REVIEW ARTICLE

"Hysteria" in Clinical Neurology

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ABSTRACT: Hysteria is an ancient word for a common clinical condition. Although it no longer appears in official diagnostic classifications, "hysteria" is used here as a generic term to cover both "somatoform" and "dissociative" disorders as these are related psychopathological states. This paper reviews the clinical features of four hysterical syndromes known to occur in a neurologist's practice, viz conversion, somatization and pain disorders, and psychogenic amnesia. The presence in the clinical history of a multiplicity of symptoms, prodromal stress, a "model" for the symptom(s), and secondary reinforcement all suggest the diagnosis, and minimise the need for extensive investigations to rule out organic disease. Psychodynamic, behavioral, psychophysiologic and genetic factors have been proffered to explain etiology. Appropriate treatment involves psychotherapeutic, behavioral and pharmacological techniques. A basic requirement is to avoid errors of commission such as multiple specialist referrals and invasive diagnostic and treatment procedures. Hysteria is a remediable condition if identified early and managed appropriately.

RÉSUMÉ: L' "hystérie" en neurologie clinique. Hystérie est un mot ancien utilisé pour désigner une affection fréquente en clinique. Bien qu'on ne le retrouve plus dans les classifications diagnostiques officielles, le mot "hystérie" est utilisé ici comme terme générique pour désigner tant les désordres "somatoformes" que "dissociatifs", ces psychopathologies étant reliées. Cet article revoit les manifestations cliniques de quatre syndromes hystériques rencontrés en pratique neurologique, à savoir la conversion, la somatisation et les troubles de la douleur, et l'amnésie psychogène. La présence d'une multitude de symptômes à l'histoire clinique, de stress comme prodrome, d'un "modèle" des symptômes et de renforcement secondaire suggèrent le diagnostic et minimise la nécessité d'avoir recours à des investigations exhaustives pour éliminer une maladie organique. Des facteurs psychodynamiques comportementaux, psychophysiologiques et génétiques ont été invoqués pour en expliquer l'étiologie. Un traitement approprié doit inclure des techniques de psychothérapie, de modification du comportement et un traitement pharmacologique. On doit avant tout éviter les erreurs de commission telles la consultation de plusieurs spécialistes et les manoeuvres diagnostiques et thérapeutiques invasives. L'hystérie est une affection curable si elle est identifiée tôt et traitée de façon appropriée.

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"Hysteria" is an ancient and venerable word. It is derived from the Greek word for uterus and was coined by Hippocrates who thought that the condition occurred only in women and that it was caused by movement of the uterus in the body. Our concept of hysteria has gone through many changes since the time of Hippocrates. Hysterics were regarded as witches during the Middle Ages, and many were burnt or hanged on this account.[1] Focus changed from the uterus to the brain only in the 17th Century but it was not until Pierre Briquet published his classic monograph in 1859[2-4] that the uterine theory was finally laid to rest. In the late nineteenth century our understanding of hysteria was further advanced by the studies of Jean-Martin Charcot and his pupils, including Richer, Janet and Babinski. Freud also was strongly influenced by Charcot and carried out his earliest psychoanalytic work on hysteria.

In some respects hysteria is an unsatisfactory term, both because of the frequency with which hysteria is now known to occur in men, and also because of its pejorative connotation. The term has been dropped from the International Classification of Diseases (ICD-10) nomenclature and also from the recently published DSM IV system.[5] In its place are two groups of disorders, the Somatoform and the Dissociative Disorders, which are dealt with under separate chapter headings. I am using the word in this paper as a generic term to cover both somatoform and dissociative disorders, so as to emphasize that these are related psychopathological processes. Despite the term's absence now from official psychiatric classifications, it remains in widespread use. Although patients with hysteria may present to any physician, they are perhaps more prevalent in a neurologists' clientele.[6,7]

It should be noted here that a number of other terms such as "chronic neurosis", "chronic hysteria", "psychosomatic disorder", "functional disorder" or "functional overlay" are commonly used by practicing clinicians to describe this group of patients. These phrases are ill defined and sometimes used loosely hence it is difficult to relate them to the DSM IV system, where diagnostic categories are described precisely and specifically. It appears likely to

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this author that the terms chronic neurosis and chronic hysteria approximate the DSM IV diagnosis of Somatisation Disorder, and that the terms "psychosomatic" and "functional" as used colloquially by clinicians, refers more to "conversion" or to the amplification of symptoms of an underlying physical disease.

The term "pseudodementia" is also encountered in clinical neurology to describe patients who have dementia-like features without apparent organic cerebral disease. Pseudodementia is not a diagnostic entity in DSM IV, but the clinical conditions associated with the patient who "appears" demented will be dealt with in the section on psychogenic amnesia.

It should be noted also that diagnostic nomenclature in psychiatry is under constant review and change. DSM III R has now been superceded by DSM IV. Hence some of these terms, and their definition are likely to be modified. There are also terminological differences between the DSM and the ICD systems; however the latter is little used at present in North American psychiatry.

In this paper I will review the theoretical concepts which are a prerequisite for understanding how hysterical symptoms develop. I will then describe the diagnostic criteria for the Somatoform and Dissociative Disorders with special emphasis on pseudoneurologic symptoms. Finally, I will review the etiology and epidemiology and provide practical guidelines for the management of these patients. It should be noted that "hysterical personality disorder" is not being discussed as it rarely presents to a neurologist as such.

Concept of Hysteria

Physicians, particularly those with a strong biologic orientation, commonly have difficulty understanding the mode of production of conversion symptoms. This may lead them to deny that hysteria exists, or to assume that there is always an organic cause, or to equate hysteria with malingering. It is easier to understand the mode of production of hysterical symptoms if one understands the theoretical, psychological and social concepts which underlie the process of conversion. Hence these concepts will be defined and discussed in some detail (see Table 1).

It is helpful at the outset to distinguish the terms "disease" and "illness". Disease describes the condition associated with pathophysiological disturbance of structure or function; illness refers to the human experience of symptoms and suffering.[8] This experience may or may not be associated with pathological damage to the tissues, hence an individual can have an illness without a disease and a disease without an illness. An illness leads an individual to exhibit "illness behaviour" a term introduced by Mechanic[9] to describe the behaviour displayed by individuals in reaction to their perception of symptoms and health problems. For example, they may go to bed, miss work, visit the doctor, take medication, or allow themselves to be admitted to hospital. In other words they adopt the "sick role". It is reasonable to ask how and why individuals who do not have a disease in the physical sense can exhibit the behaviour and accoutrements of illness. Part of the answer is that they have a fear or belief that they are ill and react accordingly. The details of this reaction are coloured by the individual's previous personal and social experience of illness.[10] In addition, the illness may fulfill a need or resolve a psychological conflict vicariously. This conflict may be an acute or a chronic situational stress at home or at work which becomes unbearable. The illness then evolves as the means of relieving and resolving part of the anxiety associated with the stress. The social and stress factors which determine illness onset have been well described by Rahe et al.[11] and Rubin et al.[12] It is worth noting that it is often "illness" rather than "disease" which determines the timing and reason for a patient seeking medical attention.[13,14]

The symptom complex and its behavioral derivates may be reinforced by environmental events, for example, the avoidance through illness, of stress at home or at work.[15] These factors have been referred to as "secondary gain". The word "reinforcer" is preferable to "gain" as the latter is pejorative and implies conscious manipulation which is more characteristic of malingering than of hysteria. Physicians may also unwittingly reinforce the patient's complaints through inappropriate medical investigations and treatments. Clearly these reinforcers cannot be observed directly; their presence is inferred from the patient's history and behaviour in response to his/her illness. Nevertheless it is useful to note their presence because they have important management implications.

There are two broad categories of hysterical symptoms. These are *somatoform,* which affect the body, and *dissociative,* which affect the mind. *Conversion* represents the process by which anxiety or conflict related to an event is transformed and becomes manifest as a physical symptom. *Dissociation* is the mechanism by which a part of an individual's mental and behavioral processes become separated from the rest of his/her psychic activity.

One might question why patients express these needs, anxieties and conflicts in terms of a physical symptom rather than

Table 1: Definitions of terms associated with hysteria.

Disease:	The condition associated with pathological disturbance of structure or function.
Illness:	The experience associated with ill-health, symptoms and suffering.
Illness behaviour:	The ways in which given symptoms may be differently perceived, evaluated and acted (or not acted) upon by different kinds of persons.
Sick role:	The behaviour associated with illness.
Conversion:	The process by which anxiety or conflict related to an event is transformed and becomes manifest as a physical symptom.
Dissociation:	The mechanism by which a part of an individual's mental and behavioral function becomes separated from the rest of their psychic activity.
Secondary reinforcement:	The intensification of symptoms which may occur as a result of the effect of the illness on the environment.
"La belle Indifférence":	The inappropriate absence of distress in an individual with seemingly disabling physical symptoms.
Modelling:	The process by which conversion symptoms are based on an individual's previous experience of that symptom, either in themselves or in others.

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experiencing and describing their particular feelings and concerns. In fact, patients with hysterical symptoms are commonly dysphoric (i.e., feel subjective emotional distress). Indeed, two recent reviews concluded that the hysterias were a group of complex multifactorial conditions and that the only shared common feature was the frequent presence of anxiety and depression.[16,17] In addition, patients may have learned through experience in their families, or previous experience with physicians that physical symptoms are more acceptable than emotional symptoms. Hence they are more comfortable in describing their anxieties in physical rather than emotional terms indicating also that they lack insight into causation. Finally, these patients may have a fear of organic disease, and this sensitizes them to develop the symptoms of the disease they fear.

Another concept helpful in understanding how conversion symptoms may develop is that of "modelling" or imitation. This refers to the observation that the patient develops the symptom(s) after observing them in others with whom he/she identifies or after having experienced them previously him/herself.[15] For example, a 20-year-old woman developed episodic left sided weakness caused by conversion after her father had been disabled following a stroke. Her own symptom, and in particular her "choice" of affected area was determined in part by her identification with her sick father. This explains why some epileptic patients have pseudoseizures. They are based on and modelled after their own previous experience of seizures and occur often in a setting of stress or conflict. The process of modelling has been little studied in the clinical and experimental literature, yet in my experience it has been a helpful feature suggestive of the diagnosis of hysteria.

In the reproduction here of Brouillet's well known painting of Charcot's teaching session (see Figure), Charcot appears to have induced by suggestion a convulsion in the patient, and a nurse is ready to catch her if she falls. It is of interest to note that behind the students, but in full view of the patient, is a painting of a woman whose back is arched in a fashion similar to that of the patient. We do not of course know to what extent the artist was faithfully reproducing the scene, but he has unwittingly captured this common clinical finding.

A final feature commonly found in hysteria is an underlying histrionic (hysterical) personality. This is characterised by exaggerated emotional reactions (often with denial of these self same emotions) and attention seeking, self-centred behaviour. Such individuals may also be emotionally labile, inappropriately seductive and suggestible.[18] Suggestibility is accompanied by increased hypnotisability and this feature enabled Charcot to demonstrate hysterical seizures to his assembled students at the appropriate time and place (see Figure).

It perhaps needs to be emphasized that these intrapsychic and behavioral phenomena are not deliberate nor are they contrived by the patient. They generally occur in a setting of acute or chronic stress, cause varying degrees of subjective distress, and reflect the individual's unconscious way of reacting and dealing with the situation at hand. Hence in describing these features, one is in no way being critical or judgmental. They should be viewed as features that help one to understand and explain the observed phenomena. It should also be noted that none of these features is diagnostic of hysteria. They are suggestive only; but taken together with appropriate physical findings, they would strongly support the diagnosis of hysteria.

Figure 1: *"La leçon de Charcot" by: A. Brouillet (reproduced with permission of the Service Central des Archives et Musée historique, Hospices civils de Lyon, France).*

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Manifestations and Diagnosis

Table 2 illustrates the classification of the disorders according to DSM IV.[5] The Somatoform and the Dissociative Disorders are grouped together under the general rubric of "The Hysterias". An excellent description of the Somatoform Disorders is given by Ford.[19] Dissociative disorders have been much less studied in the literature.

I will discuss Conversion Disorder, Somatization Disorder, Somatoform Pain disorder and Psychogenic Amnesia in more detail, these being the conditions in this group which may involve a neurologist.

Conversion Disorder

The presenting symptom in this condition is an alteration or loss of physical function which suggests a physical disorder but in fact is the expression of an underlying psychological conflict or need. The presence of the psychological factor is usually not apparent at the onset, but becomes evident in the history when it is found that there is a time relationship (hence, by inference, a cause-effect relationship) between an environmental event (or "stress") and the onset of the symptom.

The nature and character of the presenting symptom embraces practically the whole field of clinical neurology. Almost any neurological symptom can have a "conversion" basis, hence it may come into the differential diagnosis of many neurological syndromes. Weakness, paralyses, sensory disturbances, pseudoseizures and involuntary movements (e.g., tremor) are likely the commonest of these.[20] On discreet questioning it may become evident that secondary reinforcement is present. The symptom, for example, may enable the individual to avoid an unpleasant activity at home or work, or obtaining support or attention from others. A further criterion is that the symptom must not be under voluntary control. The issue of volition is a difficult one, because it must be determined by inference rather than by observation. Features suggestive of voluntary control are inconsistency, variability and obvious and immediate benefit as well as a personality which would suggest dishonesty and opportunism. In general, in cases of uncertainty it is probably wise to give the patient the benefit of the doubt, and assume the symptoms are produced involuntarily unless there is good evidence to the contrary. Symptoms induced "voluntarily" tend to be self-limiting and of brief duration.

A further useful clinical feature of conversion disorder is the presence of a "model" for the symptom. This is sought by obtaining a full history of the present and past health of the patient's immediate family and of friends and other relatives. "La belle indifférence" has also been described as a characteristic feature of conversion. It is characterised by the inappropriate and paradoxical absence of distress despite the presence of an unpleasant symptom. There is also denial of emotional difficulty. It is seen in only a minority of subjects[21] and experimental studies have shown that patients with conversion have *increased* levels of psychophysiologic arousal despite their superficial unconcern.[22,23] It should be noted also that patients with organic disease may show an inappropriate absence of dysphoria, hence "la belle indifférence" should never, on its own, be considered diagnostic of hysteria. Pincus[24] has emphasized the importance of a previous history of "psychosomatic" illness in differentiating conversion from organic disorders.

Table 2: Classification of the hysterias (DSM IV).

Somatoform Disorders	Dissociative Disorders
Somatization Disorder (Briquet's Syndrome)	Dissociative Amnesia
Conversion Disorder	Dissociative Fugue
Pain Disorder	Dissociative Identity Disorder
Hypochondriasis	Depersonalisation Disorder
Body Dysmorphic Disorder	

The absence of physical disorder is clearly an important diagnostic feature. Individuals with conversion disorder often have physical signs, but they do not follow usual neurological patterns; for example the classical dermatomes in a patient with numbness; alteration in muscle tone and muscle stretch reflexes in a patient with paralysis; the characteristics of the gait in a patient with a suspected conversion gait disturbance. I was able to suspect strongly that the blindness of a patient of mine had a psychogenic origin. During the interview, although his eyes were generally roving as if he were unable to see, every so often he would exhibit fleeting eye contact with me, as though at some level, he knew where I was in his visual field. The neurological signs of hysteria have been well described by Weintraub[25] and the neuro-ophthalmic by Keane.[26]

"Ruling out organic disease" should not be the only criterion for diagnosing conversion disorder. If this were the case, patients might be subjected to extensive physical investigations which may be not only expensive and inappropriate, but would help to perpetuate the symptom. It is necessary therefore to suspect the psychogenic nature of the condition early during the clinical interview, and to use the physical examination and special investigations to confirm the diagnosis.[27] It may even be appropriate to arrange a psychiatric assessment whilst the investigations are being carried out. This avoids the potentially threatening situation of being told, in essence "there is nothing wrong with you", with the implication, stated or otherwise, that "it's in your head". An early psychiatric evaluation gives the patient time to assimilate the possibility that the condition may be psychogenic while organic investigations are proceeding.

It is well known that some neurological diseases present with quasi conversion symptomatology, and the neurological origins become manifest only subsequently.[28,29] The diagnostician, therefore, has to strike a fine balance between not overinvestigating a patient with probable conversion disorder, while ensuring that serious neurological diseases are identified. Neurologists and psychiatrists need to collaborate to ensure appropriate management of these complex cases. There can be no inconsistency between a comprehensive neurological and a comprehensive psychiatric examination. The course and progress of the patient's condition will usually provide the final answer.

A final noteworthy feature is that the diagnoses of conversion disorder and neurological disorder are not mutually exclusive; they may occur in the same patient either concurrently or consecutively and are particularly common in chronic relapsing diseases such as multiple sclerosis[30] and epilepsy.[31] This must be borne in mind in management because physical and behavioral components may require different treatment strategies, and both need to be treated appropriately (see below in treatment section).

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Somatization Disorder (Briquet's Syndrome)

The key feature in this condition is a multiplicity of symptoms affecting several different organ systems. The criteria were developed and defined originally by Woodruff et al.[32] and have been refined by DSM III and DSM IV.[5] Patients with this condition tend to be chronically ill and manifest their symptoms at an early age.[33] They seek repeated medical attention, are subjected to many investigations and procedures, but often remain steadfast in their careers as patients.[34,35]

Other than the multiplicity of symptoms the earlier onset and the tendency for the course to be chronic rather than acute, Briquet's Syndrome shares many of the features of a Conversion Disorder. Secondary reinforcement may occur but the relation to precipitating stress may be less clear because of the chronic nature of the condition. Pseudoneurologic symptoms are common including amnesia, dysphagia, various dysesthesias and motor symptoms. The diagnosis is made according to the presence of multiple atypical symptoms involving several organ systems, and the absence of specific physical findings which may explain the symptoms.

Hypochondriasis (see Table 2) is sometimes confused with Somatisation Disorder because of its chronicity and the frequent occurrence of multiple somatic symptoms. The key feature here however is fear or preoccupation with having a serious physical disease and most commonly the focus is on cardiac, respiratory or gastrointestinal function.

Pain Disorder (previously somatoform or "psychogenic" pain disorder)

Persistent pain in the absence of an adequate physical cause is the cardinal feature of this condition. As with Conversion Disorder and Briquet's Syndrome, there is often a precipitating stress or conflict and the condition is commonly associated with secondary reinforcement.

Headache is probably the commonest symptom presenting to the neurologist and low back pain, or pain in the neck or extremities are also common. In a study of psychiatric pathology in a series of over 2000 neurology outpatients 13.2% were diagnosed as having a primary psychiatric disorder and headache was the most prevalent symptom in this group, occurring in 43% of patients.[36] A separate questionnaire study of 103 consecutive neurologic outpatients presenting with headache identified psychiatric pathology in 20% and "recognisable depression" in 7%.[37] Headache does not receive a separate diagnostic category in DSM IV but regional pain syndromes are subclassified according to their localisation. The role of "muscle tension" in the production of tension headache remains questionable,[38,39] hence it seems probable that a substantial proportion of headache patients do in fact, have Somatoform Pain Disorder rather than a true psychophysiological disturbance.[40]

The picture is often confused by the fact that the condition may have begun with a trauma or a lesion associated with pain, or there may be an objective physical finding which explains the pain at least in part.[41] This may lead physicians to carry out diagnostic and therapeutic procedures which do little to relieve symptoms. Although "ruling out organic disease" is an essential diagnostic feature, the condition should be suspected during the clinical interview and the history, and when an atypical presentation together with features suggestive of a somatoform disorder are present.

Atypical Facial Pain is another form of Pain Disorder which may present to the neurologist. It occurs most commonly in middle aged women. Psychological factors such as stress and depression are frequently found.[41-43] A recent study of long-term outcome found that conservative treatment was effective for 70% of patients.[44]

Dissociative Amnesia

Dissociative Amnesia is one of the Dissociative Disorders (see Table 2) and is the one most likely to frequent the practice of a clinical neurologist who may describe these patients as having "pseudodementia". The memory disturbance in this condition is characterized by an inability to recall important personal information. The onset is often sudden, and is not anterograde, as is memory loss due to organic cerebral disease. The loss is more in the nature of a particular event or block of time, and almost always involves a loss of personal identity, which is a late feature in organic amnesias. A further distinguishing feature is that acquisition of new information is not impaired in psychogenic as it is in organic amnesias. In an excellent review of this field, Kopelman emphasized the difficulties in differentiating psychogenic from organic amnesias.[45] The onset occurs usually after acute stress or conflict. It may also occur following a head injury and this in turn, complicates diffential diagnosis. For a condition described in a standard textbook as the most common form of dissociative hysteria[46] dissociative amnesia has been remarkably little studied in the literature. In fact, it may be rarer than supposed. A recent survey of 1517 psychiatric out patients found no cases of psychogenic amnesia.[47] DSM IV describes 4 subtypes of the condition, localised, selective, generalised and continuous, dependant on the nature of the memory disturbance. As with the Somatoform Disorders, reinforcement and modelling may be evident on history and examination. Neurological investigations for brain disease are negative or inconclusive although neuropsychologic tests may reveal striking abnormalities in certain subtests. The presence of a neurologic abnormality does not necessarily rule out psychogenic amnesia if appropriate psychopathology is present; organic and psychogenic amnesia may coexist.[48] During the episode, the individual may appear confused, perplexed or disorientated, although these features are absent if the amnesia is present only in the past medical history.

The term "pseudodementia" has been used in both the psychiatric and neurological literature but it is not a nosological entity and is best characterised as a dementia-like syndrome which is secondary to other psychiatric conditions. Kiloh[49] found that depression was most commonly misdiagnosed as dementia, but other neurotic conditions have presented with this picture.[50-52] In another 9-16 year follow up of all 51 patients admitted with a diagnosis of presenile dementia, the authors found that the diagnosis was confirmed in only 35 (69%).[53] The majority of those misdiagnosed had an affective disorder. The authors emphasized the need for a more thorough investigation at the time of the original assessment.

Etiology

The modern period in the investigation of Hysteria began with the phenomenological studies of Briquet.[2] Briquet believed that hysteria was caused by physical changes in the brain;

indeed, he described certain poorly defined cerebral abnormalities in a few of his hysterical subjects who had died. Charcot's work was mainly descriptive, but he did open up the field to psychosocial influences by noting the role of suggestion in the induction of seizures. Breuer and Freud were the first to provide a comprehensive psychological explanation for the production of hysterical symptoms.[54] They stressed the role of symbolism in the choice of conversion symptom, and of personality factors related to sexual fantasies and repressed sexual impulses.

Modern theories concerning the cause of hysteria can be grouped into four categories: psychodynamic, behavioral, psychophysiological and genetic.

Psychodynamic

In addition to the theories of Freud mentioned above, more recent psychoanalysts have emphasized disturbances in relationship with the father,[55] the mother[56] and to problems in the control of frustration and aggressiveness.[57] These approaches are mainly theoretical, and based on individual case studies, hence their applicability to the broad range of individuals with hysterical syndromes is undetermined.

Behavioral

Learning theory presupposes that behaviour is learned through experience; behaviour which is rewarded is thereby promoted and reinforced, and that which is not rewarded is inhibited. In this sense, the hysterias may be regarded as "maladaptive ways of obtaining social or instrumental needs that substitute for deficits in the patient's adaptive behavioral repertoire".[58] Although little experimental work has been carried out on this topic in regard to the cause of the hysterias, the success of behavioral therapy in the treatment of these disorders suggests that maladaptive learning may have an important role to play in the etiology of these conditions.[58-60] It seems likely and reasonable for example that illness behaviour may be learned by a child observing a sick parent or sibling.

Psychophysiologic

A number of workers have investigated a possible connection between hysteria and cerebral pathology. Both Slater[61] and Merskey and Buhrich[62] found that a high proportion of "hysterics" had associated cerebral pathology in particular epilepsy and multiple sclerosis. In a comparable study from a psychiatric, rather than a neurological hospital setting however, Roy[63] found this association in only 3% of patients. It has also been proposed that hysteria is a dysfunction of attention and memory associated with inhibition of afferent stimulation[64] and that there is bifrontal cerebral impairment particularly in the non-dominant hemisphere.[65] In a study comparing magnetic evoked potentials (MEP) in patients with psychogenic and organic limb weakness, Meyer et al. found that the former were associated with normal MEP values.[66]

Briquet[2,4] and Arkonac and Guze[10] found a strong tendency for an increased incidence of hysteria to occur in the first degree relatives of hysterics. Guze elsewhere has found that male relatives of patients with Briquet's Syndrome have an increased prevalence of antisocial personality and alcoholism, and female relatives of a male prison population revealed a high prevalence of Briquet's Syndrome.[67] These studies suggest that social influences within the family have a strong bearing on the manifestations of these disorders, but they do not rule out a role for genetic influences.

Genetic

In a study comparing concordance in monozygotic and dizygotic twins, Torgersen[68] found 29% concordance in the former and 10% in the latter. However, the author conceded that similarity of childhood experience may have influenced these rates. In an extensive familial study Ljungberg[69] concluded that hereditary factors of a polygenic kind play a significant role in the development of hysteria, but this conclusion was not confirmed in subsequent analysis of his data.[70] Similar negative conclusions were found in a study comparing concordance of hysteria in monozygotic and dizygotic twins.[71] Shields[70] after reviewing this evidence concluded that there is some evidence supporting the role of genetic factors in hysterical personality, but little for other hysterical syndromes.

Prevalence

Studies on the prevalence of hysteria have been bedeviled by terminological, diagnostic and philosophical confusion. During the 1960s, physicians were entertained by the fascinating debate in the British Medical Journal between Eliot Slater, a psychiatrist who denied that hysteria existed,[61] and Francis Walsh, a neurologist who considered it to be a common clinical problem.[6]

Many studies have shown associations between hysteria and social and demographic variables. It has long been known to predominate in women; indeed, before Briquet, it was thought to be confined to women, hence its association with the uterus. Twenty in his series of over 400 patients were men (5%) and more recently Guze et al.[72] found that only 3% of patients with Briquet's Syndrome were men. They found also that young women reported a disproportionately greater number of symptoms than men or older women. In a survey of 1752 consecutive psychiatric consultations in a general hospital, I found that hysteria (all subgroups) was diagnosed in 156 of 1127 women (13.8%) and 58 of 625 men (9.3%) and the female-male ratio was 2.7:1.[27] This study is of particular interest in that it demonstrated that as a proportion of diagnoses in each gender, the prevalence of hysteria was comparable, negating the traditional view that hysteria was proportionately more prevalent in women. It appears that Briquet's Syndrome and Conversion Disorder predominate in women, and that Hypochondriasis and psychogenic pain may predominate in men.

There is a heavy preponderance of Briquet's Syndrome and Conversion Disorder in the younger age groups.[72] However hypochondriasis is common in older age groups, and there is clinical evidence that the prevalence of conversion symptoms (although not necessarily conversion disorder) increases again in older subjects. There is also strong evidence for an association between hysteria and lower socioeconomic status[73] and hysteria and antisocial personality.[74]

Epidemiological surveys have shown that these conditions are prevalent in the community. Woodroffe et al. estimated that Briquet's Syndrome had a prevalence of 2% in the general female population[32] and Engel[15] found symptoms of conversion in 20-25% of patients admitted to a general medical service. The NIMH Epidemiologic Catchment Area Study found a cross-sectional prevalence of 4% for somatisation syndrome.[33] A strong association with other psychiatric disorders was also noted.[75,76]

There are remarkably few data on the prevalence of psychiatric pathology in unselected neurologic populations. In a diagnostic survey of 7836 consecutive neurological outpatient referrals, Perkin[7] noted that 26% were "undiagnosed" and a further 14% had psychiatric diagnoses, including an unspecified number with conversion hysteria. However these subjects were not assessed by a psychiatrist. Prevalence studies would therefore indicate that conversion symptoms are sufficiently prevalent, and their manifestations sufficiently protean to be considered in the differential diagnosis of a wide variety of clinical diagnostic problems, particularly in outpatient populations.

Management

Although there exists a literature on the management of hysteria, there is a dearth of longer-term follow-up studies of specific treatment modalities. This is unfortunate because these often are serious and disabling conditions which are costly to society and to health funding agencies. Despite this paucity of experimental research, there does appear to be an overall consistency in the treatment approach recommended by both psychiatrists[17,60,77,78] and neurologists[20,79] working in this field. This section on management will reflect these guidelines from the literature, and will be based also on my personal experience in 34 years of clinical practice. It should be noted also that the management of the patient with acute hysterical syndromes (conversion disorder and psychogenic amnesia) is substantially different from that for the chronic (Briquet's Syndrome and psychogenic pain disorder). This section therefore will discuss the general principles for both types then summarize the particular treatment modalities appropriate for acute and then for chronic hysteria.

General Principles

Once the diagnosis has been made, the next essential step is to give the patient feedback within a framework that he/she can accept and understand. It is insufficient as well as ineffective to tell the patient, "There is nothing (physically) wrong with you". Equally, it is inappropriate to attribute the cause to the patient's "imagination" or "It's in your head". These individuals are frequently defensive about psychogenic causation, and it is necessary to respect this defensiveness by explaining that although no serious physical diseases have been found, they may have local physical irritation, muscle tension or inner bruising, which is the immediate cause of their symptoms, but that the latter are amplified and perpetuated by nervous tension, emotion or stress. The therapeutic implication of this conclusion is that both the physical and the psychological aspects need to be treated in order to secure a response. It is desirable that diagnostic feedback be given with confidence and authority. In order to accomplish this, the clinician must be assured that the patient has been adequately examined and investigated, and the patient also must be satisfied that the examination has been thorough.

Once serious physical disease has been ruled out, and this has been reported firmly to the patient, it is preferable to avoid further investigations, procedures and consultation referrals. If this is not done, it implies that the physician remains uncertain of the diagnosis, which increases the patient's anxiety and preoccupation with their health, hence the tendency to somatize. Although the physician needs to be alert to the possibility that organic disease may develop, new symptoms should not be investigated unless there are clear objective signs suggesting that an organic disease has developed.

Acute Syndromes

Both Conversion Disorder and Dissociative Amnesia commonly have an acute onset. If diagnosed early and treated appropriately they tend to have a short, self limiting course. Conversely, if the diagnosis is delayed, and management is inappropriate, this can lead to life-long invalidism.

Many conversion symptoms subside spontaneously over a few days once appropriate diagnostic feedback has been given to the patient. The patient's progress, therefore, is closely monitored over this period in the context of a supportive, conservative medical framework.

If the symptoms do not subside rapidly, a therapeutic interview with the use of intravenous sodium amytal may be accompanied by rapid symptomatic recovery.[80] Briefly, this involves the administration of 100 to 500mg of sodium amytal by slow intravenous injection. The drug is dissolved in 20mls of sterile water and given into a peripheral vein using a 20ml syringe. Before the procedure, the patient's support and understanding are secured by explaining that he/she is being given a medication by injection which will assist them in relaxing and talking more easily and which may also help to relieve their symptom. They should also be told that they will remain "in control" and will not say anything that they do not wish to disclose. Any questions should be answered in a factual manner. The patient is told that he/she may feel drowsy and/or relaxed during the injection but must not go to sleep. The injection is then started while attention is secured continuously by detailing the medical and social history. After 50-150mg has been given (the amount varies between different individuals), the patient will become noticeably more relaxed and begin to verbalize feelings and fears more easily. At this point, stresses and traumatic events can be explored in more detail, and suggestions made that the symptom will disappear and recovery will occur. On occasion, patients become tearful or angry during the session and a form of abreaction may develop which may have additional therapeutic benefit. It is very important to ensure that sleep during the interview is avoided, as any therapeutic effect is thereby negated. If a therapeutic effect is obtained, this should be reinforced during and after the interview. The whole session may last between 20 and 50 minutes and patients commonly sleep for several hours after the procedure due to the sedating effects of the amytal. Using the above guidelines, I have not encountered untoward physical nor psychological effects after many dozens of amytal interviews. The worst result I have found is that it may have no effect – i.e., it provides no new information and results in no symptomatic improvement. Symptomatic relief may be dramatic in patients with conversion paralyses, blindness and amnesia, although sometimes the symptom recurs if the underlying psychopathology is not dealt with. Sodium amytal is more likely to be beneficial when given early after the onset; the longer it is delayed, the less effective it is likely to be. However I have had beneficial effects up to 18 months after symptom onset; hence it may be worth using in selected more chronic cases.

In order to minimize the likelihood of a recurrence, it may be necessary to treat the underlying psychopathology. This involves understanding the relative importance of predisposing personality

factors, precipitating stressors, and perpetuating secondary reinforcers. Generally, a combination of psychological, and pharmacological therapy is appropriate (see below).

Chronic Syndromes

Most commonly these are patients with Briquet's Syndrome and Somatoform Pain Disorder, although on occasion patients with poorly managed conversion disorder or psychogenic amnesia may become chronic.

Treatment approaches are psychological and pharmacological.

Psychological

Psychological therapies are supportive psychotherapy, behaviour therapy (including relaxation therapy and environmental modification) and an occupational or physical activity program. Psychotherapy is best provided by a physician who is sympathetic and understanding towards the patient's particular difficulties and who is able to respond appropriately to both the physical and emotional problems he/she presents. It is necessary that therapy sessions be regular, structured and consistent. They need to be sufficiently frequent to provide support and reassurance, but not so frequent that excessive dependency is fostered. In general, patients should be discouraged from emergency or "p.r.n." visits to the doctor or the Emergency Department; and also from extended "therapeutic" telephone calls. These serve only to promote and reinforce illness behaviour and preoccupation with physical symptomatology. As rapport is established, the interval between visits can be gradually extended.

The tendency to somatize is tenacious hence the treatment commitment has to be long-term. In chronic somatizers, it may be appropriate to limit therapeutic objectives to stabilising the behaviour and preventing it from escalating. Murphy[69] emphasized that "the essence of the sophisticated management of hysteria is to avoid errors of commission".

During therapy sessions, the patient's attention should as far as possible be focused away from his/her health and symptoms, and more in the direction of daily life, activities and relationships. This may involve the physician taking care not to enquire about the presence of specific symptoms; if however, these are volunteered spontaneously, the response should be limited to general sympathy and support, without pursuing a symptomatic line of enquiry. It may be of value to give the patient specific behavioral objectives, for example walking a certain distance or joining in particular social or recreational functions a certain number of times. In order to emphasize its importance, these objectives can even be given in the form of a written prescription, with the added request that the patient write down in a daily diary when and for how long this activity is performed, and to bring the diary to the physician for inspection and discussion at the time of the next visit. Goldberg et al. have described an excellent psychoeducational approach to the treatment of somatisation.[81]

The above guidelines follow the principles of behavioral methods of treatment where the therapist structures the patient's social and physical environment to promote "good" behaviour (in this case healthy adjustment without physical complaints) and discourage "bad" behaviour (i.e., illness behaviour and preoccupation with physical symptoms). The use of regular structured sessions, discouraging emergency visits, setting behavioral goals, avoiding pursuance of physical investigations and treatments may all be seen as following behavioral principles of management within a supportive, non-authoritarian psychotherapeutic framework.[60,77,82]

It is of value to work also with the patient's spouse or family. They are unwittingly involved in the patient's illness and behaviour, and will harbour feelings and reactions which may have positive or negative effects on outcome. Providing diagnostic feedback to both the patient and spouse (family) together ensures that the latter do not depend on the patient's possibly distorted report for this information. Conjoint feedback also gives the family the opportunity to report their concerns and observations and ask questions of the physician, all of which may be essential to diagnosis or treatment. Family support may be vital to an effective treatment plan.

These arrangements form part of the process of "environmental modification" of a behavioral approach to therapy, the process of structuring or altering the patient's social, physical and occupational surroundings to promote a more therapeutic milieu. In patients whose symptoms are work related, whether or not there are Workmen's Compensation claims, it is appropriate to assist in the speedy resolution of these conflicts and claims. The emotion invested in the latter may be a potent perpetuating factor in the illness. Where possible and appropriate the patient should be steered back to the work situation. It has been claimed that Somatoform Disorders which are maintained and perpetuated by litigation and compensation issues resolve after settlement of the litigation,[83] however this has not been confirmed by a more recent study.[84] Another survey showed that factors associated with good outcome were: longer time after resolution of the litigation, shorter time between injury and resolution of litigation and less severe psychopathology.[85] In my experience there is little benefit in instituting vigorous treatment measures whilst there are outstanding legal issues which are helping perpetuate symptoms unless there are well-defined and treatable medical or psychiatric conditions. When clinical problems are more severe, it is appropriate to use symptomatic and supportive measures while expediting the early resolution of litigation. In those cases where symptoms persist after settlement, I follow the psychotherapeutic and behavioral techniques described above.

Relaxation therapy may also be of value in this group of patients. The technique of instruction is well described elsewhere.[86] The patient must be instructed to carry out the exercises regularly no less than 3 times weekly, and to persist in this endeavour. The maximum benefit from relaxation therapy occurs when it is practiced frequently over an extended length of time.

Pharmacological Therapies

There are no specific pharmacological therapies for the hysterias. However, individuals with these disorders often have symptoms of anxiety or depression which may lead to a need for medication.

In general anxiolytics (e.g., benzodiazepines) should not be used for anxiety associated with hysteria. Patients often have dependent personalities, and easily become addicted. This is particularly liable to occur if the medication is given "p.r.n." Hence if these drugs are used at all, they should be given for short periods (i.e., two or three weeks) and in a fixed-time format, rather than p.r.n.

Antidepressant drugs have a more substantial role to play in the management of these conditions. Depressive disorders are

commonly associated with the chronic somatoform conditions (Briquet's Syndrome & Somatoform Pain Disorder). Principles concerning the use of antidepressants are similar to major depression.[87,88] The somatization tendency can increase the sensitivity and probability of side effects in this group of patients. This tendency can be minimised by starting with small doses and titrating gradually. Heterocyclic, monoamine oxidase inhibitors and the recently introduced selective serotonin reuptake inhibitors may be of benefit also in the treatment of chronic pain syndromes.[43,89] In these conditions their effects may be analgesic as distinct from antidepressant. They are commonly used in smaller doses than those used in major depression.

Analgesics are commonly used in somatoform pain disorder. They are also often abused, particularly by individuals with severe symptoms and dependent personalities. Their abuse can perpetuate the symptom by creating a vicious cycle effect between pain, analgesic use, symptom relief followed by recurrence of pain when the analgesic effect wears off. For this reason, they should be used sparingly. If indicated, they are best used for short periods and in a fixed time rather than a p.r.n. format.

If identified early, hysteria is a treatable condition. If it is undiagnosed or misdiagnosed, the symptoms may become reinforced by secondary influences and this may result in social, psychologic and physical sequelae or even in life-long disability. Briquet,[2] an astute observer concluded, "Hysteria is far from being a condition of incoherent phenomena about which one can make little sense. On the contrary, I found that it was an affection whose nature was easy to understand, whose symptoms all had their analogues in physiological states, and were bizarre only in appearance; that they obeyed laws which could be determined; that the diagnosis could be made with as much precision as with other diseases; and that the diverse phenomena responded to a treatment one could formulate in advance." (my translation). These words are as true today as when they were written one hundred and thirty years ago.

References

1. Veith I. Hysteria. A History of a Disease. University of Chicago Press, 1965.
2. Briquet P. Traité de l'hystérie bailliere. Paris, 1859.
3. Mai FM, Merskey H. Briquet's concept of hysteria: a historical perspective. Can J Psychiatr 1981; 26: 57-63.
4. Mai FM, Merskey H. Briquet's treatise on hysteria. Arch Gen Psychiatr 1980; 37: 1401-1405.
5. Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) American Psychiatric Association, Washington, 1994.
6. Walsh F. Diagnosis of hysteria. Br Med J 1965; 1: 1395-1399.
7. Perkin G. An analysis of 7836 successive new outpatient referrals. J Neurol Neurosurg Psychiatry 1989; 52: 447-448.
8. Kleinman A. The Illness Narratives. Suffering, Healing and the Human Condition. NY: Basic Books Inc., 1988, Chapter I.
9. Mechanic D. The concept of illness behaviour. J Chron Dis 1972; 15: 189-194.
10. Arkonac O, Guze, S. A family study of hysteria. N Engl J Med 1963; 268: 239-242.
11. Rahe RH, Gunderson E, Arthur, R. Demographic and psychosocial factors in acute illness reporting. J Chron Dis 1970; 23: 245-255.
12. Rubin R, Gunderson E, Arthur R. Life stress and illness patterns in the US navy. J Psychosom Res 1971; 15: 277-288.
13. McWhinney, J. Beyond diagnosis. N Engl J Med 1972; 287: 384-387.
14. Eisenberg L. What makes persons "patients" and patients "well"? Am J Med 1980; 69: 277-286.
15. Engel, G. Conversion symptoms in "Symptoms and Signs in Clinical Medicine." *In*: E MacBryde C, & Blacklow R. eds. 5th ed. Philadelphia: JB Lippincott, 1970: 650-668.
16. Kellner R. Somatization. Theories and research. J Nerv Ment Dis 1990; 178: 150-160.
17. Lipowski Z. Somatisation: the concept and its clinical application. Am J Psychiatr 1988; 145: 1358-1368.
18. Chodoff P. "The hysterical personality disorder." *In*: Roy A, ed. Hysteria. New York: John Wiley, 1982: 277-285.
19. Ford C. The Somatizing disorders: Illness as a Way of Life. Elsevier Biomedical. New York, 1982.
20. Pryse-Phillips W, Murray TJ. Examination for functional disorders. *In*: Essential Neurology (4th Edition). Appleton and Lange Norwalk Conn. 1992: 71-78.
21. Weinstein E, Eck R, Tyerly O. Conversion hysteria in Appalachia. Psychiatry 1969; 32: 334-341.
22. Lader M, Sartotius N. Anxiety in patients with hysterical conversion symptoms. J Neurol Neurosurg Psychiatry 1968; 31: 490-495.
23. Rice DG, Greenfield NS. Psychophysiological correlates of la belle indifférence. Arch Gen Psychiatr 1969; 20: 239-245.
24. Pincus J. Hysteria presenting to the neurologist. *In:* Roy A, ed. Hysteria. New-York: John Wiley & Sons Ltd., 1982: 131-143.
25. Weintraub Michael I. Hysterical conversion reactions: a clinical guide to diagnosis and treatment. SP medical and scientific books. Jamaica: New-York, 1984.
26. Keane JR. Neuro ophthalmic signs and symptoms of hysteria. Neurology 1982; 32: 757-762.
27. Mai FM. Briquet's syndrome (hysteria) and the physician. Can Med Assoc J 1982; 127: 99-100.
28. Fichter C, Waddington W. Suspicion of somatoform disorder in undiagnosed tabes dorsalis. Br J Psychiatr 1991; 159: 573-575.
29. Jones J, Barklage N. Conversion disorder: camouflage for brain lesions in two cases. Arch Intern Med 1990; 150: 1343-1345.
30. Caplan L, Nadelson T. Multiple sclerosis and hysteria. JAMA 1980; 243: 2418-2421.
31. Roy A. Hysterical seizures. Arch Neurol 1979; 36: 447.
32. Woodruff R, Clayton P, Guze, S. Studies of diagnosis outcome and prevalence. JAMA 1971; 215: 425-428.
33. Swartz M, Landerman R, George LK, et al. Somatisation disorder. Psychiatric disorders in America. New-York: Free Press, 1991; 220-257.
34. Perley M, Guze S. Hysteria - the stability and usefulness of clinical criteria. N Engl J Med 1962; 266: 421-426.
35. DeVaul R, Faillace L. Surgery proneness: a review and clinical assessment. Psychosom 1980; 32: 295-299.
36. Kirk CA, Saunders M. Primary psychiatric illness in a neurological outpatient department in north-east England: an assessment of symptomatology. Acta Psychiat Scand 1977; 56: 294-302.
37. Merskey H, Brown J, Brown A, et al. Psychological normality and abnormality in persistent headache patients. Pain 1985; 23: 35-47.
38. Martin P, Marie G, Nathan P. Psychophysiological mechanisms in chronic headaches: investigation using pain induction and pain reduction procedures. J Psychosom Res 1992; 36: 137-148.
39. Arena J, Blanchard EB, Andrasik F, et al. Psychophysiological comparisons of three kinds of headache subjects during and between headache states. J Psychosom Res 1985; 29: 427-441.
40. Merskey H. Hysterical mechanisms and pain. *In:* Roy A, ed. Hysteria. New York: John Wiley & Sons Ltd., 1982.
41. Lindsay P, Wyckoff M. The depression-pain syndrome and its response to antidepressants. Psychosom 1981; 22: 571-577.
42. Feinmann C, Harris M, Cawley R. Psychogenic facial pain: presentation and treatment. Br Med J 1984; 228: 436-438.
43. Lascelles R. Atypical pain and depression. Br J Psychol 1966; 112: 651-659.
44. Feinmann C. The long-term outcome of facial pain treatment. J Psychosom Res 1993; 37: 381-387.
45. Kopelman M. Amnesia: organic and psychogenic. Br J Psychiatr 1987; 150: 428-442.
46. Nemiah J. *In:* H. Kaplan & B. Sadock, eds. Comprehensive Text book of psychiatry. 4th ed. Williams and Wilkins, 1985; 948-949.

47. Das P, Saxena S. Classification of dissociative states in DSM-III-R and ICD-10. Br J Psychiatr 1991; 159: 425-427.
48. Saling M. Psychogenic amnesia? [letter] Br J Psychiatr 1991; 159: 585.
49. Kilsh LG. Pseudodementia. Acta Psychiat Scand 1961; 37: 336-351.
50. Nott PN, Fleminger JJ. Presenile dementia: the difficulties of early diagnosis. Acta Psychiat Scand 1975; 51: 210-217.
51. McEvoy JP, Wells CE. Case studies in neuropsychiatry II: conversion pseudodementia. J Clin Psychiatr 1979; 40: 447-449.
52. Liberini P, Flaglia L. Hysteria - 100 years after Charcot. Can J Psychiatr 1993; 38: 237-238.
53. Ron MA, Toone BK, Garralda ME, Lishman MA. Diagnostic accuracy in presenile dementia. Br J Psychiatr 1979; 134: 161-168.
54. Breuer J, Freud S. The psychic mechanism of hysterical phenomona from Studies in Hysteria. Nervous and Mental Disease Publishing Co. N.Y. 1936: 1-13.
55. Marmor J. Orality in the hysterical personality. J Am Psychoanal Assoc 1953; 1: 656-672.
56. Easser B, Lesser S. Hysterical personality: a re-evaluation. Psychoanal Quart 1965; 344: 390-413.
57. Aufreiter J. Psychiatric nosology and hysteria. Can Psychiat Assoc J 1969; 14: 569-577.
58. Munford P, Liberman R. Behaviour therapy of hysterical disorders. *In:* Roy A, ed. New-York: Hysteria. John Wiley and Sons Ltd., 1982; 287-303.
59. Celami D. An interpersonal approach to hysteria. Am J Psychiatr 1976; 133: 1414-1418.
60. Bird J. The behavioral treatment of hysteria. Br J Psychiatr 1979; 134: 129-137.
61. Slater E. Diagnosis of hysteria. Br Med J 1965; I: 1395-1399.
62. Merskey H, Burich, N. Hysteria and organic brain disease. Br J Med Psychol 1975; 48: 359-366.
63. Roy A. Hysteria: A case note study. Can J Psychiatr 1979; 24: 157-160.
64. Ludwig A. Hysteria: A neurological theory. Arch Gen Psychiatr 1972; 27: 771-777.
65. Flor-Henry P, Fromm-Anch D, et al. A neuropsychosocial study of the stable syndrome of hysteria. Biol Psychiatr 1981; 16: 601-626.
66. Meyer B-U, Britton T, Beneke R, et al. Motor responses evoked by magnetic brain stimulation in psychogenic limb weakness: diagnostic valve and limitations. J Neurol 1992; 239: 251-255.
67. Guze S. Studies in hysteria. Can J Psychiatr 1983; 28: 434-437.
68. Torgersen S. Genetics of somatoform disorders. Arch Gen Psychiatr 1986; 43: 502-505.
69. Ljungberg L. Hysteria: a clinical prognostic and genetic study. Acta Psychiat Neurol Scand 1957; 32: Suppl 112.
70. Shields J. Genetical studies of hysterical disorders *In*: Roy A, ed. Hysteria. New York: John Wiley & Sons Ltd, 1982: 41-56.
71. Slater E. The thirty-fifth Maudsley lecture: Hysteria 311. J Ment Sci 1961; 107: 359-381.
72. Guze S, Woodruff R, Clayton P. Sex, age and the diagnosis of hysteria (Briquet's Syndrome). Am J Psychiatr 1972; 129: 745-748.
73. Guze S, Woodruff R, Clayton, P. A Study of conversion symptoms in psychiatric outpatients. Am J Psychiatr 1971; 128: 643-646.
74. Guze S, Woodruff R, Clayton, P. Hysteria and antisocial behaviour: further evidence of an association. Am J Psychiatr 1971; 127: 957-960.
75. Simon GE, Vonkorff M. Somatisation and psychiatric disorder in the NIMH epidemiologic catchment area study. Am J Psychiatr 1991; 148: 1494-1500.
76. Tomasson K, Kent D, Coryell W. Somatisation and conversion disorder: comorbidity and demographics at presentation. Acta Psychiat Scand 1991; 84: 288-293.
77. Murphy G. The clinical management of hysteria. JAMA 1982; 247: 2559-2564.
78. Folks DG, Hawck CA. Somatoform disorders, factitious disorders and malingering. *In*: Stoudemire A, Fogel BS, eds. Psychiatric Care of the Medical Patient. New York: University Press, 1993; 267-287.
79. Adams RD, Victor M. Principles of Neurology (4th ed). McGraw-Hill, N.Y. 1989; 1197-1198.
80. Sargant W, Slater E. An Introduction to Physical Methods of Treatment in Psychiatry. 5th ed. London: Churchill Livingstone, 1972; 145-149.
81. Goldberg D, Gask L, O'Dowd T. The treatment of somatisation: teaching techniques of reattribution. J Psychosom Res 1989; 33: 689-695.
82. Sternbach R. Pain Patients: Traits and Treatments. NY: Academic Press, 1974: Ch IX.
83. Miller H. Accident neurosis. Br Med J 1961; 1: 919-925; 992-998.
84. Tarsh M, Royston C. A follow-up study of accident neurosis. Br J Psychiatr 1985; 146: 18-25.
85. Binder RL, Trimble MR, McNeil DE. The course of psychologic symptoms after resolution of lawsuits. Am J Psychiatr 1991; 148: 1073-1075.
86. Benson H, Beary J, Carol M. The relaxation response. Psychiatry 1974; 37: 37-46.
87. Kiloh L, Smith J, Johnson, G. Physical Treatments in Psychiatry. Boston: Blackwell Scientific Pubs, 1988: 117-160.
88. Potter WZ, Rudorfer MV, Manji H. The pharmacologic treatment of depression. N Engl J Med 1991; 325: 633-642.
89. Sharav Y, Singer E, et al. The analgesic effect of amitryptiline on chronic facial pain. Pain 1987; 31: 199-209.