Indian J. Psychiat., 1997, 39 (2), 90-104

DR. D.L.N. MURTI RAO ORATION

PSYCHIATRY-PAST, PRESENT & FUTURE
A PERSONAL VIEWPOINT

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ABSTRACT

An overview of psychiatry during the last three decades as practised in a general teaching hospital is presented. Psychiatry as an academic subject has matured tremendously during this period. The empirical treatments of the 1950s and the 1960s which evoke nostalgic memories, have been replaced by modern methods of treatment. However, there is a need to exercise caution against the blind acceptance of new and sophisticated research findings in biological psychiatry. Inspite of the bright future facing psychiatry, the identity of psychiatry as a medical discipline must be preserved at all cost. Psychiatrists should also realise the dangers of gradual fractionation and impersonalisation which threatens the speciality, and makes all possible efforts to prevent this.

Key Words :General hospital psychiatry, three decades overview

Mr. President, dignitaries on the dais, my distinguished colleagues, ladies and gentlemen.

I wish to express my sincere appreciation for the opportunity and the honour of delivering Dr. Murti Rao oration.

It is a special privilege to deliver this oration in our eminent society's Jubilee year. I consider myself twice blessed as this is also the jubilee year of the department of Psychiatry, KEM Hospital, Bombay which is my alma mater.

Prof. Murti Rao was a great clinician. He was a biologically oriented psychiatrist. In tune with this, I have chosen to speak to you on "Psychiatry-Past Present and Future" as I have been involved with the subject for more than three decades, from 1959 when I joined the department as a student until my retirement as Professor and Head in 1989.

I see before me faces which are familiar and friendly. Some of you have been my students, some of you are my colleagues and some of you are my seniors. Many of you have helped me, taught me, encouraged me, challenged me and given me pleasure in countless ways over a number of years.

I would like to share with you my past memories of psychiatry as it was in the good old days of yesterday, my impressions of psychiatry as I see it today, and my hopes and fears about psychiatry as it could be in the times to come.

BACKGROUND

In 1959, I qualified MD in general medicine. I was interested in the neurosciences. I was very impressed by neurologists who could localise a lesion to half a centimetre of brain tissue in patients who had massive neurological disabilities.

But the neurologists therapeutic skill was not equal to his diagnostic skill. Except for prescribing physiotherapy or an indwelling catheter, there wasn't much that could be done for most patients. So I spent some time as a visitor in psychiatry.

The psychiatry department was about 10

years old. I had heard a lot of stories about psychiatry. Its image was not as presentable as it is today. The subject was taught superficially or not at all. At conferences, the participating doctors seemed as confused as the patients they were discussing. I entered the department with a lot of uncertainty.

Everything that I observed in the department seemed new and interesting and very different from general medicine. I saw nurse going around distributing sweets to some patients. I believed it was somebody's birthday until I was told that it was part of insulin treatment and an everyday affair. This was quite puzzling. Like all fresh M.D.s I believed that I knew all there was to know in medicine. I could not recollect anything about carbohydrate metabolism and psychiatric treatment. I even referred to my text book "Price's Practice of Medicine." I don't think the book is in print any longer.

Then I saw a patient who was being resuscitated. I asked the attending doctor about the nature of the emergency. The doctor informed me that this was carbon dioxide therapy and not an emergency. Once again I referred to my text book. Carbon-dioxide was mentioned in the chapter on toxins, poisons and antidotes. But there was no mention of carbon dioxide in the chapter on therapeutics.

Next I saw a patient lying down. Some people were holding him while the anaesthetist was administering anaesthesia. I asked someone why the patient had to be restrained when he was being anaesthetized for surgery. I was told that this was not surgery but electroconvulsive therapy.

I knew nothing about psychiatry. I had skipped attending lectures and I kept on reading the wrong books. No wonder I found psychiatry different and exciting, and I decided to continue in psychiatry.

I told my father who was a family physician, about my decision. He was very upset. He told me that he had consulted the city's best psychiatrists for his patient's problems. He could see no difference between psychiatric patients and psychiatric doctors. He advised me to forget this silly idea and to practice medicine like a proper doctor.

My father had passed the MBBS examination in 1929. Psychiatry was not taught in those days. Both of us knew nothing about psychiatry, but for different reasons. It is not surprising that my father did not think much of psychiatry, as at that time there were no effective treatments like electroconvulsive therapy or drug therapy; and the physician and the surgeon both looked down upon psychiatry with disdain and considered it a lot of mumbo jumbo.

PSYCHIATRY IN THE PAST

It is difficult to divorce psychiatry's past from its present, as the dividing line between the two is imperceptible.

History tells us that the first psychiatric unit in a general teaching hospital was established in 1924 at the Henry Ford Hospital in Detroit, USA (Lebensohn, 1965). The first psychiatric unit in a general teaching hospital in India was established in 1933 at the R.G.Kar Medical College and Hospital in Calcutta (Chakarborty, 1970). The Department of Psychiatry at the KEM Hospital, Bombay started functioning in 1947, twenty two years after the hospital was founded (Vahia et al., 1974).

The early records show that in 1950 there were 746 outdoor and 7 indoor patients. In 1995 there were 7575 out patients, and 1371 inpatients (Table 1). During these five decades, the total hospital attendance (and the

TABLE 1
ANNUAL AVERAGE ATTENDANCE

	1947	1950	1971	1995
Psychiatry	-	746	7,261	7,575
Dept. of Med.	41,468	51,006	63,340	1,01,219
Total Hosp.	1,22,262	1,57,690	3,78,615	4,05,424

attendance in the Department of General Medicine) increased by two or three times. Attendance in psychiatry however had increased by many more times. Inspite of these impressive figures, psychiarty continued to be the Cinderella of medicine.

Attempts at approaching the hospital administration for more facilities and more space usually met with the standard answer"Sorry! this is a general hospital and not a lunatic asylum."

In 1954, about twenty years after its discovery, 71 patients were treated with electroconvulsive therapy (Abrams, 1994). A total number of 1447 treatments were administered during the period 1950-1959, since then the annual average number of ECTs increased to reach maximum of 8629 during the 1970s, decreasing to less than that number during the 1980s and thereafter (Table 2).

Almost all ECTs were administered straight in the 1950s and the 1960s. Almost all ECTs administered during the 1970s and the 1980s were modified with muscle relaxants and pentothal (Table 2).

TABLE 2
ANNUAL AVERAGE NUMBER OF ECT TREATMENTS

	1950-59	1960-69	1970-79	1980-89
ECT	1,447	6,589	8,629	3,595
(ST)	814	5,577	-	-
(MODI)	48	571	ALL	ALL

I am not sure whether this change is for the better or for the worse. There is evidence that musculoskeletal complications occur in less than 1% of unmodified ECT treatment and are of little clinical significance (Tharyan et al., 1993). Complications of greater clinical mortality and morbidity such as cardiac arrest are significantly more with modified ECT, which also increases the likelihood of aspiration, arrhythmias, bronchospasm and apnoea (Tharyan et al., 1993; Abrams, 1994; Hash, 1976; Shukla, 1981).

Psychotropic drugs were introduced in the 1950's (Frankenburg, 1994). They revolutionized the management of psychiatric illnesses. ECT which had been the mainstay in the treatment of psyhosis up to this time now became disreputable. In some countries there was debate about legislature to ban its use.

In our country, most patients cannot be treated as quickly and as economically with drugs as compared to ECT. It is likely that ECT will continue to remain on the scene in India for many more years.

When chlorpromazine did become easily available, and was prescribed liberally, we received frequent emergency calls. Some patients who had been prescribed chlorpromazine, had developed spasms or dystonia. The doctor who was not familiar with the drug could not decide whether the patient had tetanus or a seizure or a drug reaction.

When I joined the department in 1959, cardiazol was still being used to induce convulsions. It was effective in resistant hallucinosis not responding to other treatments. Its mechanism of action was different from electrically induced convulsions. Cardiazol convulsions were clonic-tonic rather than tonic-clonic like electrically induced convulsions. 387 cardiazol treatment were administered during the period 1956-1959. These convulsions were not smooth. They varied in intensity ranging from a mild stun to severe bone breaking seizures. Titrating the dose of the drug was difficult. The patient experienced an intense fear during the interval between the intravenous injection of the drug and the onset of the seizure. There were frequent complications like dislocations and fractures and the treatment was discontinued in 1960 (Alexander, 1966).

An amusing incident comes to my mind. One day I received an urgent telephone summons from my chief, Dr. K.M. Masani. He was the head of the department at the J.J. Hospital

where I had worked for some time. Dr. Masani had received a message that, as the doctor who regularly administered ECTs was absent, he was asked to take over the ECT sessions on that particular day. Dr. Masani was trained in psychoanalysis. When I reached the ward he was in a state of panic. He asked me to do the needful as he knew nothing about ECT and had never used the treatment. "You mean you are only familiar with cardiazol, Sir ?" I asked him. "I don't know anything about cardiazol either" said Dr. Massani. "My patients receive a different kind of shock after my treatment is over, when I send them my bill for professional services" said Dr. Masani.

Another time, two patients had been prepared for treatment. One was to be treated with cardiazol while the other was to be interviewed using intravenous pentothal. I decided to do the cardiazol first and the pentothal afterwards and instructed the nurse accordingly. She handed over a filled syringe to me. I injected the drug fairly rapidly into the first patients's vein as cardiazol is supposed to be injected, and waited for the convulsions. I was horrified when the patient yawned, closed his eyes, became limp and stopped breathing. The syringes must have got mixed up. I had injected pentothal instead of cardiazol. Luckily, both the patient and the treating physician could be resuscitated, and recovery was uneventful.

In the 35 years that I have been practising psychiatry, nothing matches the successful development of psychopharmacological therapies, and nothing is as disappointing as the dismal attempts to advance psychoanalysis as a treatment procedure, barring a few exceptions. In practice, psychoanalysis is the most time consuming and expensive of all therapies. But it is reserved for those who are minimally ill or who are not ill at all. This is a remarkable phenomenon in medicine where the most complex treatment is reserved for the least severely affected patient (Shepherd, 1982).

I remember a patient who was assigned for analytical psychotherapy to a new house psysician in the department. The therapy was continued regularly five days in a week for the first year. The house physician was then promoted to registrar. The therapy continued regularly for the next year. The registrar was then promoted to the professional staff. The therapy continued as regularly as before. By this time the therapist was heavily into analysis and he went abroad for further studies. But the therapy continued, either by correspondence or by telephone. Eventually the therapist returned to Bombay. In the meantime the patient had expired. However the therapist insisted that therapy must still continue with the patient's wife or with his son-why, because the patient had died before the analysis was complete.

There is a popular belief that certain treatments like analysis, meditation or herbal medicine are safe alternatives, as they have no adverse effects like allopathic medicines. Nothing can be further from the truth. There are many recent reports about repressed memory therapy harming patients and devastating families (McElroy, 1995; Frankel, 1995; Jaroff, 1993). More than seven thousand individuals and families have sought assistance from the False Memory Syndrome Foundation in Philadelphia since its inception in 1992.

Shapiro has reported that 62.96% of 27 patients on long term meditation therapy suffered from adverse effects (Shapiro, 1992). These were disorientation, decreased attentional clarity, increase in awareness of negative qualities of others, increased discomfort with family and friends and alienation.

Herbal indigenous products are used by a large proportion of the population. They are advertised in the lay press and are freely available everywhere. They are not regulated by governmental food and drug agencies. Their safety and efficacy have not been proved. Most contain multiple ingredients. Many contain alkaloids and aflatoxins. They can cause a wide variety of hepatotoxicity ranging from mild hepatitis to hepatocytotoxic necrosis, cholestasis,

chronic hepatitis and cirrhosis (Koff, 1995).

Sternbach synthesized chlordiazepoxide in the 1950s and the drug became valuable in 1960 (Kaplan, 1993). Our hospital did not dispense anxiolytics free of cost. So carbon dioxide inhalation therapy was used for cathartic relief in anxious patients, who were verbally unable to express confictual, guilty and devious thoughts. This rather crude and cumbersome method of producing catharsis was gradually replaced in 1980 by modern and sophisticated chemical and verbal abreactive methods.

The insulin treatment which was practiced in our department was not the classical coma therapy as described by Sakel in 1937 (Sakel, 1937). Patients who were anxious and depressed and who had lost appetite and weight, were prescribed insulin in hypoglycemic sub-coma doses. They felt better and soon regained their weight.

The treatment was discontinued in 1980 as the same results could be achieved with anxiolytic and antidepressant drugs.

TABLE 3
ANNUAL AVERAGE NUMBER OF CO_2 TREATMENTS

	1950-59	1960-69	1970-79	1980-89
CO_2	1,608	6,204	1,501	148

TABLE 4
ANNUAL AVERAGE NUMBER OF INSULIN TREATMENTS

1950-59	1960-69	1970-79	1980-89
1241	4524	1988	798

The history of medicine is largely the history of the placebo (Shapiro 1968). Quite often a placebo effect is the single action which all drugs have in common. In some instances it is also the only useful action which medications exert (Modell, 1968).

The story goes that Dr. Rebello, the first appointed chief of dermatology at the KEM Hospital had observed that a large number of patients with skin problems were highly strung and did not need medication. He used to preach "Shanti" and advise them to try and relax. Once, a disgruntled patient complained that he had come to the hospital for treatment for his skin disease and not to listen to his "Lambi chori batt". He insisted that he must have some medication. Thereafter Dr. Rebello routinely prescribed an injection of normal saline or distilled water to these patients with excellent results. To avoid further confrontations he labelled this as "Injection 'R' Solution" ('R' for Rebello).

The psychiatry department adopted this treatment for some of its patients with equally gratifying results. Patients would come for these injections regularly every week from distant suburbs many kilometers away from Bombay.

The power of the placebo cannot be underestimated. Placebo effectiveness is variously reported in the literature ranging up to 69% and averaging out at 35.2% (Beecher, 1955; Dhume, et al., 1975; Diehl, 1933; Gorden, 1970; Green, 1964; Pogge, 1963; Shapiro, 1968; Wolf & Plinsky, 1954). At the same time placebos also have been reported to produce side effects which affect almost every organ system in the body (Meyer & Herxheimer, 1972; Meyer, 1968; Gorden, 1970; Shapiro, 1968; Beecher, 1962; Fisher, 1965; Honigfeldt, 1964). Addiction, habituation and even anaphylaxis have all been reported (Lealie, 1954; Vicar, 1969) (the lethal dose of placebo is not known). The consumer protection courts should have a field day trying to pass judgement on litigations involving the use of a placebo. It had been reported just recently that placebos and

acupuncture both act by releasing endorphins in the brain periaqueductal areas and the raphae (Tyrer, 1992; Levine et al., 1978; Levine 1979).

Psychiatry is an imperfect science. Unlike general medicine where you can diagnose pneumonia, isolate pneumococci and prescribe penicillin, the exact cause of a psychiatric illness, and the mode of action of a particular treatment may not be precisely known at a point in time. Quite often, what is well known today was not known at all yesterday, and will be obsolete by tomorrow.

This was psychiatry as I saw it in the 1960s.

PSYCHIATRY IN TRANSITION

BY this time I had seen a lot of psychiatry and I decided to see what psychiatry was like in other parts of the world.

I spent some years in the United States. When I returned to India in 1970, I noticed a great deal of change. Psychological medicine had come of age. It had gradually evolved into a modern and sophisticated neurosciences. I don't know about psychiatrists, but psychiatry had certainly become more respectable. It had gained in prestige what it had lost in terms of providing entertainment. Carbon dioxide therapy, insulin therapy and analytical therapy were amusing incidents from the past.

Psychiatry had also become too important subject to be left to psychiatrists. Now it was public property. It was the province of people who were in no way connected with it either as patients or as its practitioners. Unlike cardiology or neurology, every other person had his or her own views about psychiatry and did not hesitate to express them. The subject was talked about, discussed and criticised by people who were least qualified to do it. Amateur psychology magazines, health guides and self help books had almost as large a circulation as *Playboy* and *Penthouse* magazines.

Psychiatrists too were moving away from psychological medicine into territories which were already occupied by non-medical and paramedical professionals (Ghiselli, 1963; Reese, 1972; Levinson, 1977; Greenblatt, 1977; 1978; Schenke, 1986).

Some psychiatrists had become management *Gurus*. All of us spend some time in administrative and management duties. We have learnt to do this by common sense and by experience. None of us has a MBA degree. As a matter of fact the psychiatrist is ill-suited for management either by training or by temperament. His approach to a problem is through reflection and interpretation, while in fact what may be required is quick action and clear direction (Silver, 1989). This kind of switching of sides or loyalties may be all right for politicians at the time of national elections. But it is not all right for professionals, especially when the election are already over.

When I joined the department in 1959 very few post graduate students chose to specialize in the neurosciences. Now the situation is very different. Top ranking students compete for psychiatry residency post. Most of them pas the DPM, MD and DNB examinations with credit. Their competence and training is acknowledged at various teaching institutions in India and all over the world.

In 1947 the department had one visiting lecturer in psychiatry and one house physician. Today there are six psychiatrists on the professional staff and twenty two residents, apart from clinical psychologists, social workers and occupational therapists.

The academic image of psychiatry had also improved. Staff members presented papers at local, national and international conferences. Some members were actively involved with local and national scientific bodies. They held important positions as office bearers (Appendix 1). Many collaborative studies were planned and executed (Appendix 2). A large number of prestigious papers were published. Some of them are landmarks and are quoted in standard text books and monographs. (Appendix 3). All this was largely

due to the efforts of my distinguished teachers and predecessors Dr. Vahia and Dr. Bagadia. Staff members also received many distinctions, awards and prizes (Appendix 4). I myself received many surprises and very few prizes.

THE SACRED COWS OF PSYCHIATRY

I am aware of the rich variety of recent research findings in biological psychiarty. However, I am not very clear about their significance.

To recapitulate:

DOPAMINE SCHIZOPHRENIA AND ANTIPSYCHOTICS

The dopamine hypothesis states that schizophrenia is a hyperdopaminergic disease.

Dopamine agonists worsen the symptoms while dopamine antagonists prove them by blocking dopamine D_2 receptors (Mathyse, 1973). There are limitations to this theory.

1. In schizophrenia there is hypofrontality or hypofunctioning of the prefrontal cortex. This has been demonstrated with CBF measurements using the PET scan.

2. The psychotic symptoms associated with frontal lobe damage and the cognitive and motivational symptoms in Parkinson's disease are similar to the negative symptoms of schizophrenia e.g., apathy, avolition, flattened affect, etc. They may be due to low dopaminergic activity (DA).

3. In post-stroke patients, the low prefrontal cortical activity is associated with an increase in sub-cortical DA.

4. Lesions of DA neurones in the prefrontal region result in an increase in homovanillic acid (HVA), the DA metabolite in the striatal region.

5. Varying values of HVA ranging from low, normal to high have been reported in schizophrenia.

Therefore it is possible that cortical hypodopaminergia may be related to subcortical hyperdopaminergia (Table 5).

TABLE 5

DOPAMINE, SCHIZOPHRENIA AND ANTIPSYCHOTICS

	Cortex	Striatum
PET	↓ Hypofrontality	-
Symptoms	Negative Parkinson's Dis. Fl. lobe damage	Positive
DA	↓	↑
HVA	↓ ±	↑ ±
Drugs	Atypical D1,D4 5HT2, 5HT7	Typical D2
Site	Mesolimbic	Nigrostriatal

This can explain the concurrent presence of negative and positive symptoms and also the reported inconsistent HVA values (Davis et a.,l 1991; Davidson, 1988; Davila, 1988).

In so far as drugs are concerned, the limitations are :

1. Dopamine agonists do not induce the negative symptoms of schizophrenia, but only the positive symptoms.

2. Many schizophrenics are drug resistant.

3. Antipsychotics effectively treat almost all psychotic and agitated patients including manics and not only schizophrenics.

4. Clozapine and other atypical and highly effective antipsychotics have affinity for D_1 and D_4 and $5HT_2$ receptors unlike typical neuroleptics which have D_2 receptor affinity.

The overall conclusion is that there may be a dysregulation of DA transmission in schizophrenia rather than a hyper or hypodopaminergic state.

Both typical and atypical antipsychotics

exert a modulatory action by blocking DA receptors in different brain DA subsystems like the mesaocortical or the nigrostriatal system, (Lader, 1983; Civelli, 1993; Carlosson, 1990; Meltzer, 1996).

MONOAOMINES, AFFECTIVE DISORDERS AND ANTIDEPRESSANTS

The catecholamine, indoleamine and dopamine hypotheses of depression state that some depressions may be associated with absolute or relative deficiency of norepinephrine (NE), 5-hydroxytryptamine (5HT) or dopamine (DA) respectively (Randrup et al., 1975; Lapni & Oxenking, 1989; Bunney & Davis, 1965; Schildkrant 1965).

Drugs which increase or potentiate these brain transmitters may have antidepressant properties.

Tricyclic antidepressants (TCAs) or monoamine reuptake inhibitors (MARIS) increase synaptic NE by inhibiting its reuptake.

The selective serotonin reuptake inhibitors (SSRIs) increase synaptic 5HT by inhibiting its reuptake.

The implication that some depressions may respond to TCAs while some may respond to SSRIs is naive. There is data suggesting an earlier onset of antidepressant action with combined 5HT and NE reuptake blockade (Nelson et al., 1991). Further-more Venlafaxine, a potent new antidepressant is an SNRI. It inhibits the reuptake of both 5HT and NE (Feigner, 1994).

Tianeptine, a novel TCA, is an SSRE instead of an SSRI. It enhances the synaptic 5HT reuptake instead of inhibiting it, its action is therefore opposite to the action of SSRIs. (Wilde, 1995; Ayd, 1994).

Tianeptine also blocks the heightened HPA axis response to stress and prevents structural changes in the brain induced by corticosteroids (Wilde, 1995; Ayd, 1994).

Amoxapine is marketed as an antidepressant. It increases synaptic NE like a TCA and it has a DA blocking action like an antipsychotic (Ayd, 1994).

Amineptine, another TCA increases the release and reduces the reuptake of DA (Ayd, 1994).

Risperidone, atypical antipsychotic with both D_2 and $5HT_2$ receptor blocking properties has been used successfully in treating major depression and schizoaffective depression (Ayd, 1994).

Finally, clozapine a potent atypical antipsychotic which had both D_1, D_4 and $5HT_2$ to $5HT_7$ receptor blocking properties has been used to treat schizoaffective and psychotic mood disorders (Darsa, 1993; Baun, 1994; McElory, 1991; Ayd, 1994)

I believe that by this time most of you must be as thoroughly confused as I am. The treatment of depression involves drugs with a variety of mechanisms of action viz., those which increase 5HT, DA and NE, those which decrease 5HT and DA, those which block both D_2 and $5HT_2$ receptors, those which block both D_1 and D_4 and 5 HT_2 to $5HT_7$ receptors. And those which attenuate the stress response of the HPA axis (Table 6).

TABLE 6

MONOAMINES, AFFECTIVE DISORDERS AND ANTIDEPRESSANTS

	DA	NE	5HT	HPA
TCA/MAO		↑		
SSRI			↑	
SNRI		↑	↑	
SSRE			↓	↓
AMINEPTINE	↓ D2	↑		
AMOXAPINE	D1 D4 ↓		↓ $5HT_2$ $5HT_7$	

How can this be possible? Can drugs having diverse and even diametrically opposite modes of action produce the same end result?

One speculative answer to this question is "YES." It is possible because the isodendritic core of the brain stem which has extensive projections to various brain areas, comprises of dopaminergic, cholinergic, serotoninergic and noradrenergic neurons. Each neuronal system may be modulating the action of some other neuronal system rather than acting in isolation (Jeste et al., 1988).

The second answer to this question, which is probably more correct is "GOK" or "God Only Knows"; or better still "EGDNK" which means "Even God Does Not Know."

THE ANATOMY OF THE MIND

As if all this is not confusing enough, a number of neurobiological and neuroimaging studies have added fuel to the fire.

A neuroanatomical basis for panic disorders at the brain stem, limbic and prefrontal cortex areas is reported (Gorman et al., 1989).

Obsessive compulsive disorder has been associated with lesions of the caudate nucleus and orbitofrontal gyrus (George et al., 1992).

Anxiety is accompanied by arousal changes in reticular formation and frontal lobe, and increased CBF or hyperfrontality (Mathew and Wilson, 1990).

The dexamethasone suppression test which is reported abnormal in depression is also reported to be abnormal in hysteria (Tunea et al., 1996).

Schizophrenia is a brain disease with abnormal hemispheric asymmetry, increased lateral ventricular size, abnormalities in the left temporal lobe, and limbic structures, hypofrontality and changes in cerebellar vermis and festigial nucleus (Chua & McKenna, 1995; Shenton et al., 1992, Martin & Mathias, 1995, Mesulam, 1990).

Neuroanatomical observations in patients who develop an affective disorder after cerebrovascular disease have shown involvement of both cerebral hemispheres, and all four hemispheric poles viz.right anterior, right posterior, left anterior and left posterior, depending upon whether the affective disorder is manic or depressive, and whether the depression is primary or secondary, unipolar or bipolar (Starkstein, 1989; Bolla-Wilson et al., 1989; Jeste et al., 1988).

The structural changes reported in both schizophrenia and affective disorder show no significant differences between the two groups (Jeste et al., 1988). Neither are there significant differences in the CT scan indices or CBF values between the two groups. Several reasons have been cited for these findings (Jeste et al., 1988).

Frontal lobe dysfunction has been involved in many psychiatric disorders (David, 1992) e.g., personality disorders, obsessive compulsive disorder, schizophrenia, depression, catatonia, conduct disorders, manic thought disorders, anorexia, hysteria and delusional disorders (Gorenstein, 1982; Behar et al., 1984; Parfiit, 1956; Reading, 1991; Robinson, et al., 1980; Matters, 1991; Mc Grath, 1991; Benson, and stuss, 1991).

Even the concept of empathy or emotional interpersonal communication, a term which is so difficult to define, is hypothesised to have a neurological basis involving the right hemisphere, amygdala, hypothalamus, and the brain stem (Brothers, 1989).

This list probably includes most of the brain structures mentioned in Gray's anatomy. So far the cauda equina seems to have escaped.

What does all this mean? What is its significance?

I have reached an age when I am too old to be called young and too young to be called old, so I can afford to read the bottom line. And the bottom line is that we should know the differences between medical facts and medical myths. All new, speculative and sophisticated findings should be interpreted with caution. We must know what we do know and

what we do not know very clearly, with regard to new discoveries.

If this is not kept in mind, then in the future all of psychiatry will be medical, none of it will be psychological. And as Professor Eysenck had predicted physicians and neurologists will treat the psychotics, psychologists and social workers will treat the neurotics, and psychiatrists like dinosaurs will soon cease to exist.

THE FUTURE OF PSYCHIATRY

What about the future of psychiatry? The future of psychiatry is as controversial as its past.

On the one hand, psychiatry has a bright future. No one in the early forties could have predicted that in few years we would see the appearance of a variety of drugs which have almost emptied the beds in mental hospitals all over the world. Today the psychiatrist can pick and choose a drug from a mountain of medications which can modify almost every aspect of human behaviour. There are stimulants, sedatives, uppers, downers, drugs to make you sleep, drugs to keep you awake, drugs to turn you on and to turn you off, tranquillisers, antidepressants, mood stabilizers, mind expanders, memory enhancers and what have you.

Future discoveries and innovations whose nature we cannot even imagine will bring about still greater progress. Newer medicines, manipulations and measurements will increase our knowledge and add sophistication to our therapies.

On the other hand, the future of psychiatry faces two real dangers.

The first is the danger of the fractionation or decimation of psychiatry.

On the somatic side, psychiatry has already lost a large proportion of its patient population to other disciplines like neurology and internal medicine. Patients with dementia are now referred to neurologists for diagnosis, investigations and treatment.

But all are agreed about the need to preserve psychiatry's connection to medicine. This integration of psychiatry within the mainstream of medicine requires that the physician must be a psychiatrist and the psychiatrist must also be a physician.

On the psychic side, psychiatry is surrounded by practitioners of alternative methods of medicine. An encyclopedia of alternative medical practice lists more than 30 such methods of healing (Olsen, 1989). We have lay counsellors, hypnotists, psychic healers, astrologers, soothsayers, poojaris who use pagers, God men who may not be good men, sadhus and saints. We even have what is called sexologists. Some time back, sex was a pleasant pastime which involved a little bit of pushing and a little bit of pulling in the right places. Sexologists have changed all that. And today, you have to know the physiology and the biochemistry of human reproduction before you can experience an orgasm.

If psychiatry is to survive, then it is important that we psychiatrists create such a climate, that all these people who seek alternatives to psychiatry to be able to cope with their problems will want to seek help from us instead.

The second danger facing psychiatry is the danger of impersonalisation of medicine. At all times the doctor is the person on whom the patient and his family rely in times of physical or psychological distress. They know that he is there and is willing to listen with sympathy. Quite often this is the only treatment which is required. This traditional relationship between the doctor and the patients is unique, and medicine is no longer medicine if this relationship disintegrates.

Today conventional medicine is being replaced by computerized medicine. The doctor tends to be a technician. He is busy studying esoteric investigations and punching computer keys, while he expects someone else to talk, to listen and to relate to the patients.

In future societies, it is perhaps possible that psychiatry may not exist as a medical

speciality. But it is impossible that the traditional doctor can be replaced by a machine. If medicine does become progressively impersonalised, then the disintegration of psychiatry which is one of the youngest branches of medicine, will precede the disintegration of medicine.

If we wish to preserve psychiatry and clinical medicine for future generations,it is necessary that we make all possible efforts to be personally involved with the patient.

It is said that a good lecture must be like a woman's dress. It must be long enough to cover most of the subject, but short enough to be interesting. I have followed President Roosevelt's advice for my talk today. I have been sincere in what I had to say, I have tried to be brief, and it is time for me to be seated.

Ladies and gentlemen, this has been memorable day for me. I want you to know that it has been a singular honour to have been given the opportunity of addressing you today. Once again I thank the organizers of this conference for arranging this function, and I thank you all for gracing the occasion with your presence.

ACKNOWLEDGEMENTS

I am very greatful to Dr. Ashit Sheth (Retd.) Hon. Professr and Dr. Shubha Thatte, (Retd.) Clinical Psychologist, Department of Psychiatry, King Edward VII Memorial Hospital and G.S. Medical College, Bombay, for their invaluable help in the compilation of this paper.

The Dean's permission to use hospital statistics is acknowledged.

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Appendix 1

Office bearers

IPS President	Dr. N.S. Vahia
	Dr.V.N. Bagadia
IPS Secretary	Dr. L.P. Shah
	Dr. V.N. Vahia
	Dr. R. Abhyanker

Appendix 2

Collaborations

NIMH, U.S.A.	Tata Institute of Social Sciences
	Nirmala Niketan
WHO	Bombay University
ICMR	Tata Memorial Hospital & Research Centre
Harvard University	Thane Mental Hospital

Appendix 3

Publications cited in standard texts or monographs

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Appendix 4

Awards

Dr. D.L.N. Murti Rao Oration

Dr. D.R. Doongaji

Tilak Venoba Rao Oration

Dr. Rajesh Parikh

Dr. R. Abhyanker

Dr. A.K. Srivastava

Dr. M. T. Gada

Marfatia

Dr. D.R. Doongaji

Sandoz

Dr. D.R. Doongaji

Dr. N.S. Vahia

E. Merck Medal

Dr. D.R. Doongaji

Bombay Psy. Society Silver Jubilee

Dr. A. K. Srivastava

Dr. A. Singh

DINSHAW R. DOONGAJI, M.D., D.P.M. (Bom.), M.S. (Minn.), F.R.S.M., F.R.C. Psych. (Lond.), F.A.C.P. (Hon.), F.A.P.A. (Corr.). F.A.M.A., Retd. Hon. Professor & Head, Deptt. of Psychiatry, & Hon. Associate, Deptt. of Clinical Pharmacology, K.E.M. Hospital & G.S. Medical Colge, Bombay.