INVITED ESSAY*

HYPOCHONDRIASIS

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Summary—Hypochondriasis and other syndromes in which health anxiety is prominent are frequently seen in clinical practice and often pose problems of management. In contrast with other anxiety problems, the conceptualization and treatment of health anxiety has developed very little in recent years. Behavioural approaches to treatment have only recently been applied. In the present paper, current theoretical models are critically evaluated and a cognitive–behavioural approach is proposed to account for the development and maintenance of hypochondriacal problems. Principles of cognitive–behavioural treatment based on this approach are outlined, together with some suggestions for research.

The last decade has seen the successful application of behaviour therapy to branches of medicine previously considered to fall outside the province of psychology. Development of this field, usually known as behavioural medicine, was firmly founded upon experimentally validated principles of assessment and treatment for anxiety disorders (Rachman & Philips, 1975). The continued and rapid progress of behaviour therapy in both behavioural medicine and anxiety disorders is well recognised (see, for example, Brownell, 1984; Hawton, Salkovskis, Kirk & Clark, 1989; Wilson, 1984). It is therefore surprising that the behavioural approach has seldom been applied to that substantial proportion of patients who seek medical consultation with complaints related to anxiety about health and its more extreme manifestations, illness phobia and hypochondriasis (e.g. between 20 and 84% of those attending physicians and surgeons; Kellner, 1985). There are probably two main reasons for the neglect of hypochondriasis by those involved in the application of behavioural approaches. Firstly, the field has been influenced by the traditional psychiatric perspective that hypochondriasis is usually secondary to other conditions, particularly affective disorders. Secondly, early behavioural formulations relied on conditioning theory and the presence of readily identifiable external stimuli—such approaches are not easily applied to hypochondriasis. In this paper, the current state of our knowledge of primary hypochondriasis is reviewed. A cognitive–behavioural formulation of the development and maintenance of health anxiety and hypochondriasis is outlined, and considered in terms of a three systems analysis. The applications of the model to assessment and treatment are described, and the specific implications for future research considered.

THE CONCEPT OF HYPOCHONDRIASIS

The essential feature of hypochondriasis is preoccupation with a belief in or fear of having a serious illness. This occurs 'without adequate organic pathology' to account for the reaction, and 'despite medical reassurance'. Such fears are associated with the perception of bodily signs and sensations which are misinterpreted as evidence of serious illness. Primary hypochondriasis is a recognised diagnosis in ICD 9 (WHO, 1978) and DSM III R (American Psychiatric Association, 1987). Secondary hypochondriacal symptoms occur in a variety of other psychiatric conditions. Health anxiety also occurs in a range of non-psychiatric Ss, including the physically ill. Patients suffering from health anxiety are a drain on resources in every area of medical practice (Katon, Ries & Kleinman, 1984; Kellner, 1985; Mayou, 1976). Available treatments for primary hypochondriasis have not been shown to have any impact on prognosis, which is generally poor.

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Hypochondriasis: primary or secondary?

The neglect of hypochondriasis may be due to the widespread belief that hypochondriasis is secondary to other disorders, particularly depression (Kenyon, 1965). The primary/secondary distinction has major implications for the assessment and management of patients presenting with hypochondriacal symptoms. Kenyon (1964) examined the notes of 512 cases seen at the Bethlem Royal and Maudsley Hospitals, dividing them into either primary or secondary hypochondriasis based on the notes made at the time of their admission. Three hundred and one cases were diagnosed as primary with 211 diagnosed as secondary to another condition. These groups were compared on a number of variables to see whether there was any justification for a diagnosis of primary hypochondriasis. The results were interpreted as indicating that there was no difference between these conditions. As secondary hypochondriasis was most frequently associated with depressive conditions, Kenyon suggested these results meant that hypochondriasis is always part of another syndrome, usually an affective one.

This study must be interpreted with caution particularly because it was based on the retrospective examination of casenotes and no operationally defined diagnostic criteria were utilized. An unexpectedly high proportion of patients were given an initial diagnosis of primary hypochondriasis. The primary group showed a poorer response to inpatient treatment compared with the secondary group and with the general outcome of all patients treated at the joint hospitals over the same period. It is not justifiable to conclude, on the basis of this study, that primary hypochondriasis does not exist. The results of other studies support the view that there is a primary syndrome of hypochondriasis. Pilowsky (1970) personally evaluated 147 cases, and diagnosed 66 as primary and 81 as secondary. The primary cases had longer histories when first referred, fewer suicide attempts and had received less electro-convulsive therapy, anti-depressant and ‘sedative’ medication. Bianchi (1971) studied 235 psychiatric inpatients using more objective assessments and diagnosed 30 as cases of ‘disease phobia’, which was not secondary to any other condition.

These studies are probably unrepresentative because they deal with a particular sample of hypochondriacal patients. It is likely that the majority of hypochondriacal patients are dealt with in non-psychiatric settings (Bridges & Goldberg, 1985) because, by definition, they are reluctant to regard their problems in any other than physical terms. This means that hypochondriacal patients accepting referral to psychiatric services are likely to be atypical of the population of patients fulfilling diagnostic criteria, because these patients have come to accept that they are suffering from some kind of psychological disturbance (e.g. debilitating depression) in addition to the hypochondriacal concerns. Hypochondriacal patients seen in psychiatric clinics may complain of being depressed because they believe that their ‘physical illness’ has neither been diagnosed nor treated properly. The patients' recognition of this psychological disturbance, whether it is accurately attributed or not, will be a crucial factor in their acceptance of psychiatric referral, resulting in referral of a selected population.

In the evaluation of the primary–secondary distinction, it would be most appropriate to consider the chronological development of hypochondriacal and depressive symptoms. This approach has been utilized in obsessive-compulsive disorder. Although patients with obsessive-compulsive disorder may not differ on demographic variables from cases of depression with secondary obsessional features, the existence of primary obsessive-compulsive disorder is not in doubt. The crucial consideration in the clinical distinction between primary and secondary problems is the sequence in which they develop (Gittleson, 1966). Systematic examination of this issue in hypochondriacal patients is required.

A further complication is that patients with long standard hypochondriacal concerns may be labelled as ‘personality disorders’. There have been no satisfactory studies in which specific patterns of personality have been identified (Kellner, 1989). Such patients may be ‘dissatisfied’ and ‘hostile’ due to the failure to obtain a resolution of their problems or even a satisfactory account of their problems.

Characteristics of health anxiety and hypochondriasis

Anxiety about health may occur transiently in normal Ss and can be a secondary phenomenon in a variety of other conditions. Extreme health anxiety or hypochondriasis may present as a
Hypochondriasis is a morbid preoccupation, an obsessive-compulsive phenomenon and (rarely), be of delusional intensity (Pilowsky, 1984). Kellner et al. (1987) used questionnaires to examine attitudes, fears and beliefs in 21 cases fulfilling DSM-III criteria for hypochondriasis and matched non-hypochondriacal controls. The results showed that hypochondriacal patients differed from anxious and depressed psychiatric patients by reporting more fears of and false beliefs about disease, more attention to bodily sensations, more frequent fears of death and more distrust of physicians’ opinions, although they sought more medical care than did other Ss in the study.

Pilowsky (1967) administered a standardized questionnaire to 100 cases of hypochondriasis and 100 control Ss. The responses were used in a principal components analysis and three factors were identified as reflecting three dimensions of hypochondriasis—bodily preoccupation, disease phobia and conviction of the presence of disease with non-response to reassurance. Consistent with such a view, Kellner (1985) subdivides hypochondriasis into an unrealistic fear of disease (or illness phobia) and the conviction of having a disease. A variety of alternative classifications have been suggested, with most recognizing some distinction between hypochondriasis and illness phobia (Bianchi, 1971; Leonhard, 1968; Marks, 1987; Mayou, 1976; Pilowsky, 1984; Ryle, 1947). At present, little is known about the relative importance of factors such as avoidance and reassurance seeking in these groups.

Studies comparing the treatment of disease conviction and disease phobia may clarify the extent to which existing behavioural strategies (such as graded exposure or anxiety management techniques) can be applied to health anxiety. It may be theoretically and therapeutically important to consider whether particular characteristics of hypochondriacal patients arise from psychological processes that are also important in other, more readily manageable conditions. There is preliminary evidence that therapy based on principles of exposure may be effective in those patients who show a phobic pattern of behaviour (Warwick & Marks, 1988). It is not clear at this stage whether such treatment would be less effective in patients with stronger disease conviction. In an even more striking fashion, the definition of hypochondriasis suggests a similarity with current cognitive–behavioural conceptualization of panic attacks, in which the misinterpretation of bodily sensations is also a crucial element.

Panic attacks, misinterpretations and health anxiety

In panic attacks, avoidance and preoccupation with thoughts of physical/mental disease are particularly prominent (Clark, 1986, 1988; Salkovskis, 1988). Barlow et al. (1985) showed that panic co-existed with a variety of other DSM-III anxiety disorders, but did not consider hypochondriasis (cf. Wolpe, 1986). In a recent study we found that 59% of hypochondriacal patients also fulfilled DSM-III criteria for panic disorder (Salkovskis, Warwick & Clark, unpublished data). This finding is consistent with other evidence supporting the hypothesis that panic arises from the catastrophic (usually illness related) misinterpretation of bodily sensations (Clark, 1986, 1988; Clark et al., 1988; Salkovskis & Clark, 1986). Such misinterpretations may lead to avoidance of situations which are likely to trigger panic attacks and if these attacks are usually experienced outside the home, an agoraphobic picture may develop (Thyer, 1986). The relationship between specific misinterpretations and panic has been documented (Rachman, Lopatka & Levitt, 1988); Salkovskis (1988) proposes that avoidance represents the patient’s attempt to deal, cope with or avert the catastrophe which he or she perceives as being about to occur. Thus, panic patients who interpret dizziness and faintness as sign that they are about to faint will try to sit down and will avoid areas where there is no seating and which have hard floors, would tend to avoid situations where fainting would be particularly embarrassing and so on. Therefore, patterns of escape and avoidance related to illness are likely in panic patients. In hypochondriacal fears, such escape behaviour should differ from that associated with panic because, although in both instances the behaviour results from an anticipated disaster, the anticipated harm is perceived as much less imminent in hypochondriacal ideas (see below). This difference gives the hypochondriacal patient more time to prevent the anticipated disaster by seeking medical attention. It seems very likely that these different patterns will co-exist in many patients if both hypochondriacal and panic ideation are present.

Bianchi (1971) found that agoraphobia was significantly more common in disease phobics than in a matched control group of patients who had no health anxiety. In a study of agoraphobic
housewives (Buglass et al., 1977) concern with the possibility of physical collapse was common among patients when in their feared situation. Examination of attitudes to health when not in phobic situations showed that 16 or 30 Ss had persistent worries about health compared with none of the controls. Of these cases, 10 viewed their concerns as irrational, 3 had 'fluctuating insight' and 3 were convinced that they were physically ill. Noyes et al. (1986) found that hypochondriacal concerns as measured by questionnaire were prominent amongst a group of panic disorder patients. The scores were similar to norms reported for hypochondriacal patients and declined significantly after treatment of panic. Katon's study of 55 patients with panic disorder (1985) found that 89% of the patients initially presented with somatic complaints; misdiagnosis of a physical cause of these was common, suggesting that panic may be an important cause of 'somatization' in primary care settings.

There are important differences between the way misinterpretations affect panic and hypochondriacal patients. Panic patients tend to misinterpret autonomic symptoms, most commonly (but not invariably) those involved in acute anxiety responses, thus providing an obvious feedback mechanism by which anxiety may rapidly escalate. An example would be someone who noticed that his/her heart was pounding, and interpreted this as a sign that they were having a heart attack; this thought would make them anxious, increase their heart rate and so on round in a vicious circle culminating in a panic attack. Another class of symptoms which can be involved in the same way are anxiety-related mental sensations often interpreted as signs of 'losing control', such as racing thoughts, confusion or mental blanks. In hypochondriacal problems, the symptoms misinterpreted are more likely to be those which are not subject to such direct amplification, e.g. lumps and blemishes. The feedback mechanism is more likely to be behavioural and longer term; for instance, maintaining focus on particular parts of the body by repeated checking, increasing sore areas by physical manipulation.

The second difference concerns the time scale of feared catastrophes. Both panic and hypochondriacal patients interpret bodily sensations as indicators of catastrophic physical or mental illness. However, panic patients believe that this is happening already, or about to happen in a few moments, whereas hypochondriacal patients tend to believe that the symptoms indicate a more insidious course. Panic patients typically fear conditions which occur suddenly, such as heart attacks and strokes as opposed to hypochondriacal fears such as multiple sclerosis or cancer.

Theories of hypochondriasis

There is no empirical evidence to support the numerous psychodynamic conceptualizations of hypochondriasis which have been proposed (Kellner, 1985). The suggestion that interpersonal rewards ('secondary gain') are important in the maintenance of the condition is widespread in clinical practice. This 'hypothesis', whilst superficially appealing, is inconsistent with the learning theory principles from which interventions are derived (e.g. Goldiamond, 1976). It has pejorative connotations, sometimes resulting in the easy (for the clinician) but distressing (for the patient) conclusion that their problems serve a 'necessary function'. Such a view commonly detracts from a careful analysis of the phenomena exhibited in individual cases and has no empirical support.

Cognitive misinterpretation of bodily sensations is assumed to be of importance because it forms part of the definition of hypochondriasis. However, there is little experimental evidence showing that hypochondriacal patients differ from other anxious patients either in their perception or misinterpretation of normal bodily sensations. An important study by Tyrer et al. (1980) examined the awareness of pulse rate in cases of hypochondriasis, anxiety neurosis and phobic anxiety. Subjective ratings of pulse rate were compared with ECG recordings taken during films designed to induce varying levels of anxiety. There was a significantly higher correlation between subjective and measured pulse rates in cases of hypochondriasis and anxiety neurosis than in cases of phobic anxiety. Patients who originally expressed cardiac concerns had the highest awareness of pulse rate.

Nemiah (1977) has proposed that hypochondriacal patients may suffer form biologically determined alexithymia, a neurophysiological incapacity to experience emotion. No such deficit has been identified, and there is no other evidence supporting a categorical/disease model of hypochondriasis as distinct from less severe forms of anxiety about health. A cognitive–behavioural hypothesis, in which hypochondriasis represents an extreme manifestation of a number of normal attitudes and behaviours is described in detail below. Biological factors (for instance, a hypothetical
tendency to show more fluctuations in bodily state than average) may serve to predispose to hypochondriasis, but seem unlikely to be either necessary or sufficient to account for the clinical picture.

**TREATMENT**

Primary hypochondriasis is currently regarded as very difficult to treat, although there are no controlled studies of treatment. Ladee (1966) reported a series of 23 cases treated with psychotherapy or psychoanalysis and reported “satisfactory to good” improvement in only 4. In Kenyon’s (1964) retrospective study of the clinical management of hypochondriasis, a wide range of treatments were used, particularly drugs, E.C.T. and psychotherapy. Forty per cent of the primary group were rated as ‘no change or worse’ on discharge, compared with 15% of the secondary cases. The widespread and poorly founded view that hypochondriasis does not exist as an independent syndrome led to the use of diagnoses such as atypical or masked depression (Lesse, 1967). Thus, treatment has often been as for affective disorder, although there is no evidence to suggest antidepressant drug treatments are useful in primary hypochondriasis.

Pilowsky (1968) treated and followed up 147 cases of hypochondriasis over 31 months. He found that 48% had a good outcome, 28% a fair outcome and 24% a poor outcome. This study is uncontrolled and used a ‘wide variety of treatments, both physical and psychological’—28% of the sample in fact received electroconvulsive therapy. It is not clear if the cases were of primary or secondary hypochondriasis; note that a good prognosis was found in cases having symptoms of anxiety and depression. Kellner (1983) described a study of 36 patients who had had, what was described as ‘hypochondriacal neurosis’ for 6 months or longer. The study was of long duration and the treatments changed as new research emerged, it included individual psychotherapy focussed on accurate information about the symptoms, selective perception of symptoms and persuasion. The patients also received repeated physical examinations and reassurance and were given anxiolytic drugs if severely distressed, 64% were rated as either recovered or improved. The sample was uncontrolled and it is unclear which parts of the treatment package were given to those patients who improved. Supportive measures remain the mainstay of routine treatment in primary hypochondriasis (Gelder, Gath & Mayou, 1984). If the presence of a primary condition, such as depression, is established, it is thought that its treatment should deal effectively with secondary hypochondriacal symptoms (Burns, 1971; Lesse, 1967; Stenback & Rimon, 1964). Results of a recent outcome study confirmed this view (Kellner, Fava & Lisansky, 1986). It is not clear whether direct attention to the hypochondriacal symptoms is still necessary in a proportion of cases.

In view of the well documented behavioural correlates of both hypochondriasis and illness phobia (Marks, 1981; Salkovskis & Warwick, 1986; Warwick & Salkovskis, 1985), the marked anxiety component (Wolpe, 1986) and the lack of current successful treatment strategies, it is surprising that more attention has not been paid to a possible role for cognitive behavioural treatment in such conditions. The only reports in primary hypochondriasis are of treatment of a small number of cases with systematic desensitization, thought stopping, hypnosis, implosion (see Kellner, 1985) or graded exposure and response prevention (Warwick & Marks, 1988). Recent work has pointed out the similarities between obsessive–compulsive disorder and preoccupation with health (Marks, 1981; Warwick & Salkovskis, 1985) and Marks (1978) suggests that illness phobia should be treated along the same lines as other phobias. As part of a study reporting successful treatment of two cases employing elements of exposure and response prevention as part of a comprehensive cognitive behavioural intervention, Salkovskis and Warwick (1986) also demonstrated a functional similarity between reassurance seeking and obsessional ritualising (cf. Rachman, de Silva & Roper, 1976). In another case series of patients with fears concerning AIDS (Miller, Green, Farmer & Carroll, 1985), 7 cases showed significant improvement after cognitive–behavioural treatment. This study and related work on cognitive–behavioural approaches to panic and obsessive–compulsive disorder suggest that cognitive–behavioural treatments may be useful in hypochondriasis given an appropriate theoretical foundation. Some of the key differences between obsessional ruminations and morbid preoccupations have already been identified by Rachman (1974); many of these considerations can be applied to hypochondriasis, which often takes a form similar to obsessional thinking without the perception of senselessness.
A cognitive–behavioural approach

From a cognitive perspective, anxiety occurs because a particular situation or stimulus encountered is judged to involve an element of threat and one's ability to effectively cope with the perceived threat is doubted (Beck, 1976). The cognitive hypothesis of health anxiety and hypochondriasis proposes that bodily signs and symptoms are perceived as more dangerous than they really are, and that a particular illness is believed to be more probable than it really is (Salkovskis, 1989; Salkovskis & Warwick, 1986; Warwick & Salkovskis, 1989). At the same time, the patient is likely to perceive himself as unable to prevent the illness, and unable to affect its course, i.e. as having no effective means of coping with the perceived threat.

Figure 1 illustrates how the cognitive hypothesis accounts for the development of hypochondriasis. Knowledge, and past experiences, of illness (in self or others) leads to the formation of specific assumptions about symptoms, disease and health behaviours. These are learned from a variety of sources, particularly from early experience (cf. Bianchi, 1971), but also from events in the patient's social circle or the mass media. Previous experience of physical ill-health in patients and in their families and previous experience of unsatisfactory medical management may be important (see Bianchi, 1971). A further factor is the information carried by the media. A striking example is provided by the influx of cases of 'AIDS phobia' (Miller et al., 1985; Miller, Acton & Hedge, 1988) noted after the recent massive publicity campaign on this topic.

Examples of potentially problematic assumptions are “bodily changes are usually a sign of serious disease, because every symptom has to have an identifiable physical cause”; “if you don’t go to the doctor as soon as you notice anything unusual then it will be too late”. Other beliefs relate to specific personal weaknesses and particular illnesses; for example, “there’s heart trouble in the family”, “I’ve had weak lungs since I was a baby”. Such beliefs may be a constant source of anxiety and/or may be activated in vulnerable individuals by critical incidents. Assumptions can also lead the patient to selectively attend to information which appears to confirm the idea of having an illness, and to selectively ignore or discount evidence indicating good health. Thus, particular assumptions often lead to a confirmatory bias in the patient’s thinking once a critical incident has resulted in the misinterpretation of bodily symptoms and signs as being indications of serious illness. Situations which constitute critical incidents and activate previously dormant assumptions include unfamiliar bodily sensations, hearing details of illness in a friend of a similar age, or new information about illness. Further bodily sensations may then be noticed as a consequence of increased vigilance arising from anxiety. In patients who become particularly anxious about their health, such situations are associated with thoughts which represent personally catastrophic interpretations of the bodily sensations or signs. Catastrophic interpretations can in turn lead to one of the two patterns of anxiety. If the sensations or signs are not those which occur as a result of anxiety (as a consequence of autonomic arousal), or the patient does not regard the feared catastrophe as immediate, then the reaction will be hypochondriacal anxiety about health, with the cognitive, behavioural, physiological and affective correlates as detailed in Fig. 1 (e.g. “The pains in my stomach mean I have an undetected cancer”). On the other hand, if the symptoms which are misinterpreted are those which occur as part of anxiety-induced autonomic arousal and the interpretation is that the symptoms are the signs of immediate catastrophe (e.g. “these palpitations mean that I am having a heart attack right now”), a further immediate increase in symptoms will result. If this process continues, then a panic attack is the more likely response (Clark, 1988; Salkovskis, 1988). Despite the differences in type of symptoms and time course of feared illness, the ideation in panic and hypochondriasis is similar and the two presentations often overlap (see Noyes et al., 1986).

MAINTENANCE

Once anxiety about health has developed, other mechanisms may be involved in the maintenance of the problem.

(1) Anxiety about health matters and symptoms themselves is likely to result in physiological arousal. Patients often misinterpret increased autonomic symptoms as further evidence of a physical disease.
Hypochondriasis

Previous Experience
Experience and perception of:
(i) Illness in self, family: medical mismanagement
(ii) Interpretations of symptoms and appropriate reactions

"My father died from a brain tumour."
"Whenever I had any symptoms I was taken to the doctor in case it was serious."

Formation of dysfunctional assumptions
"Bodily symptoms are always an indication of something wrong; I should always be able to find an explanation for my symptoms."

Critical incident
Incident or symptom which suggests illness
"One of my friends died of cancer a few months ago: I have had more headaches recently."

Activation of assumptions

Negative automatic thoughts/imagery
"I could have a brain tumour. I didn't tell the doctor that I have lost some weight. It may be too late. This is going to get worse. I will need brain surgery."

HEALTH ANXIETY, HYPOCHONDRIASIS

BEHAVIOURAL
Avoidance and self-imposed restrictions
Repeated self-inspection
Repeated manipulation of affected area
Consultation, reassurance seeking
Scanning for information
Preventative measures

AFFECTIVE
Anxiety
Depression
Anger

PHYSIOLOGICAL
Increased arousal
Changes in body function
Sleep disturbance

COGNITIVE
Focus on body and enhanced bodily perception
Monitoring of body changes
Attention to negative information
Helplessness
Preoccupation, rumination
Discounting positive information

Fig. 1. Cognitive model of the development of severe health anxiety.

(2) Selective attention to illness-related information, such as the perception of normal bodily changes (e.g. gastric distention after eating) or previously unnoticed bodily features (e.g. blotchy complexion) is often important. Focussing prompted by worries about health brings slight bodily variations to awareness at times when ideas about illness are already present, leading to a bias towards noticing information that is consistent with the worries about illness, and with a pre-existing confirmatory bias (see p. 110).

(3) Behaviour designed to avoid, check for, or totally exclude, physical illnesses (e.g. avoiding physical exertion, contact with disease, reading medical textbooks, frequent medical consultations, bodily checking, manipulation and inspection) will maintain anxiety. However, unlike the more
immediate misinterpretations of panic patients, hypochondriacal misinterpretations allow the patient more scope to seek safety by attempts to obtain a medical resolution of the perceived threat to health. Behaviour occurring as a consequence of anxiety can increase anxiety in a variety of ways. Firstly, it keeps attention focussed on fears about health and can therefore result in elaboration of those fears. Secondly, it can increase the range and scope of catastrophic interpretations.

These mechanisms can sustain preoccupation with health and exacerbate hypochondriacal symptoms as shown in Fig. 2. Consistent with a dimensional approach based on cognitive vulnerability augmented by cognitive and behavioural mechanisms, transient examples of these processes are seen in any individual gaining access to new information regarding health risks (e.g. medical students perceiving symptoms of diseases under study, public reaction to media coverage of new medical dangers, patients undergoing or waiting for the results of diagnostic tests). The selective attention and checking behaviour serve to sustain long term anxiety in the same way as neutralization in obsessional disorder.

Three systems

Cognitive–behavioural hypotheses of anxiety related conditions utilise a 3-systems analysis as a useful way of conceptualizing clinical phenomena (Lang, 1970). The following, more detailed analysis of these factors considers physiological, behavioural and cognitive aspects of psychopathology as loosely coupled systems requiring separate analysis.

Cognition. Appraisal of potentially threatening cues (such as bodily sensations and intrusive thoughts about possible hazards to health) together with an evaluation of being unable to cope with such threat can lead to health anxiety. Such preoccupation with health, associated with perceptual amplification and misinterpretation of bodily sensations, is a key cognitive element in such patients (see also Barsky, Geringer & Wool, 1988). Consultation will not be sought simply because of the handicap, inconvenience and physical discomfort arising directly from the symptoms, but will also be due to the presence of a variety of anxiety-provoking cognitive events. We suggest that these are intrusive thoughts about the possible causes of the symptoms—e.g. the

Fig. 2. Hypothesized maintaining factors in severe health anxiety.
patient who feels his stomach pains might be due to carcinoma is more likely to seek advice than the patient who decides that they can be explained by an innocuous, temporary problem, e.g. indigestion. Intrusive thoughts of the possible negative consequences of not taking further action are also important, in terms of future handicap, inconvenience and physical discomfort and the possible irreversible progression of the feared underlying cause, e.g. the patient may fear that stomach pains will become excruciating and that their cause may be untreatable. The patient may also experience discomfort at the negative consequences of taking further action, in terms of painful or noxious medical procedures. Cognitions can thus have a major impact on illness related behaviour.

We have also identified several cognitive factors which are likely to affect anxiety in relation to medical consultation. Some of these factors will increase anxiety in both the short and long term; e.g. previous experience of related diagnoses (“My father died of a heart attack”); high anxiety and threat perception (“I know it is silly now, but when I get the symptoms I’m really frightened and I’m sure I’m dying”); misperception of doctors’ communication (“the doctor said it was something to do with my white blood cells; the Readers Digest says that’s to do with leukaemia”). This misperception can be further increased by confirmatory bias in which patients selectively attend to information which is consistent with the negative interpretation of their condition. This often affects the way in which patients understand conversations with others, particularly health professionals.

Other factors affect anxiety in the longer term only; e.g. ineffectiveness of interventions (“the doctor said that a couple of aspirins would stop the pain, but I’ve had 4 now and it’s no better”); expected failure of communication (“it’s no good when I go, because I can’t explain my problems to the doctor”). It would seem likely that such factors will serve to maintain and increase the range of negative interpretations of health related perceptions.

Although imagery has recently been increasingly recognized as an important cognitive phenomenon in anxiety disorder (Beck, Emery & Greenberg, 1985), little is known about its role in health anxiety. It has been clearly established that fleeting, unpleasant, anxiety related images occur in normal clinical populations (de Silva, 1986), and it would therefore seem likely that these specifically occur in health anxiety. Such imagery might well increase the accessibility and elaboration of cognitions regarding the seriousness and likelihood of feared catastrophe, and clearly requires further elucidation.

Work by Butler and Mathews (1983, 1986) and Lucock and Salkovskis (1988) supports the view that increased anxiety has the effect of increasing the perceived probability of negative outcomes, both for events related to the origin of the original anxiety and for other, unrelated events. These studies suggest that life stresses and associated anxiety might increase the perceived probability of illness, and may be a further mechanism by which stress can result in hypochondriacal ideas.

**Behaviour.** An important and often neglected aspect of the behaviour of hypochondriacal patients is effort made to prevent exposure to anxiety provoking cues. Such cues are likely to provoke thoughts of serious illness, and hence health anxiety. This may result in a wide range of avoidant behaviours which may not be readily recognizable, e.g. frequent visits to hospitals for reassurance, restriction of physical effort. Such behaviour includes a wide range of actions intended to check on health state. Patients whose anxieties lead to catastrophic interpretations of symptoms may not be satisfied by the clinical diagnosis and repeatedly seek reassurance about their symptoms. Repeated attempts at providing such reassurance will have the same effect as compulsive behaviour in obsessions, that is, the immediate relief of anxiety followed by its return in the longer term and increased frequency of such requests (see Salkovskis & Warwick, 1986, for a demonstration of the paradoxical effects of reassurance). Repeated seeking of reassurance maintains the pre-occupation, anxiety and bodily sensations. Reassurance can be sought in a variety of subtle ways, including repeated checking on bodily appearance, functions and symptoms by the patients themselves, reading medical textbooks and discussion of symptoms with family and other social contacts.

Longer term alterations in behaviour may include special activities which they feel will protect them from, or prevent, the progression of the feared illness, e.g. a cholesterol free diet. Repeatedly bodily checking can sometimes be so extensive that it causes physical changes commensurate with
the feared harm; e.g. repeated rubbing of a painful area to check if it is still painful, leading to inflammation and continued discomfort.

**Physiology.** Physiological factors are well known to potentiate cognitive effects and vice versa (Salkovskis & Clark, 1990; Schachter & Singer, 1962). Although it is widely assumed that hypochondriacal patients both perceive more and augment physiological symptoms (cf. Barsky & Klerman, 1983; Kellner, 1985), there is as yet no evidence to support this. Few studies have been performed in clinical populations, although a number have considered experimentally manipulated variables such as critical flicker fusion in which the relevance to clinical hypochondriasis is questionable. A notable exception is that of Tyrer et al. (1980) who showed that hypochondriacal patients, although different from phobic patients in the accuracy of their perception of heart rate, were not different from anxiety states.

Thus, physiological responses can trigger illness related thoughts (e.g. palpitations leading to the thought "my heart is about is about to stop"). These and other triggers may lead to scanning or searching for further symptoms (e.g. hearing about an acquaintance who has developed heart disease makes the patient aware of their own pulse at a time when they are already anxious, leading to the thought "my pulse is too fast—it could happen to me"). The organ systems most commonly implicated are gastrointestinal, musculo-skeletal and central nervous; the most commonly involved regions of the body are the head and neck, abdomen and chest, and the most common symptom is pain (Barsky & Klerman, 1983). Any benign changes, e.g. rashes and swellings may become the focus of anxiety and distress.

**Implications of a cognitive approach to assessment and treatment**

Cognitive-behavioural treatment should involve the clear and positive formulation problems in cognitive, physiological and behavioural terms and proceed by the direct testing of such psychological hypotheses. This approach is consistent with the growing body of effective psychological treatments. Cognitive and behavioural treatments are particularly helpful as they are open to scientific investigation, and are already validated in conditions such as anxiety, panic and depression.

The principal obstacle to the treatment of hypochondriacal patients is their reluctance to view their problems as being caused by anything other than a medical condition. Psychological treatment therefore depends on persuading the patient to engage in treatment. Cognitive techniques can be employed in a variety of ways of rapidly facilitate this process, usually by engaging the patient in treatment as an hypothesis testing exercise (Salkovskis, 1989; Warwick & Salkovskis, 1989). Once the patient is engaged, treatment should have two major components:

(i) Identification and modification of automatic thoughts and dysfunctional assumptions about health through the use of techniques derived from cognitive therapy for anxiety (Beck et al., 1985). The most helpful techniques include reviewing the patient's evidence for physical illness, then identifying testable alternative explanations for the symptoms experienced by the patient. Providing the patient with an account of the source of their symptoms then verifying the alternative account is a more effective way of correcting misinterpretations than procedures intended to prove that the patient does not have the feared illness.

(ii) Abnormal behaviour has to be identified and modified appropriately. The correct management of persistent requests for reassurance seeking in such cases is central to the approach and is particularly controversial. It is generally accepted that reassurance seeking functions as a compulsive ritual in obsessive–compulsive disorder (Rachman & Hodgson, 1980). It has been suggested that it functions in the same way in health preoccupation (Warwick & Salkovskis, 1985), a view supported by some preliminary data on the short and long term effects of reassurance provision (Salkovskis & Warwick, 1986). It follows therefore that such inappropriate reassurance seeking and repeated provision of information which the patient already has, should be prevented, and used as the basis for re-attribution of the type described in (i) above. Provision of appropriate 'reassurance can be helpful (Mathews & Ridgeway, 1982), and is defined as the provision of new information. Consistent with this component of treatment, several authors have proposed an important role for reassurance, although the possible mechanisms have not been discussed, e.g. Kenyon cites the views of Wychoff (1928) who stated that hypochondriasis is largely iatrogenic in
the sense of being initiated or perpetuated by doctors, particularly by those ordering further physical investigations "just to make sure".

Subtle and persistent ways of seeking reassurance can evolve, as recognised by Leonhard (1968) who notes "any discussion of the state of health will only be of disadvantage to the hypochondriac...these constant discussions must be stopped at all costs". This view is not supported by Pilowsky or Kellner; the latter states that "treatment strategies include repeated physical examination when the patient fears he has acquired a new disease...and repeated reassurance". Resolution of this issue is urgently required. It seems most unlikely that this will be a useful strategy in patients fulfilling DSM III-R criteria, since by definition these patients' "fear or belief persists despite medical reassurance and causes impairment in social; or occupational functioning". This highlights the urgent need for a careful definition of reassurance. As outlined above, reassurance can be offered in a variety of ways, some of which should be helpful, others which may actually increase anxiety. In some cases this criterion might be better revised: fear or belief can persist because of medical reassurance.

CONCLUSION

A great deal of work remains to clarify the psychopathology of cases of health anxiety and an adequate classification the definition of separate syndromes within the area cannot be successfully achieved until this has been done. Too much emphasis has been placed in the past on the use of drug treatments in the absence of evidence for their efficacy; cognitive–behavioural methods of treatment are promising and require evaluation. Apart from the implications for treatment, the cognitive hypothesis gives rise to a number of specific predictions. These include

(i) Hypochondriacal patients will selectively attend to and preferentially process information which is consistent with their illness beliefs.

(ii) Hypochondriacal patients will show an enduring tendency to misinterpret innocuous bodily sensations and changes as a sign of illness.

(iii) Hypochondriacal patients will be more likely than controls to endorse attitudes to symptoms which are threatening, extreme and erroneous (e.g. more likely to believe "Unfamiliar symptoms are always a sign of illness")

(iv) The onset of hypochondriasis will tend to be associated with the occurrence of illness and/or misdiagnosis in others perceived as similar to the patient.

(v) When a patient's hypochondriacal beliefs concern (a) immediately catastrophic illness and (b) the fears of illness are activated by symptoms which can increase as a result of autonomic arousal, panic attacks occur in association with the hypochondriasis.

(vi) Hypochondriacal patients will show an enduring tendency to react to bodily sensations by changes in behaviour (overt and/or covert) focussed on those sensations.

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REFERENCES


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