Respected Chairperson, distinguished guests, members of the Indian Psychiatric Society, ladies and gentlemen. It is a matter of great privilege and honour for me to deliver the Tilak Venkoba Rao Oration on the occasion of ANCIPS 2000. I am highly grateful to the Indian Psychiatric Society for bestowing me with this honour. As you know, this oration was instituted by Prof. A. Venkoba Rao and Prof. Parvathi Devi in the memory of their beloved son Tilak Venkoba Rao who lost his life in prime youth. I pay my humble respect to late Tilak. I would also like to pay my respect to my teachers, Prof. V.K. Varma, Prof. P. Kulhara and Prof. Savita Malhotra, from whom I learnt psychiatry and basic principles of research. I am grateful to my earlier colleagues, Dr. M.S. Bhatia and Dr. Sabita Shome, who were also involved in my research on somatisation. I would also like to express my gratitude to the late Prof. Z.J. Lipowski, whose review paper on somatisation in Nov., 1988 issue of American Journal of Psychiatry initiated my interest in somatisation, the topic I have chosen for this oration.

Patients presenting with physical symptoms without any organic basis have always remained an enigma for the physicians. These patients have been recognised for more than two millennia. The illnesses have received a wide variety of names like hysterike pnox by Plato in 427 BC, hysterika given by Galen in the second century AD, English malady by Cheynes in 1789, neurasthenia by Beard in 1868, conversion hysteria, hypochondriasis, psychosomatosis, etc. The term 'somatisation', which was introduced by Stekel in 1911 for the illness, has become quite popular in the last two decades, especially after the formal recognition of somatisation disorder as a diagnostic entity by DSM-III in 1980. Somatisation can be defined as a tendency to experience and communicate somatic distress and symptoms in response to psychosocial stress, unaccounted for by pathological findings, to attribute them to physical illness, and to seek medical help for them (Lipowski, 1988).

Somatisation is neither a discrete clinical entity, nor the result of a single pathological or psychological process, and cuts across diagnostic boundaries (Kellner, 1994). It is not an equivalent of somatisation disorder or somatoform disorders. The patients usually present in primary care settings and to physicians in general hospitals. They are often misdiagnosed and wrongly treated as suffering from physical illnesses. The phenomenon is responsible for significant distress and disability in the sufferers. It is estimated that a large part of physicians' time and effort is spent on investigating and treating somatising patients and between 10-20% of the medical budget is spent on patients who somatise or have hypochondriacal concerns (Ford, 1983; Kellner, 1991).

In this oration, I shall be discussing about evolution of the concept of somatisation, clinical presentation of somatisation, related nosological issues and biological research in somatisation.

EVOLUTION OF THE CONCEPT

Stekel (1943), who introduced the term 'somatisation' in scientific literature, described it as the process by which neurotic conflicts may present themselves as a physical disorder. Meninger (1947) described 'somatisation reactions' as the visceral expression of anxiety.
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that may in this way stay unconscious. Thus, initially the term originated in psychoanalytic frame of reference. Different dynamic explanations continued to be given by different workers in the next twenty years. In 1955, Schur explained it in terms of ego regression, which by primary process thinking may result in the ego's incapacity to neutralize aggression, and in turn leads to resomatisation of responses. Kaufman described it as regressed libidinization of an organ or organ system or fantasy of function of an organ system manifesting in symptoms or signs of somatisation. Rubins defined the process of somatisation as the 'psychological mechanisms through which the various personality elements, the idealization and rejection of attitudes and self-aspects, and body-image distortions get translated into somatic symptoms' (van der Feltz-Cornelis and van Dyek, 1997).

In contemporary psychiatry, somatisation is described more in clinical terms without going into a hypothetical actiological explanation. No presumptions are made about its having a biological or psychological basis. Katon et al. (1984) describe it as an idiom of distress in which patients with psychosocial and emotional problems articulate their distress primarily through physical symptomatology. It is an expression of personal and social distress in an idiom of bodily complaints with medical help seeking (Kleinman & Kleinman, 1986). Lipowski's definition as given earlier is also somewhat similar. The term 'medically unexplained symptoms' has also been suggested as a substitute (Mayou, 1993), but this has less theoretical implications than the term somatisation (van der Feltz-Cornelis & van Dyek, 1997). It needs to be emphasised here that somatisation should not be taken as a diagnosis, because this would limit attempts to reach a more complete understanding of the etiology and prognosis (Stewart, 1992; Sensky, 1994).

It may be argued that somatisation is a residual category covering symptoms that can't be ascribed solely to the body, as in the case of neurasthenia or solely to the mind. The model behind somatisation is that the mind influences the body. A link between biological and psychological symptoms is presumed without having specific knowledge about the nature of these influences (van der Feltz-Cornelis and van Dyek, 1997). Somatisation can occur in a diversity of psychiatric disturbances, but may also exist independently as a transient phenomenon. It both involves cognition and behaviour, but may be considered a fallout product of the conceptual gap between body and mind in the contemporary conceptualisation of disease.

CLINICAL PRESENTATION OF SOMATISATION

Somatisation symptoms can involve any body area or system. Pain involving different body parts, cardiopulmonary, gastrointestinal and neurological symptoms can occur alone or in different combinations (Lipowski, 1988; Srinivasan et al., 1986). In our study, subjective weakness, headache, vague somatic sensations, pain in extremities and palpitations were the five commonest symptoms reported by somatising patients. Symptoms of subjective weakness, headache, pain in extremities and pseudoseizures were more common in female patients than in males, whereas palpitations were more common in male patients compared to females (Chadda et al., 1991). Somatisation presents in three overlapping, though conceptually distinct, patterns of behaviour. These include high levels of medically unexplained symptoms referring to multiple physiological systems; somatic preoccupation or illness worry beyond what is expected for demonstrable physical disease as in hypochondriasis, and, the predominantly or exclusively somatic clinical presentation of psychiatric illnesses like depression, anxiety, etc. (Kirmayer & Robbins, 1991). All these forms are quite common in clinical practice, both independently as well as in co-existing situations.

The first form of somatisation is the medically unexplained symptoms. An emotionally distressed person may selectively present physical complaints to a doctor on the assumption that this is the best approach to
secure relief for the psychological distress. A careful interview is likely to reveal the underlying psychosocial distress. To operationalise the phenomenon, Bridges and Goldberg (1985) have given two criteria for somatisation; one must attribute the symptoms to physical illness, and; one must report, when properly interviewed, symptoms that justify psychiatric diagnosis. The physical symptoms may belong to different bodily systems, occurring in different combinations. Somatisation disorder is an extreme form of this kind of presentation (American Psychiatric Association, 1994). The disorder as defined in DSM-III, DSM-III-R, DSM-IV or ICD-10 is relatively rare. The multicentric-NIMH Epidemiological Catchment Area (ECA) study showed a prevalence of 0-0.7% across different sites (Escobar et al., 1987; Swartz et al., 1986). WHO's multicentric study done in 15 centres in 14 countries, found a prevalence of 2.8% of somatisation disorder as defined by ICD-10 and of 0.9% when DSM-III-R definition was used (Gureje et al., 1997). Considering the rarity of somatisation disorder and commonness of somatisation phenomenon, Escobar and Canino (1989) suggested abridged criteria of four unexplained symptoms for men and six for women to define a sub-syndromal somatisation disorder or somatic symptom index. This disorder had a community prevalence of 9-20% in ECA samples. Use of similar concept found a prevalence of 19.7% across different sites in the WHO's multinational study (Gureje et al., 1997). The concept of somatic symptom index or abridged somatisation gets further validation from the findings that these patients show high utilisation of mental health and medical services and disability (Escobar, 1987; Escobar & Canino, 1989). In WHO's multicentric study, symptoms showed a modest association with low education. The rates were much higher in South American sites. However, frequency of unexplained somatic symptoms did not clearly vary according to geography or level of economic development. Somatising patients were at elevated risk for self-reported disease burden, negative perception of their health, and comorbid depression and generalised anxiety disorder. Somatisation was commonly associated with disability. Cultures did not differ markedly in the pattern of these associated features (Gureje et al., 1997; Gureje & Simon, 1999). In a study by the author, patients presenting with abridged somatisation alongwith depression showed more psychosocial dysfunction than the somatising patients without a depressive diagnosis (Chadda et al., 1993). The dysfunction was seen in different areas of psychosocial functioning like personal, social, vocational and familial areas, but was minimal in cognitive sphere.

The second concept of somatisation, somatic preoccupation or hypochondriacal worry, developed out of the study of clinical illness behaviour (Pilowsky, 1990). Probably an amplifying somatic style leads to experience and expression of high levels of somatic distress in response to mild disturbances or even normal physiological processes (Barsky & Klerman, 1983; Barsky & Wyshak, 1990; Barsky, 1992). The prevalence of hypochondriacal concerns in clinical and non-clinical samples ranges from 3 to 13% (Barsky et al., 1986; Kellner, 1991). In hypochondriasis, the symptoms or level of distress experienced by patients is unexplained by physical disease and symptoms are not usually multiple in nature.

The third definition of somatisation, the somatic clinical presentation of psychiatric disorders other than somatoform disorders, has been studied primarily by consultation-liaison psychiatrists in primary care and specialty medical settings. Depression and anxiety disorders have been reported to have a combined prevalence of 10-20% in primary care population (Barrett et al., 1988). More than 50% of these patients make somatised or masked clinical presentations (Katon et al., 1984). A similar study by Bridges and Goldberg (1985) found that 56% of primary care patients with psychiatric disorders had somatised presentation, 17% had psychological symptoms and the remaining 27% had a concomitant
physical disease. In a study on somatisation in psychiatric patients in India, 81.9% of patients presented with somatic symptoms. This included 97.8% of patients with depression, 85.8% of patients with anxiety neurosis, all cases of hysterical neurosis, 6.7% of schizophrenia patients and 11.1% of patients with mania (Chadda & Bhatia, 1990).

There is a frequent co-occurrence of the three forms of somatisation. Major depression and anxiety disorders are common among patients with medically unexplained symptoms (Lipowski, 1990; Chadda & Bhatia, 1990). More than 60% of people with full or subsyndromal somatisation disorder have a past history of some other psychiatric disorder, mostly major depression or anxiety disorder (Escobar & Canino, 1989). Hypochondriacal patients tend to report high levels of current somatic symptoms. However, the prevalence of somatisation disorder in hypochondriacal patients has not been reported (Kellner, 1994). Similarly, the prevalence of hypochondriacal beliefs in somatisation disorder is not known (Kellner, 1991). Increased levels of hypochondriacal worry have been reported in patients with chronic idiopathic benign pain and other medically unexplained symptoms. Hypochondriacal fears are also found in 25 to 33% of patients with depression (Hamilton, 1989). Almost all patients with major depression or anxiety disorders have somatic complaints that include pain or other vegetative symptoms (Katon et al., 1984; Chadda & Bhatia, 1990; Srinivasan et al., 1986; Nelson & Charney, 1981). Patients with diagnosis of hypochondriasis have often elevated levels of depressive and anxiety symptoms, not fulfilling criteria for a coexisting major affective disorder (Kellner 1991).

**Nosological issues**

Somatisation may occur as a transient stress response or may be persistent. The former would hardly warrant a psychiatric diagnostic label. Persistent somatisation is a matter of great concern and cuts across different diagnostic entities, as also stated earlier. Somatisation may occur as a primary disturbance, as in somatoform disorders; or in association with other psychiatric disorders, such as major depression or anxiety disorders (Escobar, 1987).

Psychiatric diagnoses in somatising patients include depressive and anxiety disorders with secondary somatisation or somatoform disorders where somatisation is the primary phenomenon (Lipowski, 1988; Chadda et al., 1991). The category of somatoform disorders is a relatively recent diagnostic category introduced by DSM-III in 1980 and also included by ICD 10 in 1992. Nosological issues related to somatisation can be discussed in relation to occurrence of the phenomenon in somatoform disorders, depression, anxiety disorders and psychotic disorders.

**Somatisation and somatoform disorders**

Inclusion of the category of somatoform disorders by DSM-III in 1980 was a major step in recognising the importance of the phenomenon of somatisation. This helped in bringing together a group of disorders known since long by different names as distinct diagnostic entities into a more discrete category. However, except for somatisation disorder which had been extensively studied for diagnostic stability and validity (Perley & Guze, 1962; Guze, 1970; Cloninger et al., 1975; Gordon et al., 1986 a,b), the other somatoform diagnoses are just non-specific symptom clusters, that can not be properly operationalised and rarely occur in pure forms. Each of these disorders has arisen in a different school of psychiatric thought. The concept of conversion disorder is basically a psychodynamic one, whereas somatization disorder is a result of phenomenological and descriptive research. The pain disorder has achieved recognition primarily because of its clinical significance (Chadda, 1993, 1999).

Out of the various somatoform disorders, conversion disorder and hypochondriasis were traditionally grouped under neuroses, whereas somatization disorder was considered a type or variant of hysteria. Psychogenic pain disorder
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(pain disorder in DSM-IV) has also gained a honorable position, as earlier it was put in miscellaneous group in various classification systems. DSM-III-R and DSM-IV have also included the categories of undifferentiated somatoform disorder and body dysmorphic disorder in an attempt to be more specific. ICD-10 differs from DSM-IV in not including the categories of conversion disorder in somatoform disorders. Another important diagnosis, neurasthenia (chronic fatigue syndrome) does not find place in somatoform disorders, though its presentation is predominantly somatic (Chadda, 1999).

Similarly, in case of certain culture bound syndromes like Koro and Dhat syndrome, the presentation is predominantly somatic, and therefore there is a cause to include these under somatoform disorders (Chadda & Ahuja, 1990; Chadda & Shome, 1991; Chadda, 1995; Chaudhary, 1992). Some beginning has been made in this direction in ICD-10 by including such disorders under other unspecified neurotic disorders. The duration criteria of 6 months for pain disorder and undifferentiated somatoform disorder in DSM-IV result in too many not otherwise specified (NOS) diagnoses in clinical practice. In our study on diagnostic uncertainties in somatising patients, 39% of patients received NOS diagnoses on DSM-III-R. Such duration criteria are of more utility in the research settings rather than in clinical practice (Chadda et al., 1991; Chadda, 1992).

Somatisation and depressive disorders

Depression is one of the most common causes of somatisation (Katon et al., 1984). A majority of the patients presenting with depressive disorders in primary care settings do so for somatic rather than psychological distress and symptoms (Bridges & Goldberg, 1985). Depressed patients in psychiatric settings also show high prevalence of somatic symptoms (Chadda & Bhatia, 1990). Some depressed patients are convinced that their physical symptoms are due to somatic illness and may deny being emotionally distressed, while the majority seem to be aware of both bodily and affective distress but are unsure which of them is primary (Katon et al., 1984). Most of these patients, however, seek help from non-psychiatric physicians for somatic symptoms. If one accepts the notion of somatisation trait, then those possessing it will tend to report primarily physical symptoms when depressed. Others may choose to present such symptoms not because they do not experience depressed mood but because they do not wish to be given psychiatric diagnosis and hence be stigmatised or because they believe it inappropriate to discuss about psychosocial issues with general practitioners (Lipowski, 1988).

Somatising depressed patients may complain of multiple and vague somatic symptoms and often express related hypochondriacal concerns (Katon et al., 1984; Chadda et al., 1991). Pain is also a common symptom in depression. Prevalence of depressive disorders in patients with chronic pain ranges from 30%-50% (Fishbain et al., 1986). The arguments that depression is the cause of pain, and the pain is the cause of depression are equally plausible (Lipowski, 1988).

Somatisation and anxiety disorders

Anxiety disorders are frequently associated with depression, somatisation and hypochondriasis (Katon et al., 1984; Wilson et al., 1987). Patients with panic disorder and generalised anxiety disorder commonly present with various somatic complaints, such as chest pain, palpitations, dyspepsia, headache, dizziness, fainting and dyspnea. Preoccupation with the fear or belief of being physically ill is so common in some panic disorder patients that hypochondriasis has also been suggested as its essential feature by some investigators. Patients with panic disorder who are diagnosed early and treated properly tend to give up their tendency to somatise earlier than the depressed patients whose tendency to somatise and utilise health care is more persistent (Wilson et al., 1987).

Somatisation and psychosis

Somatic or hypochondriacal delusions of
having a physical disease or of a body change, malfunction or deformity may be seen in psychotic depression, schizophrenia and delusional disorders. These delusions may be regarded as the most pathologial form of somatisation. A psychotic patient may seek medical help for somatic symptoms, such as pain or weakness, lacking organic explanation but elaborated in delusional manner.

**Biological basis of somatisation**

The concept of somatisation arose from psychoanalytic school of thought. As earlier discussed, it cuts across different psychiatric diagnoses and seen as a part of clinical presentation of anxiety, depressive and somatoform disorders. Biological basis of somatisation is still not clear and different hypotheses have been given, based on some experimental findings in different psychiatric disorders in which the phenomenon has been reported. There is some evidence though not conclusive about biological abnormalities or genetic factors associated with somatisation and somatoform disorders. Besides genetic factors, other investigations have been done in neurophysiological parameters, neurotransmitters and neuropsychological functions like cognition and perception. Figure gives a diagrammatic representation of various etiological factors operating in the genesis of somatisation.

**FIGURE**

**ETIOLOGICAL FACTORS IN SOMATISATION**

- Somatoform-Primary Disorders
- Secondary-Other Psychiatric Disorders
- Persistent
- Transient
- Somatisation
- Genetic
- Environmental
- Neurophysiological/Neuropsychological abnormalities
- Enhanced Disturbed Amplification Cortical Neuro Response CNS filtering Augmentation Hyperarousal trans- of sensations mitters

Role of genetic factors is mainly established in somatisation disorder. Antisocial personality disorder and somatisation disorder may have a common genetic background. Somatisation disorder may be the female expression of a genetic tendency with antisocial personality disorder being its male counterpart (Guggenheim & Smith, 1995). Familial basis is established in Briquet's syndrome, of which the somatisation disorder can be easily called a descendent. Briquet's syndrome is seen in 10-20% of the female first degree relatives of probands with the syndrome which is a fivefold to tenfold increase over the lifetime risk of the disorder in women in the general (Cloninger, 1994; Cloninger et al., 1975). The male relatives of women with Briquet's syndrome show an increased risk of antisocial personality and alcoholism (Cloninger et al., 1975). However, men with many somatic complaints are probably heterogeneous and don't aggregate in families with male or female somatisers (Cloninger et al., 1984). Somatisation in women has a common etiology with antisocial personality, but not with somatisation in man (Cloninger, 1994). The familial aggregation could be due to genetic factors, environmental factors or the both, as shown by an adoption study of somatoform disorders from Sweden (Bohman et al., 1984). Using comprehensive lifetime medical records in Sweden, Cloninger et al. (1984) distinguished two discrete kinds of somatoform disorders in women: high frequency somatisation (complaints of headache, backache, gastrointestinal and gynaecological complaints with psychiatric disability, and the other group, diversiform somatisers, characterised by diverse but fewer complaints and less frequent disability). Women who were adopted away at an early age had a fivefold increase in high frequency somatisation disorder if their biological parents were antisocial or alcoholic (Cloninger et al., 1984; Cloninger, 1994).

Studies of biological and adoptive parents of hyperactive children have provided evidence of sharing of genetic factors between Briquet's syndrome and attention deficit disorder with hyperactivity in children. Briquet's syndrome and antisocial personality are increased in the biologic parents of hyperactive children in intact families but not in adoptive parents (Cloninger, 1994).
There is also some evidence from twin studies towards role of genetic factors in etiology of somatisation. A MMPI study of MZ and DZ adolescent twins favoured only a small contribution of genetics to somatic symptoms formation (Gottesman, 1962). Another twin study from general population using self rating scales suggested role of genetic factors in somatic anxiety (Kendler et al., 1987). One small twin study of somatoform disorders which included 35 twins of different categories of somatoform disorders, found 29% concordance in MZ twins and 10% in DZ pairs. The differences were not statistically significant. Anxiety disorders especially generalized anxiety disorder were also common in co-twins of probands with somatoform disorders. The results can't be said conclusive since sample size was quite small (Torgersen, 1986).

Neurophysiological abnormalities in evoked potentials have been found in patients with somatisation disorder and conversion disorder. Similarly, abnormalities in information processing have been hypothesised in various somatoform disorders.

Some abnormalities in information processing system have also been reported, like distractibility, difficulty in differentiating between target and non-target stimuli, and impaired verbal communication. This leads to inability to habituate to repetitive stimuli (Flor-Henry et al., 1981; James et al., 1990). The distractibility and impaired differentiating ability leads to vague non-specific labels for various experiences, and unclear and incomplete statements of ideas and feelings, and distortion of new information to fit earlier preconceptions. Selective attention also increases the perception of somatic sensations. Flor-Henry et al. (1981) found that Briquet syndrome patients, when compared with normal controls, have a bilateral and symmetrical pattern of frontal lobe impairment. They also have bilateral, but principally non-dominant (right sided) posterior temporal deficits.

Evoked potential studies have reported large N1 amplitudes in somatization disorder patients, suggesting a disturbed capacity to filter out trivial afferent stimuli (Gordon et al., 1986 a, b). Similarly, increased P1 N1 amplitude change as a function of stimulus intensity inpatients with somatization disorder suggest an enhanced CNS response to sensory input (James et al., 1990). Mismatch negativity (MMN), another EP parameter has been reported to be smaller, suggesting that somatizers probably respond more similarly than the normals to relevant and irrelevant stimuli (James et al., 1989). Loss of stimulus discrimination, self regulation and cognitive integration of experience appear to be in background of tendency to somatize (van der Feltz-Cornelis and van Dyck, 1997).

Patients with conversion disorders suffer from excessive cortical arousal, which causes reactive inhibition of signals at synapses in sensory-motor pathways by way of negative feedback relationship between cerebral cortex and the brainstem reticular formation. This explains the diminished awareness of bodily sensations in some conversion disorder patients (Behrman & Levy, 1970). This also explains the precipitation of symptoms by stress, reduction of anxiety following appearance of symptoms and effectiveness of amobarbital infusion and other sedatives in symptom remission (Raskin et al., 1966; Lader, 1982, Frumkin et al., 1981). The increased susceptibility of certain individuals, like those with histrionic, dependent or antisocial personality traits, and others has also been explained on neurophysiological basis. Probably, some of them have low sedation threshold, dominant cerebral hemisphere dysfunction or have suffered from frontal lobe trauma or impairment (Flor-Henry et al., 1981).

Most patients of conversion disorder have bilateral symptoms. But when the symptoms are unilateral, left side is usually affected. This phenomenon is more common in women and is probably a direct consequence of brain neural organization (Flor-Henry et al., 1981; Min & Lee, 1997; Merskey & Watson, 1979).

Hypochondriasis is the result of an underlying perceptual or cognitive abnormality.
The patients amplify and augment normal bodily sensations and perceive them as more noxious and intense than does the person who is not hypochondriacal. They have constitutionally lower threshold and tolerance for physical discomfort (Barsky, 1977; Robbins and Kirmayer, 1996; Mayou, 1976). They misinterpret normal bodily sensations, physiological functions, the trivial symptoms of every day life and the somatic symptoms of emotional arousal by misattributing them to a serious disease process. Recently, it has been reported that, though the hypochondriacal patients are more sensitive to normal physiological sensations and minor bodily parameters than the non-hypochondriacs, objective parameters like ability to discriminate between two factual bodily signals don’t confirm it (Barsky & Wyshak, 1990; Haenen et al., 1997; Barsky, 1992). They are also probably constitutionally predisposed to thinking and perceiving in concrete and physical terms rather than in emotional and subjective terms (Barsky & Wyshak, 1990).

Abnormalities in neurotransmitters have specifically been seen in pain disorders. Serotonin has been reported to have an inhibiting effect on pain perception and has been implicated in pain disorder. Endorphins and serotonin metabolites are decreased in cerebrospinal fluid of chronic pain patients (von Knorring et al., 1979).

Substance P, another neurotransmitter, is also involved in altering the pain threshold. Patients with somatoform pain syndromes have higher autonomic and muscle activity as compared to controls. There is dysfunction of corticofugal inhibitory system resulting in insufficient inhibition of the afferent stimulation, which results in amplification or heightened arousal to somatic sensory input.

Somatisation in depressive disorders may have different origins. Some of the physical complaints are vegetative symptoms concomitant with depressed mood, while others are somatic metaphors with which patients communicate their emotional distress. The depressed mood may also influence cognition in the direction of augmented perception of bodily sensations and their interpretation in terms of physical illness. Some investigators suggest that somatic complaints constitute a major feature of depression and should be included among its diagnostic criteria (Wilson et al., 1987).

Somatisation in anxiety disorders patients has been attributed to enhanced awareness of and selective attention to bodily sensations and danger related information, increased sympathetic nervous system arousal, and a negative bias in appraising one’s health (Noyes et al., 1986; Macleod et al., 1986).

In conclusion, somatisation appears a complex phenomenon, quite common in clinical practice, occurring in a wide range of disorders. A critical analysis of the phenomenon has led to identification of the new diagnosis group of somatoform disorders. Certain neurophysiological abnormalities have been identified in somatisation disorder, conversion disorder, hypochondriasis and somatoform pain disorder. Evidence of genetic basis in causation of some of these disorders has also emerged.

The phenomenon is an interesting example in psychiatry in recent years, where creation of new diagnosis groups on the basis of an empirical phenomenon may probably lead to understanding the biological basis of these groups of disorders, a field where earlier biological research had not led to any conclusive findings.

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