As I stand before you today, to deliver the Dr. D.L.N. Murti Rao Oration, I am overwhelmed by a series of emotions. Gratitude, because I realise that the Indian Psychiatric Society has honoured me by inviting me to deliver this oration this year; humility because I am aware of my inadequacy to do full justice to this great responsibility; happiness because I am getting a chance to deliver an oration which carries the name of my teacher at whose feet I learned my first lessons of Psychiatry; apprehension because I realise that my predecessors who delivered this oration in the past were stalwarts such as Dr. N. N. Wig, Dr. A. Venkoba Rao, Dr. J. S Neki, Dr. K. C. Dube and Dr. Kripal Singh.

I consider myself very fortunate to belong to the 1960-62 batch of D. P. M. students at the All India Institute of Mental Health, Bangalore, when Dr. D. L.N. Murti Rao was the Director. I remember with a sense of loss and gratitude his abilities as a clinician and teacher. I wrote one of my earliest papers on Mongolism under his guidance. His Myerian approach in understanding the different psychiatric conditions influenced my concepts in psychiatry. He was a man of few words; but he made a lasting and deep impression on his students. He showed a keen interest in the study of the phenomenology of schizophrenia and used to point out that schizophrenia is a mystery, a jigsaw puzzle and we must take it as a challenge to put these pieces together to get a complete understanding of this mystery. My interest in schizophrenia has been a result of Dr. Murti Rao's inspiration and I have selected the following title for my oration in homage to him: "A search into the mystery of Schizophrenia". I would like to spend some time in elaborating a few theoretical viewpoints regarding some aspects of schizophrenia and also to highlight some areas of research in schizophrenia in which I have been involved.

DIAGNOSIS AND CLASSIFICATION

Several centres have been involved in research into various aspects of schizophrenia. But there has been very little consistent agreement among the various workers. The main cause of this confusion is that there is no agreement in the criteria of diagnosis of schizophrenia. According to Kreitman et al (1961) the agreement in the diagnosis of schizophrenia among psychiatrists in Britain is only about 60%, the rate varying according to the theoretical background of the psychiatrist. Several attempts have been made to minimise this problem of diagnosis by establishing a widely accepted operational definition of schizophrenia. The ICD-9; DSM III; Feighner's criteria; New Haven Schizophrenic Index; Research Diagnostic Criteria; criteria developed by Carpenter et al from the I.P.S.S. and the criteria developed by Taylor and Abram (Endicott et al, 1982) are some of the diagnostic systems, widely used. Though the reliability of the above systems is high, they vary greatly in their rates of diagnosis of schizophrenia. I believe that we must...
accept restricted criteria of diagnosis of schizophrenia. The concept of schizophrenia as it stands now is so vague and wide that it ceases to be a well defined entity. A restricted criterion of diagnosis, bringing in both cross sectional and longitudinal aspects will be useful and DSM III is a step towards the same.

Adolf Meyer’s (1937) concept of reaction types in psychiatry is a useful one in the understanding of schizophrenia (Meyer, 1957). According to him, schizophrenia is the outcome of progressive maladaptation of the individual to his environment. When an individual who is predisposed to develop schizophrenia by genetic loading, has to face several problems in his life situation, abnormal reactions appear which have certain general similarities and these are the common schizophrenic symptoms. Those who have a heavy genetic loading, find it difficult to cope with even the normal burdens of life. Those who have a light genetic loading can break down into schizophrenic symptoms when loads of life become too difficult to carry. Thus we have two groups of schizophrenia, on the extreme ends of a spectrum. In some cases of schizophrenia, the genetic factors are predominant in etiology, and in others the environmental factors are more important. These two groups of schizophrenia, classified from an etiological point of view are very similar to the two groups of schizophrenia, described by Langfeldt and Krapelin from a prognostic point of view (Langfeldt, 1937; Krapelin, 1919).

Since the terms endogenous and reactive are widely used in affective disorders, these terms could be retained in schizophrenia also. Thus endogenous schizophrenia where genetic factors are more important in etiology is similar to Langfeldt’s process or nuclear schizophrenia and Krapelin’s Dementia Precox. Reactive schizophrenia where environmental factors are more important in etiology is similar to Langfeldt’s schizophrenic form psychosis. This dichotomy should not be viewed as separate diagnostic entities, but should be considered functional units at two ends of a continuum of genetic vs environment model which will be useful in research and clinical management.

The validity of this functional dichotomy depends on whether this differentiation is possible in clinical practice. I believe that it is. All of us are impressed that some schizophrenic patients do not respond well to treatment and some do. We also are able to predict with a reasonable degree of accuracy the prognosis of patients. The main bad prognostic factors are a schizoid premorbid personality, family history of schizophrenia, early and insidious onset, several attacks of illness and absence of obvious precipitating factors. These factors are those which refer to genetic involvement. The endogenous schizophrenia will be characterised by schizoid personality, insidious and early onset; family history of schizophrenia; of a tendency to deteriorate. The emphasis is more to a longitudinal diagnosis. In the reactive type of schizophrenia, the schizoid personality is not common; significant precipitating factors are usually present; age of onset is usually late. The diagnostic criteria of endogenous or process schizophrenia used in our department are as follows:

A. A diagnosis of schizophrenia must be made as per ICD-9-295. B. Deterioration from previous level of functioning in such areas as work, social relations, and self care. C. Duration: continuous signs of illness for at least 6 months. D. At least 3 of the following:

(i) Schizoid personality: I.C.D.-9: 301.2 (ii) Family history of schizophrenia (iii) Insidious onset without precipitating factors (iv) Early onset: before 20 years.

Those who satisfy A and not B to
D will be Reactive Schizophrenia.

There will be several patients who may show a mixed phenomenology which only supports the dynamic concept of schizophrenic reaction types. These come in between the two extreme groups and could be called the mixed type. This dichotomy does not suggest two separate diseases; but two ends of continuum of genetic involvement. Because of the differences in the degree of genetic involvement, they show differences in various aspects.

If the above dichotomy is valid, it should be possible to differentiate these two forms in the areas of epidemiology, biochemistry, genetic studies, somatotyping, family dynamics and other parameters. There are some reports which suggest that these two groups of schizophrenia differ in some of these respects.

Rosenthal (1959) examined the case histories of monozygotic twins with schizophrenia that has been previously published by Slater. He found an almost total absence of schizophrenia among the families of the discordant twins in contrast to a positive history of schizophrenia among 60% of families of the concordant twins. Moreover the discordant male twins tended to have a later age of onset, more stable premorbid personality and more favourable outcome than the concordant group. Rosenthal concluded that biologically speaking at least two broad groups of schizophrenia were differentiated by this method of analysis. In one, the genetic contribution was absent or minimal; in the other, the genetic contribution was considerable. Inouye (1961) found 74% concordance in the group of monozygotic twins with progressive chronic schizophrenia and only 39% among the mild transient types of schizophrenia. It is possible that these two subgroups may be differentiated biochemically. In this connection Crow's (Crow, 1962) suggestion of Type I and Type II schizophrenia becomes relevant. The Indian Council of Medical Research is conducting a multicentred study on the factors associated with the course and outcome of schizophrenia. One hypothesis of this study is that it is possible to identify two groups of schizophrenia one with poor course and outcome and the other with good course and outcome.

In all research work on schizophrenia, the existing confusion and contradictory reports can be minimised if not avoided if these two subgroups are recognised and separated. This dichotomy will also give different emphasis on treatment. Psychotherapy of varying degrees of supportive, reeducative and reconstructive involvement, as suggested by Arieti (1974), will be required in these two types of schizophrenia. Long term drug therapy may be imperative in endogenous schizophrenia and not in reactive type.

Thus a subgrouping of schizophrenia into endogenous and reactive types will be more useful than the present subtyping of schizophrenia into simple, catatonic, hebephrenic and paranoid types. The ICD-9 adds the following subgroups also: acute schizophrenia, latent schizophrenia, residual schizophrenia and schizo-affective type. All of us are aware that we often find it difficult to allot many patients to the above diagnostic categories. The same patient can change these subtypes from time to time. If at all this traditional subtyping of schizophrenia is valid, it would be paranoid schizophrenia and non-paranoid schizophrenia. There are differences between these two groups in age of onset, bodybuild, neurophysiology etc. The diagnosis of simple schizophrenia is difficult in our culture. The many features of this subgroup viz. insidious onset of oddities of conduct, inability to meet the demands of society and decline in total performance
are sometimes difficult to be branded as diagnostic schizophrenia. If we do so, we may get into the danger of calling many of our sages and prophets schizophrenic just because they chose to be different from the general stream of society.

PREVALENCE RATE OF SCHIZOPHRENIA

There are several reports which suggest that schizophrenia is a common psychiatric disorder, prevalent in all communities, in all cultural back grounds. I was therefore interested to find out the prevalence rate of schizophrenia in and around Vellore. A mental health survey was therefore conducted in 1972 (Verghese et al, 1973). Using a three stage random sampling technique, from among 26039 households, 539 households consisting of 1827 adults were studied. Of them, 5 were diagnosed as suffering from Schizophrenia thus giving a prevalence rate of 2.6 per 1000. This is compared to the prevalence rate of schizophrenia reported by other workers (Table 1). It is found that there is a good degree of agreement in the prevalence rate of schizophrenia around 2 to 3 per 1000 in different communities, in spite of differences in culture. This similarity in the prevalence rate of schizophrenia across cultures which would still be closer if more restricted criteria of diagnosis are followed is one point in favour of a genetic transmission in schizophrenia.

CLINICAL FEATURES OF SCHIZOPHRENIA

A retrospective psychosocial study was done on 292 patients diagnosed as schizophrenia and treated as inpatients in our Department (Subramanian and Verghese, 1977) 57% were males; 59.2% were married; the peak age group was 15-29 years; 33.5% had positive family history of schizophrenia; 36.3% showed some significant precipitating factors; the majority of them had only secondary education; 58.2% had some features of schizoid premorbid personality; 12.3% had experienced parental deprivation; sleep disturbance, paranoid delusions, bizarre thoughts, restricted affect, auditory hallucinations, impulsiveness and incongruity of affect were the commonest symptoms in order of descending order frequency. At discharge, there were varying degrees of improvement in about 92% of patients. 118 patients were followed up by contacting them through letters. Among the 118 patients, 40 made social recovery; 47 were improved as far as symptoms were concerned; 20 patients were the same and 9 became worse and 2 died. When the 40 patients who made social recovery were compared to 29 patients who did not improve, three factors which were associated with poor prognosis were identified. These were, longer duration of illness, insidious type of onset and family history of schizophrenia. These variables were found to be significantly related to poor outcome in the IPSS study as well.

We made another study on the
PREVALENCE, DIAGNOSTIC USE AND PROGNOSTIC IMPLICATIONS OF SCHIZOPHRENIA

A prospective study of 88 schizophrenic patients who satisfied the criteria of Feighner et al (1972) for schizophrenia was made. 35% of these had one or more of first rank symptoms (FRS). Patients who had FRS and those who did not have FRS were compared. They did not differ in age, sex, duration of symptoms, mode of onset, premorbid adjustment, family history of schizophrenia or mode of outcome. It was also found that paranoid schizophrenias vs. nonparanoid schizophrenias as well as endogenous vs. reactive type of schizophrenia did not differ in the frequency of FRS.

Considerable variation has been found in the prevalence of FRS in schizophrenia at various centres (Table 2). Mellor (1970) in U.K. found that about 72% of his sample of schizophrenic patients had FRS while Silverstein and Harrow (1978) found a prevalence rate of only 24%. This range is seen in the observations of the International Pilot Study of Schizophrenia as well: (Carpenter et al, 1973) USSR 31%; India 48%; U.K. 76%; and Taiwan 79%. Chandrasena and Rodrigo (1979) in their study of schizophrenia in Sri Lanka found a prevalence rate of 25.4%. The findings of this study suggest that the presence of FRS, although useful in the diagnosis of schizophrenia, is not pathognomonic of schizophrenia.

**BIRTH ORDER**

Interest in the relationship between birth order and psychiatric disturbance has been stimulated by the teachings of Adler (1937) who pointed out that the personalities of the oldest, the middle and the youngest child in a family are likely to be different and this difference could be attributed to the distinctive experiences of each child as a member of the social group. Most of the studies in this field have been done with special reference to schizophrenia. Prabhru and Ramachandran (1973) have reviewed various studies on birth order and schizophrenia with in the west and in the east. As in other studies of schizophrenia, here also we are left with confusing and contradictory reports. Several studies reported from the west have claimed an overrepresentation of the later born among schizophrenic patients (Wahl, 1956; Farina et al, 1963; Schooler, 1964; Granville-Grossman, 1966; Barry, 1967). Some workers have though, failed to find any significant relationship between birth order and schizophrenia (Grosz and Miller, 1958; Burton and Biro, 1963; Smith and McIntyre, 1963; Tsuang, 1966). In the few studies reported from the east a consistent finding has been that the early borns were over represented among schizophrenic patients (Murphy, 1959; Rao, 1964; Sundar Raj and Rao, 1966; Teja, 1967; Prabhru and Ramachandran, 1973). But Ajita Chakraborty (1969/70) did not find any association in her study of birth order and illness.

An investigation was done in our Department to look at this aspect (Abraham et al, 1973). Expected and observed

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**Table 2. First rank symptoms in schizophrenia**

| Patient | Prevalence Rate |
|---------|----------------|
| Mellor (1970) : U.K. | 72% |
| Silverstein & Harrow (1978) : U.K. | 24% |
| I. P. S. S. (USSR) | 31% |
| I. P. S. S. (India) | 48% |
| I. P. S. S. (UK) | 76% |
| I. P. S. S. (Taiwan) | 79% |
| Chandrasena & Rodrigo (1979) : Sri Lanka | 25% |
| Radhakrishnan et al (1983) (Vellore) | 35% |
birth order were calculated in a group of schizophrenic patients, a group of neurotic patients and a third group of medical patients, using the method of Greenwood and Yule (1914) and Slater (1962). The results indicate that the oldest were over represented among the female schizophrenia group, the female neurotic group and the male medical group (Table 3). Hence if at all there is any association between birth order and schizophrenia, it is not specific to schizophrenia. We have to be very careful in drawing any conclusions and to make interpretations. The possibility of an artefact must be ruled out. Price and Hare (1969) and Prabhu and Ramachandran (1973) have enumerated the possible biases which have bedevilled many birth order studies. They have emphasised biases in the selection of populations; incomplete sibbling, definition of a case and sib; and statistical techniques used. Critically examining these birth order studies it seems that if at all there is any etiological association, it is very minimal.

HISTAMINE TOLERANCE

Several clinical reports from very early times suggested that a psychotic patient was less likely than the normal to exhibit allergic symptoms (Cassell and Fisher, 1963; Simpson & Khane, 1961). While the allergic status of the schizophrenic patient is not settled, one consistent finding observed by several workers is that schizophrenic patients especially those who were sick for a long time, tolerate larger doses of histamine. Lucy (1954) studied this phenomenon of increased histamine tolerance in schizophrenia. He found that the schizophrenic patients as a group tolerated larger doses of histamine. There was no correlation between body weight and the amount of histamine tolerated. But there was a marked positive correlation between duration of the illness and the amount of histamine tolerated, the long standing patients tolerating larger quantities of histamine. Ermala and Autio (1951) studied skin response to histamine in 252 subjects and found consistently smaller wheals in the schizophrenic patients than in other psychotic or control groups. Freedman et al. (1956) skin tested 22 schizophrenic patients and 22 medical students with a battery of allergens and histamine. The patient group showed no diminished ability to react to the allergens; but did have consistently smaller wheal response to histamine. Several workers have reported higher histamine levels in schizophrenia than normal serum (Wyatt et al., 1971). Sanyal et al. (1970) reported increased blood histamine levels; reduced blood hist-

| TABLE-3. Birth-order and schizophrenia | Male | Female |
|----------------------------------------|------|--------|
|                                       | Ex- | Ob- | Ex- | Ob- |
|                                       | spected | served | spected | served |
| Schizophrenia                          |     |     |     |     |
| Eldest                                 | 57.9 | 65.0 | 38.5 | 51.0 |
| Youngest                               | 57.9 | 42.0 | 38.5 | 24.0 |
| Intermediate                           | 143.2 | 152.0 | 101.0 | 103.0 |
| $x^2=4.6, df=2, p<0.02$                |     |     |     |     |
| $x^2=8.8, df=2, p<0.02$                |     |     |     |     |
| Neurotic                               |     |     |     |     |
| Eldest                                 | 27.6 | 33.0 | 19.0 | 26.0 |
| Youngest                               | 27.6 | 18.0 | 19.0 | 9.0  |
| Intermediate                           | 68.8 | 73.0 | 43.0 | 46.0 |
| $x^2=3.1, df=2, p<0.02$                |     |     |     |     |
| $x^2=8.1, df=2, p<0.02$                |     |     |     |     |
| Medical                                |     |     |     |     |
| Eldest                                 | 38.5 | 49.0 | 15.8 | 18.0 |
| Youngest                               | 30.5 | 25.0 | 15.8 | 15.0 |
| Intermediate                           | 81.0 | 84.0 | 37.4 | 36.0 |
| $x^2=7.8, df=2, p<0.03$                |     |     |     |     |
| $x^2=0.6, df=2, p=NS$                  |     |     |     |     |
A SEARCH INTO THE MYSTERY OF SCHIZOPHRENIA

Histaminase levels; and reduced skin sensitivity in a group of schizophrenic patients.

An investigation into the histamine tolerance in a group of schizophrenic patients; a group of non-schizophrenic patients and a group of normals was carried out in our Department (Verghese and Thomas, 1972). Each subject was given an intradermal injection of 0.05 mgm histamine on the flexor side of the right forearm. The size of the wheal was measured in m.m. 30 minutes after the injection which was taken as the measure of response to histamine. Larger the wheal, greater the response (decreased tolerance). The 3 groups were compared in their histamine responses (Table 4).

**Table-4. Histamine tolerance in schizophrenia**

| Group          | No. | Histamine Wheal (m.m.) |
|----------------|-----|-----------------------|
| Schizo : Total | 42  | *9.2±4.8              |
| Schizo : >5 Yrs.| 7   | *4.9±2.3              |
| Schizo : <5 Yrs.| 35  | **10.1±4.4            |
| Nunschizo      | 24  | 13.9±6.9              |
| Normal         | 25  | 15.8±4.7              |

*p<0.01 (VS. Normal)  **p<0.05

The schizophrenic patients had smaller wheals. Those who were sick for more than 5 years, showed the least response. The response was significantly correlated to duration of illness; but not to body weight or age.

Thus our finding is in agreement with the observations of other workers. As Smythies puts it, an increased histamine tolerance in schizophrenia is one of the very few consistent findings in schizophrenia. But it is not certain what the significance of this is, especially in relation to the Dopamine hypothesis of schizophrenia.

MONOAMINE METABOLITES IN SCHIZOPHRENIA

Serotonin is 5 Hydroxy Tryptamine which is an indole amine and its major metabolite is 5 Hydroxy Indole Acetic Acid (HIAA). Dopamine is a catecholamine and its major metabolite is Homovanillic acid (HVA). Woolley and Shaw (1954) postulated that schizophrenia is associated with deficiency of Serotonin, since LSD which is a serotonin antagonist produces symptoms similar to schizophrenic symptoms. Smythies et al (1970) followed this up and developed the serotonin hypothesis of schizophrenia. He suggested that acute LSD psychosis resembles acute schizophrenia and chronic LSD usage resembles chronic schizophrenia. Serotonin neurons have their cell bodies concentrated in the Raphe nuclei in the brain stem and this area is related to sleep, mood, and filtering of incoming sensations and connected with the meta organisational system which controls the symptoms often seen in schizophrenia. The basic disorder may be a disturbance in Serotonin transmission because of a relative deficiency of Serotonin. One of the recent observations that serotonergic neurones are deficient in the Frontal Lobes of schizophrenic patients is important in this connection (Bennet et al, 1979).

The strongest available hypothesis for schizophrenia is the Dopamine hypothesis. The observations of several workers that CSF HVA is normal in schizophrenic patients (Bowers, 1974; Post et al, 1975) suggest that the schizophrenic symptoms are the result of a hypersensitive Dopamine receptor system rather than a hyperactive Dopaminergic system. There are a number of inadequacies for the Dopamine hypothesis of schizophrenia. Most probably a number of systems are involved. Along with a hypersensitive dopamine receptor system, an associated deficiency of serotonin also may be an impor-
tant factor in the etiology of schizophrenia.

We estimated the CSF HVA and HIAA on 14 patients who satisfied the Feighner's criteria of diagnosis for schizophrenia and compared them to a matched normal group (Verghese et al., 1982). The group of schizophrenic patients had normal HVA and lower HIAA levels (Table 5). These observations are in agreement with both the Dopamine hypothesis and Serotonin hypothesis of schizophrenia, it is most likely that in the etiology of schizophrenia more than one neurotransmitter is involved. There is experimental evidence to suggest that neurotransmitters such as acetylcholine, norepinephrine, GABA, and phenylethylamine are also important in the etiology of schizophrenia.

| TABLE-5. Monoamine metabolites in schizophrenia |
|-----------------------------------------------|
| Group | No. | HVA (ng/ml) | HIAA (ng/ml) |
|-------|-----|-------------|--------------|
| Normal| 10  | 28.9±11.4   | 51.1±21.6    |
| Schizo.| 14  | 41.0±23.0   | 33.1±19.2    |

*P<0.05

PLASMA CORTISOL IN SCHIZOPHRENIA

The activity of the adrenal cortex has been intensely studied in human subjects undergoing emotional disturbance. Several stress conditions such as hospitalisation and anticipation of surgery have been shown to be associated with increase in plasma cortisol (Mason et al., 1965). Patients also were diagnosed as suffering from anxiety state showed high level of plasma cortisol and urinary corticosteroids (Rubin and Mandell, 1966). The study of adrenocortical function has been most active and important in depressive illness (Carrol et al., 1968). The adrenal corticosteroid activity in schizophrenia has not been studied as extensively as in depressive illness. Some workers have reported underactivity of corticosteroids in schizophrenia while others have not observed any change. Bliss et al. (1955) assessed the corticosteroid activity in 26 chronic schizophrenic patients and reported that the adrenocortical physiology was normal in those patients. Anderson and Dawson (1965) reported a raised level of 17 hydroxy corticosteroids in the fasting blood of schizophrenic patients.

We estimated the plasma cortisol in 12 patients who satisfied the Feighner's criteria of diagnosis of schizophrenia (Verghese et al., 1973). The results showed that in the group of schizophrenic patients, plasma cortisol level is raised, but the diurnal variation and dexamethasone suppression were normal. This shows that in schizophrenic patients with florid symptoms, there is an increase in corticosteroid function and that this increase is not specific to schizophrenia.

"The investigations to use the neuroendocrine system to identify biological abnormalities in schizophrenia have produced inconsistent results. However the approach does permit the assessment of specific responses to neurotransmitter agonists and antagonists at specific receptors which might lead to a biological means of identifying individuals with aberrant receptor dopamines." (Meltzer, 1979).

SOMATO TYPING : BODY BUILD IN SCHIZOPHRENIA

The relationship between body build and temperament has been widely proclaimed from early days, but the scientific study of the relation between physical constitution, temperament and mental illness was first initiated by Kretschmer (1936), followed by Sheldon et al. (1940), Rees Eysenck (1945), Parnell (1958) and
There have been several reports on the relationships between personality, body build, and susceptibility to mental illness. Eysenck (1947) found that leptomorphs tended to develop dysthymic symptoms and endomorphs hysterical symptoms. Moore and Hsu (1946) reported that patients who had nonparanoid schizophrenia were different from patients who had paranoid schizophrenia and Manic depressive psychosis in being more leptomorhpic. Glueck and Glueck (1950) and Epps and Parnell (1952) reported that delinquents were more mesomorphic and less ectomorphic. Rees (1960) found that schizophrenic patients were more leptomorphic. Betz (1942) reported that female schizophrenic patients were more leptomorphic, paranoid schizophrenics, according to Connolly (1939), were more pyknic than other types of schizophrenic patients. Tangfeldt (1937) claimed that schizophrenic patients with leptomorphic body build tended to have an early age of onset and greater degree of withdrawal. Kallman (1953) suggested that schizophrenic patients with leptomorphic body build had a poor prognosis. Thus a review of literature suggests a relationship between the type of mental illness and body build. The consistent findings are that the nonparanoid schizophrenic patients are more leptomorphic than paranoid schizophrenic patients and neurotic patients with dysthymic symptoms are more leptomorphic than those with hysterical symptoms.

We have been interested in this topic. All male patients are routinely assessed to estimate the Rees Eysenck body index. This index is a good measure of body build (Rees and Eysenck, 1945), and makes use of height and chest diameter which are two measurements which do not change much after reaching adulthood. These two measurements are combined in a ratio to give a mean around 100 which gives an idea as to where an individual would be on the body continuum. Higher the score, greater the tendency to be leptomorphic and vice versa.

\[
RE \text{ Bodybuild } = \frac{\text{height} \times 100}{\text{Chest diameter} \times 6}
\]

A preliminary report on our findings was published in 1971 (Vellore, 1971). The main observation was that the nonparanoid schizophrenic patients were more leptomorphic than paranoid schizophrenic patients. We have now repeated this study in a group of schizophrenic patients who were admitted in our Department during the period 1981-1983 and who satisfied the Feighner's criteria of diagnosis for schizophrenia. The analysis of data shows that the nonparanoid schizophrenic patients are significantly more leptomorphic than paranoid schizophrenic patients.

A similar study was conducted by me on a group of male schizophrenic patients admitted in Royal Park Psychiatric Hospital, Melbourne, Australia (Vellore et al., 1978). In all these studies, similar results were obtained. The nonparanoid schizophrenic patients were more leptomorphic than paranoid schizophrenic patients (Table 6).

| Group            | No.  | R.E.B.I. (Mean ± S.D.) |
|------------------|------|------------------------|
| Vellore (1971)   |      |                        |
| Nonparanoid      | 75   | 106.9 ± 8.0            |
| Paranoid         | 20   | 98.4 ± 7.7 p < 0.05    |
| Melbourne (1978) |      |                        |
| Nonparanoid      | 97   | 102.5 ± 7.5            |
| Paranoid         | 18   | 99.9 ± 7.0 p < 0.05    |

Thus there is a consistent tendency for the nonparanoid schizophrenic patients to be more leptomorphic than para-
noid schizophrenics. The relevance of this observation is not certain. It is probable that paranoid schizophrenia is different from nonparanoid schizophrenia as pointed out earlier. There is some evidence to suggest that paranoid schizophrenia should be classified along with paranoid state and not with schizophrenia (Kendler and Davis, 1981). We are in the process of finding out whether there will be any difference in body build if we separate the schizophrenic patients into endogenous and reactive type.

ATTENTION DEFICIT IN SCHIZOPHRENIA

Several workers have postulated that the primary deficit in schizophrenia is in attention, an impairment in the central filter mechanism which normally screens out irrelevant sensory input to allow the efficient processing of relevant data (Venables, 1964; Payne & Caird, 1967). This failure of normal filtering mechanism leads to various kinds of thinking disorders. Lehmann (1966) suggests that schizophrenic patients have a primary, possibly constitutional susceptibility to be subject to the impact of a higher number of discrete sensory stimuli per time unit of experience. Broen (1968) suggests that the schizophrenic patient narrows his range of observation and thus restricts the number of stimuli actually received. Broadbent (1958); Yates (1966) and McGhie (1970) have hypothesised that schizophrenic deficit lies in the inability to screen out relevant from irrelevant information with the result that the single channel becomes overloaded.

The above hypothesis of a primary deficit in the central filter mechanism in schizophrenia leads to the possibility of sensory disturbances both auditory and visual in schizophrenia. Venables and Tizard (1958) measured reaction times to auditory and visual stimuli over a range of stimulus intensities. They reported that reaction times to auditory stimuli were larger than those to visual stimuli. This is a reversal of the normal reaction where reaction times to auditory stimuli are faster than those to visual stimuli. This suggests a disturbance of function in the auditory modality in schizophrenia.

We have conducted pure tone audiometry studies on 12 schizophrenic patients (I.C.D.-9) and 10 normals (staff and students) between the age range 16-38 years. The results showed that schizophrenic patients had a higher threshold to elicit a response, in both the ears, for both airconduction and bone conduction. If this observation is confirmed, it can be due to the fact that schizophrenic patients are usually preoccupied. It could also be due to a primary deficit of the central filtering mechanism in schizophrenia.

VESTIBULAR FUNCTIONS

Mechl (1962) reviewed the literature on vestibular dysfunction in schizophrenia and found that a defect in the vestibular system is a consistent observation of several workers. Early studies by Freeman and Rodnick (1942) showed that schizophrenic patients exhibit less unsteadiness after rotation than do normals. More recent studies (Myers et al, 1973) using electronystagmographic techniques provide similar evidence of dysfunction following rotation. Other workers using caloric stimulation to elicit vestibular activity have confirmed the above findings (Angyal and Sherman, 1942). It is suggested that the impairment of vestibular activity may be one of the factors that underlie the disturbances of body image and feelings of depersonalisation often complained by schizophrenic patients. We have conducted vestibular function tests (caloric test) on the two groups of subjects who...
had audiometry. Although there was no difference in directional reponderance, the schizophrenic patients had deficient canal paresis.

LIFE EVENTS

The role of stressful life events in the etiology of various diseases has been a field of research nearly for the last 25 years. In general the purpose of such research is to demonstrate a temporal association between the onset of illness and a recent increase in the number of events that require socially adaptive responses on the part of the individual. Most investigators in the field of life events research have adopted in original or modified form a 43 item check list developed by Holmes and Rahe in (1967). In India, Venkoba Rao and Nammalvar (1976) and Gurmeet Singh et al (1981) developed check lists consisting of 67 and 51 items respectively.

The large body of research in the past 15 years has provided evidence that recent life events contribute to the onset of psychiatric impairment. Retrospective studies indicate that life events are experienced with greater than expected frequency prior to the onset of depression, schizophrenia, and suicide attempts. In the New Haven Study, Jacobs and Myers (1976) reported that in a group of 62 patients with acute schizophrenic illness, there was a significantly higher rate of life events during the one year prior to the onset of illness. In the London study, Brown & Birley (1968) reported that there were more crises and life changes in the group of schizophrenic patients during the 3 month period prior to the illness.

We have modified the stressful life events scale developed by Gurmeet Singh et al. Three parameters can be measured using this life events scale; the number of life events; the objective stress score and subjective stress score. 30 subjects in each of the following were studied. Schizophrenia as per Feighner's criteria; Depressive illness (DSM III), Neuroses (DSM III) normals (staff and students). The mean number of events experienced, subjective stress and objective stress were calculated for all the groups. There were no differences in the mean number of events and objective stress experienced. The schizophrenic group had the least amount of subjective stress experienced.

We are continuing this work on life events. It may be possible to have more meaningful observations if the schizophrenic group is subdivided into paranoid and nonparanoid groups as well as endogenous and reactive groups. Brown and his colleagues believe that life events are less important than dispositional personality factors for schizophrenic breakdown (Brown et al, 1973). It is my feeling that life events are more important in the etiology of reactive schizophrenia than in endogenous or process schizophrenia.

DERMATOGLYPHICS IN SCHIZOPHRENIA

Dermatoglyphics is the study of epidermal ridge configurations on the finger tips, palms and soles. The fine dermal ridge patterns appear around 13th to 19th week of gestation and are genetically determined. They are constant throughout life. The study of dermatoglyphics will help us to determine the degree of genetic involvement; to identify populations at risk; and to make clinical diagnosis more reliable. Several workers have reported on various aberrations in dermatoglyphics in schizophrenia. Balgir and Murthy (1982) has reviewed the major Dermatoglyphic studies in schizophrenia. The commonly reported dermatoglyphic abnormalities are the following: Decrease of interdigital patterns except 13; increased SRBC; decreased DRBC; increased atd angle; decrease in
whorls and increase in arches in males and vice versa in females; and differences in total ridge count.

As in other studies in schizophrenia, there does not seem to be a characteristic pattern of dermatoglyphic abnormality in schizophrenia. These contradictory findings are mainly due to problems of diagnosis. The following precautions should help to get more consistent and meaningful observations. The population must be large; the control subjects must be from the same ethnic group; the population studied must be separated into males and females. A restricted criterion of diagnosis of schizophrenia should be used. The left and right hands should be separately studied. The schizophrenic patients can be subgrouped into paranoid and nonparanoid groups as well as into endogenous or process and reactive or schizophrenic form groups. One could expect to get more consistent dermatoglyphic aberrations in endogenous schizophrenia where genetic factors are more important in etiology. We are looking into these aspects. Initial impressions suggest that there are some significant dermatoglyphic aberrations in schizophrenia. Schizophrenic patients have a tendency to have an increase in SRBC; decrease in DRBC; increase in 4th interdigital area pattern; decrease in 3rd interdigital area pattern; and an increase in hypothenar pattern. There is also a tendency for most of these variations from the normal to be more in nonparanoid type of schizophrenia.

**CONCLUSIONS**

I have made an attempt to highlight on some aspects of schizophrenia which have been areas of my interest. All of us would agree that there are many aspects of schizophrenia which need a synthesis to give any meaningful interpretation. Schizophrenia continues to be a syndrome, not a clinical entity. So the most urgent need is to come to an agreement as to what exactly we mean by schizophrenia. For this process of circumscribing schizophrenia, we must agree on a restricted criteria of diagnosis. Again within this spectrum of criteria of diagnosis of schizophrenia, we should explore the possibility of making a functional dichotomy of endogenous and reactive types of schizophrenia. I remember how Dr. Murti Rao used to refer to this idea by comparing schizophrenia to an elephant and all the research workers engaged in the study of schizophrenia to the blind men who try to understand the elephant through their limited experience of the different parts. I believe that we have come a great way in the understanding of schizophrenia and that the riddle of schizophrenia will be solved soon. Let us hope that this will happen in our life time. Thus, with this note of optimism, I conclude this oration which I had the honour to deliver today in honour of my teacher and guide in Psychiatry, Dr. D.L.N. Murti Rao, I thank the Indian Psychiatric Society again for giving me this honour and privilege.

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