I am deeply conscious of the great honour that is bestowed by the Indian Psychiatric Society in selecting me to deliver the D. L. N. Murti Rao Oration. It is in the nature of giants to take giant strides where lesser mortals proceed in a pedestrian way. So it was that Dr. D. L. N. Murti Rao who strode through the beginning of Indian Psychiatry and the breath and vision of his personality is still felt by the large number of students who have strived to keep alive the shining example that he set, and the institute that he headed is even today one of the foremost academic centres in the country. I would like to discuss today what I believe to be a common clinical entity—the Emotional Distress Syndrome.

**NATURE OF STRESS**

The soldier who sustains a wound in battle, the player exerting himself to the full in the match, the mother worrying about her children’s health, the gambler watching the races, and the rider and horse are all under stress. The beggar suffering from hunger, the big businessman concerned about his next million and the doctor presenting a paper in front of an audience are all under stress. What is this mysterious thing that touches on everyone’s life, in fact it touches on the very essence of life and health and disease.

At this point I may mention the semantic confusion involved in the indiscriminate use of the word stress. The word stress is used to describe the specific agent responsible for the disturbance of equilibrium in the subject as well as to describe the state of organism or object which is subjected to stress e.g. stress fractures in bones, airplane bodies etc. Selye in the stress of life (1965) suggests that the word stress should be used to designate the sum of all the non-specific effects of various factors acting on the body whereas these agents should be referred to as stressors when we refer to their ability to produce stress. He describes three stages in the stress syndrome—

(a) the initial response or alarm reaction. During this stage the organism is on full alert with marked increase in output of adrenal cortical hormones. Since no organism can continue to be in a state of alarm indefinitely if the noxious agent is very damaging, death may occur within a few hours or days. However, in most cases the organism brings into operation certain defensive measures to cope with the stress this he referred to as—

(b) the stage of resistance. In case the exposure to stress is continued, this adaptation progressively fails leading to the third—

(c) stage of exhaustion. The totality of this non-specific response pattern is referred to as the “General Adaptation Syndrome”, hence he defines “Stress is a state manifested by a specific syndrome which consists of all the non-specifically induced change within a biologic system”, hence although it has certain common specific manifestation, it is non-specifically induced by a variety of causes. This is in contrast to the local adaptation syndrome in which a specific noxious agent produces a localized adaptive responses which is specific for that particular noxious agent.
The Nature of Anxiety

Any hypothesis about the origin and development of such a complex and universal phenomenon of the human condition as anxiety, has a very close relationship with the particular ideas of the author about human nature with the prevailing scientific and philosophic spirit of the times. Our concepts are formed, partly by reflections about ourselves and at the same time about the human condition in general. Reflection is to be understood in its deeper anthropological meaning which is evoked in the following words of Romano Guardini (1954). It is characteristic of human life that man can once again overcome the immediate context of his existence and the possibility of gaining a distance to consider is "reflection". Reflection is first of all directed towards that which is most disturbing, what Saint Augustine called the 'enigma of life' viv. "From whence I came here, this, which I call life—that dies, and death that lives."

Anxiety thus occurs at the point when some emerging potentiality faces the individual. Some possibility of fulfilling his existence, but this very possibility involves the destroying of the present security, thus often giving rise to the tendency to deny the new potentiality. In relation to the world a person is exposed to new possibilities, hence to feelings of anxiety, as long as he is alive (The world as used here refers to the structure of unmeaningful relationships in which a person exists and the design in which he participates). The world is not limited to the past experiences but also includes all the possibilities which open up before the person in the future. The world is not something static something 'here' which the person then 'accepts' or 'adjusts to' but rather, it is a dynamic pattern which so long as I possess self-consciousness, I am in the process of forming and designing. The existentialist distinguishes three modes of this world (a) the Umwelt meaning the biological and environmental world around us (b) the Mitwelt—refers to the world of beings like oneself—the world of fellow men and women and (c) The Eigenwelt—literally the own world—the mode of relationship to oneself.

The difference between this normal existential anxiety and the clinical anxiety has not received any attention in the literature. Briefly, existential anxiety is the result of awareness of the contingency of human existence. The confirmation of the lack of meaning of life leads the authentic man to search for a meaning—his never being able to rest, or be at peace, is at the core of existential anxiety and is seen mainly in relation to the self i.e. eigen welt. However, clinical anxiety is characterised by an inability to cope with stress, in the outer world i.e. the unwelt and mitwelt.

Nature of Disease

24 centuries ago, Hippocrates, the Father of Medicine told his disciples that disease is not only suffering (Pathos) but also toil (Ponos), that is, the fight of the body to restore itself towards normal. In other words disease is not mere surrender to a noxious agent or stress but a fight for health and unless there is a fight, there is no disease but simply death. Claude Bernard in France and Walter B. Canon at Harvard described this tendency of the body as an attempt of the body to maintain a constancy of their internal milieu i.e. to maintain homestasis. Nearly 80 years ago, Freud the Father of modern psychiatry and Psychoanalysis also came to the same conclusion with respect to mental disorders the disease as seen clinically was a manifestation mainly of these defensive ego mechanisms i.e. the fight for survival and health.

The similarity is obvious—physical or emotional stress produces certain changes in the body which are signs of damage but these are also quite characteristic manifestations of the body's adaptive reactions which together constitute the symptoms of the disease.

Our studies of the normal grief reaction among parents who have suddenly lost their child (a severe degree of distress) also showed
THE EMOTIONAL DISTRESS SYNDROME

a relatively uniform pattern of emotional disturbance going through a) an initial stage of shock or emotional numbness lasting a few hours to a few days and followed b) by a stage of marked anxiety, sadness and hopelessness. (c) this is then followed by the stage of resolution and readjustment. (d) In some cases a residual state of sadness may persist. Thus at the psychological level this common response to stress may be viewed as a non specific universal response, varying in nature and severity with the degree of stress and the adaptive capabilities of the individual, akin to the general adaptation syndrome of inflammation seen at the physical level, and termed as Emotional Distress Syndrome. This E. D. S. or grief reaction was seen to be a well defined and uniformly predictable pattern, and in almost all cases, clinical recovery and return to normal functioning occurred within a period of 6 month (Singh and Tewari, 1980; Singh et al., 1989), (Table I).

Only very recently the Federal Centre for Disease Control in the U. S. A. in 1987 has formally defined the C. F. S. or Chronic Fatigue Syndrome. In 50% of these patients severe physical and or mental stress is a major source of their problem. According to Dr. Richard Poddol—"They have too many demands in their lives and cope by being tired." When obvious stress is not apparent, most experts suspect underlying psychological and emotional problems. A study published in 1988 in the Archives Internal Medicine found that out of 100 patients with C. F. S. the vast majority (64%) had a psychiatric diagnosis, primarily anxiety and to lesser extent depression. Only 5 patients had a medical condition to explain their exhaustion and fatigue and no explanations could be found in 31 patients.

Most of these patients are clinically labelled as suffering from anxiety or depression, but according to Dr. Poddol most of them are aware of the reason for their excessive fatigue or depression. Roughly half, stress that they are primarily feeling listless and tired and not depressed. He is often tired, unable to enjoy social or sexual activity or even family events but is not necessarily depressed although some of them may ultimately develop feelings of sadness and hopelessness. These persons who are suffering with symptoms of anxiety and depression in the face of stress, in my opinion can not be labelled as suffering from a disease because of the absence of the ponos or fight. Clinically they resemble subjects in the second stage of grief reaction. It is only after the stage of resolution that we can label the person as suffering from anxiety or depression as a disease entity.

PREVALENCE OF ANXIETY NEUROSIS IN GENERAL POPULATION

Although an estimated 5% of the general population suffer from anxiety neurosis, there is little data concerning the range or degree of distress resulting from the disorder and its long term outcome (Noyes, 1976; Blair et. al.,

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**Table 1. The Emotional Distress Syndrome**

|   | A. General Adaptation Syndrome (Selye) | B. Grief Reaction in Children (Bowlby) | C. Emotional Distress Syndrome (G. Singh) |
|---|--------------------------------------|---------------------------------------|----------------------------------------|
| 1. | Alarm Reaction                       | 1. Initial Stage of shock or disbelief | 1. Initial Stage of shock or disbelief  |
| 2. | Stage of Resistance                  | 2. Stage of Despair                    | 2. Stage of anxiety, sadness, withdrawal |
| 3. | Stage of Exhaustion                  | 3. Stage of Detachment                 | 3. Stage of active resistance and resolution |
In a 6 year follow up study by Noyes et al. (1980) on 112 anxiety neurotics and 110 surgical controls, found a generally favourable outcome in about two thirds of all patients, 29% were symptom free and another 39% reported only mild symptoms. These together constituted 68% with a favourable outcome. This is similar to the figure of 67% reported in an earlier study and 50% reported by Greer et al.

More interestingly 22% of their surgical controls also met the study criteria for anxiety neurosis, suggesting that the actual prevalence of anxiety neurosis, is much higher than the 5% figure usually cited or they are not really cases of anxiety neurosis but simply a state of emotional distress. Community surveys such as the one conducted in Framingham tend to support this possibility. 21% of the residents of this community were identified as having anxiety neurosis alone.

RELATIONSHIP OF ANXIETY AND DEPRESSION

D.S.M. III differentiates between generalized anxiety and panic disorder in both of which there is marked over anxiety, and also between phobic and obsessive-compulsive disorders-in both these conditions, the patients goes to great lengths to avoid experiencing the anxiety related to the dreaded object or situation. Similarly, D.S.M III recognizes a number of depressive disorders based on such considerations as severity and whether or not there is a history of manic or hypomanic attacks. Dysthymic disorder-literally dysthymia means "ill humored with an indication to melancholy." Hence it has been used to include all unpleasant affective states including depression, anxiety and obsessional traits. These divisions are not yet based on firm scientific data which would convincingly prove that they have any clinical, therapeutic or prognostics import (Marks, 1987).

PSYCHOPHYSIOLOGICAL PARAMETERS

Let us briefly look at the psychophysiological parameters whether they are able to distinguish between anxiety and depression. Lader at al. (1967) showed that patients with anxiety states showed high levels of electrodermal activity and slow habituation of conductance responses to repeated auditory stimuli, when depressives were divided into subgroups of retarded and agitated depressives similar differences in electrodermal activity emerged (Lader and Wing, 1969). The retarded group showed almost no electrodermal activity while the agitated depressives were very similar to the anxious patients.

Investigating evoked potentials in depressed and anxious patients, Shagass (1955) applied photic stimulation at 10 and 15 H Z and measured cortical evoked responses in normals, anxious patients, neurotic depressives and psychotic depressives. The responsiveness was highest in the anxious patients and lowest in the retarded psychotic depressive, with normal and neurotic depressives in between.

CLINICAL FEATURES OF ANXIETY AND DEPRESSION

The symptoms and signs of these two conditions can be divided into three classes:
1. Symptoms that are specific to anxiety
2. Symptoms that are specific to depression
3. Signs and symptoms which are common to both syndromes.

Although the clinical relationship of anxiety and depression still remains a subject of controversy in psychiatric practice. A majority of workers in recent years have pointed to the fact that mixed anxiety-depressive picture is the most common manifestation in primary care populations although by force of habits we artificially tend to label them as primarily anxious or primarily depressed. Nuttinger and Zepotoczky (1985) found 83% of patients with panic disorder also received
a diagnosis of depression. Shehan and Shehan (1982) reported 92% of his patients with panic disorder as having a typical depression, while the Newcastle group (Gurney et al., 1972; Roth et al., 1972; Shapira et al., 1972) reported that 65% of patients with anxiety disorders had episodes of secondary depression. In addition a considerable number of family studies suggest a casual relationship between anxiety and depression particularly in panic disorder, agoraphobia and obsessive-compulsive disorder (Cloninger et al., 1981; Dealey et al., 1982; Munjack et al., 1981; Crowe et al., 1982; Weissmann et al., 1984; Lockman et al., 1983; Matuzas et al., 1983; Sargent, 1962; Pollitt and Young, 1971 and Shehan; et al., 1980).

ASSESSMENT OF ANXIETY AND DEPRESSION

Another conceptual and methodological problem is the fact that most instrument designed to measure anxiety or depression give a single global score. Yet we know that neither anxiety nor depression is a unitary concept. In fact each is composed of a number of behavioural, psychophysiological, biochemical, cognitive and emotional components which are only imperfectly correlated. The nature and degree and correlation between anxiety and depression therefore will vary greatly, depending upon the specific component being investigated.

The most commonly used scales are:

(I) Doctor Rating scale :
1. Hamilton Anxiety Scale
2. Hamilton Depressive Scale
3. Beck Depressive Inventory
4. New Castle Anxiety-Depressive Scale
5. Physicians Questionnaire

(II) Self Rating Scales
1. Hopkins Symptom Checklist and S.C.L. 90.
2. Profile of Mood States
3. Taylor Manifest Anxiety Scale
4. Zung Self Rating Scale
5. Amritsar Depressive Inventory.

Clinical studies using various self report scales and clinical diagnostic criteria eg. Dinardo et al. (1983) Barlow (1986) Johnstone (1980), Mendels (1972) Gurney et al. (1972) Singh and Sharma (1988) all reported inability to distinguish pure anxiety from depressive neurosis.

In our attempt to develop a simple instrument to measure depression, we finally developed the 30 item Amritsar Depressive Inventory after every effort was made to exclude items that showed a correlation of 0.3 or more with anxiety. In spite of this the normals score up to 5 on this scale. Patients of anxiety neurosis score up to 10, and the cut off point for diagnosing depression is 15 and for severe depression 20. Thus it is not possible to clearly distinguish anxiety and depression when present in mild degree.

Clancy et al. (1979) in a follow up study reported that the most common complication of anxiety neurosis appears to be depression which developed in over half of their patients. The same conclusions were reached by Kendell (1974) and Noyes (1980) who found 44% of 112 anxiety neurotics showed a potentially serious depression when re-interviewed after six years.

USEFULNESS OF DOCTOR VS PATIENT SELF RATING SCALES

The issue of relative value of using doctor rating scales versus patient self rating scale has been a subject of much controversy (Derogatis et al., 1970; Park et al., 1965; Prusoff et al., 1972 and Keller et al. 1977). Jacobson et al. (1977) carried out a multi-centred study at three centres at Florida, Massachusetts and Pennsylvania. The three populations did not differ in terms of socio demographic variables. The relative severity of psychopathology for the three populations was examined separately for both
physician ratings and patient self ratings. The baseline symptom profiles as determined by the H.A.S. and H.D.S. indicated marked difference between the three populations. These differences were statistically significant at 0.05 level for six items between Floridra and Pennsylvania groups and on 10 items between Floridra & Massachusetts group.

In marked contrast to the physician ratings, the comparisons using patient self rating scales showed no significant difference in the three populations except for 1 item (Depression dejection factor). Similarly in the 56 item S.C.L. again showed a marked similarity in the symptom profile between the three populations with no significant differences between the three groups. On the average 74% of doctors ratings indicated significant differences in pathology between the populations as compared to only 13% for patients ratings.

In a very large collaborative study supported by N.I.M.H. and John Hopkins, Baltimore and published by Covi et al., 1984; Downing et al., 1981; Kahn et al., 1981 and Lipman et al., 1981, 240 primary anxious and 424 primarily depressed patients were studied using the S.C.L. 90 which measures 9 symptom clusters.

Despite the statistically significant differences in level of distress on three dimensions of symptomatology the overall pattern of profile was very similar in the two groups. Indeed, were it not for the differences between the groups on the relative rank ordering of anxiety (1.5 in anxiety group versus 6 in depressive group) and depressive (4 in anxious group versus 1 in depressed group) the symptoms correlations in ranks would be almost identical. Clearly then there is a remarkable similarity of symptom profiles among the anxious and depressed patients attending the outpatient departments.

USE OF FACTOR ANALYSIS TO DISTINGUISH ANXIETY AND DEPRESSION

In a recent study by Mountjoy and Roth (1982) a total of 117 patients meeting the inclusion criteria as suffering from anxiety (74) depression (43 patients) were administered Hamilton Depressive Rating Scale, Gelder and Marks scale for agoraphobia and social phobia and the self rating scale for anxiety (Lipsedge, 1971) and modified Zung self rating scale for depression and the New castle Anxiety—Depressin Scale (Burney, 1972).

A principal component analysis of the total initial scores of all the rating scales yielded two factors. Factor 1 was a general factor of severity as the loadings of all the scales on this were positive and it accounted for 43% of the variance. There were no statistically significant difference between the two diagnostic groups in their mean scores on this component.

The second component was a bipolar factor which accounted for 21.3% of the variance. Plots of patients scores showed more anxious patients at one end, more depressives at the other with some overlap in between. Depending on the cut off point the misclassification rate in relation to clinical diagnoses was around 33%. The most powerful discriminators were the Hamilton Depression and Hamilton Anxiety Scales in one direction and the anxiety phobic scales on the other, while both the anxiety and depression self rating scales were totally nondiscriminating.

Similarly Zubin and Fliess (1970) carried out a review of all earlier factor analytic studies using self report and doctors rating scales and concluded that it was difficult to arrive at separate anxiety and depressive factors.

A study carried out in our department (Singh and Sharma, 1988) on a group of 30 patients each of generalized anxiety disorder, dysthymic disorder and mixed anxiety depression, the clinical symptoms were recorded as per DSM III criteria, factor analysis by varimax rotation was carried out and evaluation was done by using doctor rating scales viz. HAS and HDS and two patient self rating scales.
THE EMOTIONAL DISTRESS SYNDROME

scales viz. TMAS and ADI, yielding five factors—Factor DI, this factors had high loading on depressed mood, feelings of worthlessness and suicidal ideas (0.864) but also has fairly high loading on anxious mood (0.54). The second Factor (P Factor) was constituted by generalized fatigue, aches and pains, palpitation, breathlessness etc. This was called the physiological arousal factor and interestingly was correlated with high scores on HAS (0.641) and TMAS (0.51) suggesting that this accounts in large measure for syndrome of anxiety as measured by these tests. This along with the psychic anxiety factor (Factor-III) and somatic anxiety (Factor-IV) correlate well with HAS-Factor-III represents psychic anxiety (HAS 0.417) and factor IV somatic anxiety HAS (0.448).

Although Factor I Depressive Factor covers most of the symptoms of depression but also includes Anxiety—the main symptom of anxious mood is part of the Depression factor DI while Psychic anxiety and somatic anxiety are separate factors which correlate strongly with these symptoms on HDS but not with HAS.

Thus it is clear that all these standardized rating scales which were ostensibly developed for the purpose of detecting and grading either anxiety and/or depression, have in each instance, items descriptive of the other type of emotional change and are therefore heterogeneous rather than unidimensional in content. Scales such as the Hamilton depression and anxiety scale which have proved clinically to be the best discriminators also contain the widest span of items relating to a general emotional disorder, both the psychic anxiety and somatic anxiety components of the HDS have a very strong correlation with the anxiety syndrome.

What stands out from this is that the self rating scales which are the least likely to be contaminated by the observers preconceptions prove unable to differentiate between anxiety and depression which the observer rating scales do quite well suggesting that the observers preconceived notions are highly responsible for this difference. However a counter argument often raised is that the differentiation between the emotional states of anxiety and depression require a certain degree of introspection and linguistic sophistication which the patient may be unable to clearly formulate. To my mind this objection is not very convincing since it is possible that he is not suffering either from anxiety or depression which are clinical entities created by psychiatrists but is basically describing a state of dysphoria with varying levels of anxiety and depression.

EFFECT OF DRUGS IN TREATMENT OF ANXIETY AND DEPRESSION
(Is the drug effect anti anxiety or antidepressant)

The question whether drugs used to treat patients with mixed anxiety and depression really effect the anxiety or depression or both is a very relevant question. It is commonly observed that symptoms of both anxiety and depression improve simultaneously, but many authors feel that the main effect is on depression and the relief of anxiety is secondary to this antidepressant effect. Another possibility is that both anxiety and depression are symptoms of a unitary underlying disorder and the drugs act on this basic disturbance resulting in improvement of both.

Overall (1977) using data from four double blind studies of low dose phenothiazines

| Treatment Group Measured | Phenothiazine | Placebo |
|--------------------------|--------------|---------|
| Change score on anxiety factor | 4.15 | 2.97 |
| Change scores on depression factor | 4.15 | 2.23 |
| Anxiety change score adjusted for depression change | 1.94 | 1.78 |
| Depression change score adjusted for anxiety change | 0.94 | 0.98 |
versus placebo in the treatment of anxious-depressed patients—factor analysed the data. The results show a highly significant difference between drug and placebo in producing improvement in both anxiety and depression. Analysis of co-variance was then applied to see whether change in one was effected by improvement in the other.

The analysis of change in anxiety with change in depression and vice versa revealed no significant difference between drug and placebo. It is thus clear that in anxious and depressed patients, phenothiazines have no effect on anxiety that is separate from that on depression and vice versa and therefore probably it acts on a common underlying psychopathology.

In a study carried out in our department (Sharma and Singh, 1984), the effects of imipramine and diazepam was studied on 30 patients each of generalized anxiety disorder, dysthmic disorder (as per DSM III criteria) and 30 patients of mixed anxiety depression. Evaluation was done using two doctor rating scales viz. HAS., and H. D. S. and two patient self rating scales viz. Taylor Manifest Anxiety Scale and Amritsar Depressive Inventory.

Both drugs were found to be equally effective in reducing anxiety in all groups (62.8% and 62.2% by imipramine and diazepam respectively). Imipramine was significantly better in reducing the symptom of depression while diazepam was significantly better in reducing fear. None of the other symptoms was discriminatory. Our findings thus support the suggestion of Akiskal (1984) that mixed anxiety-depression is the most common dysphoric mood state and responds equally well to antidepressants as antianxiety agents.

The patterns of antidepressant drug effects are of considerable interest. Apart from their established role in treatment of severe depressions, they have been reported to be effective for treatment of phobic and obsessive compulsive phenomenon (Ananth, 1980; Insell, 1982; Marks, 1980; Sheehan et al., 1980). It is relevant here, that in anxiety disorders, too, imipramine was more effective than chlordiazepoxide or placebo in reducing anxiety as well as depression (Kahn et al., 1981; Singh et al., 1986). In patients of mixed anxiety-depression and related syndromes the few studies that report measures across a broad spectrum of different symptoms, antidepressant drugs seem to improve both in a pattern of a general patholytic effect e.g. Sheehan et al. (1980) found that imipramine and phenelzine patients improved significantly on every measure, regardless of whether it concerned phobia, anxiety, depression, hostility, interpersonal sensitivity, somatic and obsessive compulsive symptoms. Marks et al. (1980) reported similar findings of a general pathalysis for rituals, depression, anxiety and social adjustment.

Given the general patholytic effect of antidepressant drugs it may be misleading to concentrate on one or two aspects eg. panic anxiety attack or depressive features as their central focus of action because these drugs also block a number of other symptoms which all intercorrelate highly to the order of 0.6 or more, it is illogical to accept any one of these features as primary unless and until detailed analysis has shown that change in that feature precedes change in the other features. This has not been done, one predictor of drug effect seems to be the starting level of dysphoria—drug effects being best when this level is high and being absent when it is low. This is shown clearly in a comparison of some studies reported in the literature (See Table II and Table III).

It is suggested that there may be a minimal threshold of dysphoria below which a drug effect is unlikely. The potential importance of this point is testified by the study of Marks (1980) who found a strong correlation between initial depression and outcome in a group of obsessive compulsive patients when the top and bottom quartiles were compared, but
TABLE II. General Patholytic Effect of Imipramine

|                  | Imipramine effect | Imipramine X Depression | Imipramine X Phobic Severity |
|------------------|-------------------|-------------------------|-----------------------------|
| Agoraphobia      | .05               | .05                     |                             |
| Phobic anxiety (SCL) | —                 | —                       | .05                         |
| Panic            | .05               | —                       |                             |
| Tension-anxiety (POMS) | .1               | —                       | .05                         |
| Depression (SCH-Self) | .05            | —                       |                             |
| Depression (SCL-Dr.) | .05            | —                       |                             |
| Anger (SCL-Self)  | .001              | .05                     | .001                        |
| Anger (SCL Dr.)   | .05               | —                       |                             |
| Somatic (SCL-Self) | .1               | .1                      | .01                         |
| Global improvement | —                | —                       | .05                         |

.05, .01, etc.—Significant on ANCOVA — Not Significant

TABLE III. Outcome Versus Initial Depression Score

| Drug Effect Moderate | Initial Mean Depression Score | Mild Absent |
|----------------------|-------------------------------|------------|
| Present              |                               |            |
| Sheehan et al. (1980)| Tyner et al. (1973)           | Zitrin et al. (1980) |
| Mcl Nair & Khan (1984)| Zitrin et al. (1984)         |            |
| Pecknold et al. (1980)| Mavissakalian &               |            |
|                      | Michelson (1982)              |            |
| Ananth et al. (1980) | Gittelman-Klein &             |            |
|                      | Klein (1971)                  |            |
| Marks et al. (1980)  | Insel et al. (1982)           |            |
| Thoren et al. (1980) |                               |            |
| Absent               | Marks et al. (1982)           |            |
|                      | Solyom et al. (1981)          |            |
|                      | Marks et al. (1980)           |            |
|                      | Thoren et al. (1980)          |            |

This effect disappeared when it was analysed for the entire sample.

Johnstone et al. (1980) were not only unable to find any difference in response to amitryptiline with or without benzodiazepines, between anxiety states and depressive disorders, but found it impossible to differentiate clinically between these two syndromes by any definable criteria.

From the above review it is clearly evident that anxiety and depression are neither clearly defined nor distinct entities either in terms of their clinical phenomenology, in their course, outcome or response to treatment. Further, they can not be distinguished as such by patients when asked to rate whether they are suffering from anxiety or depression using self rating scales. Factor analytic studies also confirm that anxiety symptoms invariably have their highest loading on the depressive factor while psychic and somatic anxiety constitute separate factors. The same is
true when we look at the numerous controlled studies of treatment of anxiety and depression with antianxiety, antidepressant or phenothiazine drugs. In all cases there is no significant difference in overall response between the different diagnostic groups except that antidepressants are more effective in alleviating the symptoms of sad mood, while anti-anxiety drugs are more effective against symptoms of fear.

**DISCUSSION**

What then is this entity that the psychiatrists are easily able to identify and label as anxiety neurosis but which the patient himself is unable to pinpoint. To my mind this is a historical artifact like Kraepelin's dichotomy of schizophrenia and MDP. What we are seeing is in fact a condition of 'dysphoria'—something similar to what Lopez-Ibor refers to as anxious thymopathy, but which I would prefer to term as the Emotional Distress Syndrome.

This E.D.S. which could result from various causes should be seen as a non specific reaction of the individual to emotional conflict or trauma at the conscious/psychic level, just as inflammation of the body occurs as a response to any noxious agent or injury at the physical base. This can then be said to be the mental equivalent of the General Adaptation Syndrome as described by Selye. If this is so, then it is logical to assume that this EDS will be equally responsive to all thymopathic/ or antidysphoric drugs—including antianxiety and antidepressant drugs—which act through the central neural sites which are also concerned in the perception and expression of emotions, it is then important and possible to distinguish, the symptomatology and treatment of nonspecific stress resulting in the emotional distress syndrome which would be characterised by three clusters of symptoms a) mood changes—anxious or depressed b) physical symptoms affecting any organ or system, c) change in behaviour.

The Emotional Distress Syndrome would thus replace the nonspecific term of generalized anxiety and include all cases of mixed anxiety depression, thus doing away with the artificially created anxiety—depression dichotomy within the broad area of emotional distress. This I believe would lead to more definitive research on the causes, clinical manifestation and treatment of these non-specific emotional disorders in terms of the specific nature of psychological and social stressors that produce these specific defense mechanisms.

Finally I would like to suggest that all anxiety is not necessarily neurotic. As I have tried to bring out in this presentation every human being as long as he is alive is suffering from what I have referred to as the normal existential anxiety in relation to his inner self, and is to be distinguished from this adaptive human response to environmental stress and manifesting as this emotional distress syndrome.

Only when this adaptive process fails and the individual develops a chronic residual state characterised by one or more maladaptive patterns of behaviour should the person be said to have a neurotic disorder (See Table III).

An understanding of the individuality of each person is essential if we are to understand the factors that predispose one person and not another to some specific clinical disorder as well as the particular event which proves noxious, in that it seems to precipitate the disorder at this time and not some other time. Perhaps in the future, research will help to identify the persons predisposed to specific stress and to enable us to prevent the subsequent break down.

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