diagnosed as a case of Gender Dysphoria without the disorder of sex development as per the diagnostic criteria of DSM-5.

Treatment focused on his depression and to save his life due to the impending threat of suicide. He was given SSRI (Sertraline 50mg/day) and psychotherapy for strengthening his biologic sex role as much as possible.

## Discussion

The first case of Gender Identity Disorder was described by Friedreich in 1830, although the condition was not considered worthy of further investigation until many years later. Patient with gender identity disorder is convinced that his/her own psychological gender is the opposite of his/her anatomical sex [4]. Patient with a gender identity disorder feel that they are trapped in the wrong bodies. Male patients feel feminine from childhood and often believe they were 'girls'. But this belief is not delusional in nature. The belief is always consistent with their distaste of their own genitalia, as in our case [5]. Male-to-female transsexuality is 1.5 to 3 times more prevalent than female-to-male [6-9]. The prevalence of transsexualism in the Netherlands is estimated to be 1:11900 males and 1:30400 females [10]. The estimates for the USA are 1:100000 for males and 1:400000 for females. No definite figures are available for India [10].

GID is comorbid with a variety of other psychiatric disorders, particularly mood and anxiety disorders, at rates higher than those reported for comorbid schizophrenia. It is not known if these disorders are the psychological consequence of living with GID, if they reflect shared vulnerabilities that need to be examined in their own right, or if patients with GID are at a non-specifically elevated risk for a variety of disorders [11].

Several studies have shown that those with GID who do present for treatment may have developed additional, secondary diagnoses [12]. In case of our patient also, patient had developed secondary depression and significant suicidal ideations following puberty, which was considered extremely stressful life event by the patient. Early identification and timely intervention may lead to the remission of GID during childhood. If the disorder persists into adolescence, there may be periods of remission but usually it tends to be chronic in nature [13]. Our patient is responding well to therapies attempting to strengthen his biological sex role. His family members were very supportive during the therapy process and

considering his social circumstances, the patient also tried to involve in the therapy.

Gender dysphoria patients are frequently difficult disorder to deal with. An ongoing therapeutic relationship is required for patients regarding his distress so that the patient does not go to the next level of treatment like hormone replacement or surgical sex reassignment.

## References

1. Sexual orientation and gender identity definitions [online] [2013?] [cited 2015 Aug 11]; Available from; URL: <http://www.hrc.org/resources/entry/sexual-orientation-and-gender-identity-terminology-and-definitions>.
2. Stein, E. The mismeasure of desire: The science, theory, and ethics of sexual orientation. New York, NY: Oxford University Press, Inc. 1999.
3. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. Fifth edition. Arlington, VA : American Psychiatric Association; 2013.
4. Roberto, L. Issues in diagnosis and treatment of transsexualism. Archives of Sexual Behavior 1983; 12(5):445-73.
5. Abdul Wahab Yousafzai, Naila Bhutto. Gender identity disorder: Is this a potentially fatal condition? J Ayub Med Coll Abbottabad 2007; 19(4).
6. Bakker, A., van Kesteren, P. J. M., Gooren, L. J. G., & Bezemer, P. D. The prevalence of transsexualism in the Netherlands. *Acta Psychiatr Scand*, 1993; 87:237-238.
7. Garrels, L., Kockott, G., Michael, N., Preuss, W., Renter, K., Schmidt, G., et al. Sex ratio of transsexuals in Germany: The development over three decades. *Acta Psychiatrica Scandinavica*, 2000; 102:445-448.
8. Olsson, S. E., & Moller, A. R. On the incidence and sex ratio of transsexualism in Sweden, 1972-2002. *Archives of Sexual Behavior*, 2003; 32(4):381-386.
9. Wilson, P., Sharp, C., & Carr, S. The prevalence of gender dysphoria in Scotland: A primary care study. *British Journal of General Practice*, 1999; 49:991-992.
10. Richard F. Docter. A Biography of Christine Jorgensen; Notes. New York; The Haworth Press, Taylor & Francis Group; 2008.p. 270.
11. Ravi Philip Rajkumar. Gender Identity Disorder and Schizophrenia: Neurodevelopmental Disorders with Common Causal Mechanisms?. *Schizophrenia Research and Treatment Volume 2014*, Article ID 463757, 8 pages <http://dx.doi.org/10.1155/2014/463757>.
12. Blanchard R, Steiner B, eds. *Clinical Management of Gender Identity Disorders in Children and Adults*. Washington, DC: American Psychiatric Press 1990.
13. Roberto L. Issues in diagnosis and treatment of transsexualism. *Arch Sexual Behav* 1983; 12(5): 445-73.

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[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Kälestål C, Lagerlöf F, et al. Caries-preventive effect of fluoride toothpaste: A systematic review. *Acta Odontol Scand* 2003; 61: 347-55.

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[3] Fleischer W, Reimer K. Povidone iodine antisepsis. State of the art. *Dermatology* 1997; 195 Suppl 2: 3-9.

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[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. *J Periodontol* 2000; 71: 1792-801.

### Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. *Dent Mater* 2006.

### Personal author(s)

[6] Hosmer D, Lemeshow S. *Applied logistic regression*, 2<sup>nd</sup> edn. New York: Wiley-Interscience; 2000.

### Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd EAM,

editors. *Dental caries: The disease and its clinical management*. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

### No author given

[8] World Health Organization. *Oral health surveys - basic methods*, 4<sup>th</sup> edn. Geneva: World Health Organization; 1997.

### Reference from electronic media

[9] National Statistics Online – Trends in suicide by method in England and Wales, 1979-2001. [www.statistics.gov.uk/downloads/theme\\_health/HSQ\\_20.pdf](http://www.statistics.gov.uk/downloads/theme_health/HSQ_20.pdf) (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

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