

INVITED REVIEW

SOMATIC PRESENTATIONS OF PSYCHIATRIC ILLNESS IN PRIMARY CARE SETTING

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INTRODUCTION

MUCH of the recent literature on somatisation has been based upon patients with chronic somatisation, or the 'somatisation disorders' of DSM-III [1-5]. Although the various surveys have produced many interesting findings, the social psychiatrist is left with nagging doubts. First, it is possible that the patients who form the subject of the various investigations are in fact a highly skewed sample of the larger population of somatising patients in the community. Thus, some of the features attributed to these patients may tell us more about the selection processes that govern referral to the specialist clinics than about the phenomenon of somatisation itself. The second problem is that by the time somatisation has lasted several years we are likely to learn more about factors which perpetuate the disorder, and rather less about the factors which brought the phenomenon about in the first place.

We have therefore concentrated our attention on patients attending primary care physicians with new episodes of illness who are found to have a psychiatric disorder according to DSM III criteria at independent interview. The focus of our research is on the factors which determine why one patient will express their distress in the language of somatic symptoms, while others will 'psychologise' their distress. In the course of our work we discover that the patients themselves, with their combinations of affective and somatic symptoms, are strikingly similar to descriptions of patients which go back as far as George Cheyne, in 1743.

EARLY CONCEPTS OF 'NEUROSIS'

The word 'neurosis' started off as a synonym for nervous disease, and therefore included conditions as diverse as coma, apoplexy and St. Vitus's Dance. However, as the organic aetiology of more and more brain diseases became understood, the concept became progressively more restricted, so that it referred to patients whose somatic symptoms could not be accounted for by known organic disease.

Used in this newer sense, the 19th century began with two major neuroses — hysteria and hypochondriasis — and one minor neurosis: neuralgia. From about 1840 onwards, physicians gave considerable emphasis to the concept of the spinal

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reflex as underlying neurotic phenomena; and concepts such as 'spinal irritability' and the 'vasomotor hypothesis' of Brown-Séguard explained the presence of various bodily discomforts as symptoms of neurosis. Theorising moved up the neuraxis as 19th century progressed so that patients with predominantly cardiac symptoms were thought to suffer with cerebrocardiac neurosis, while those with dyspeptic symptoms had cerebrogastric neurosis. The same patients fill our clinics today: only the theorising has changed.

The emergence of neurasthenia in 1870 saw an end to the myriad subdivisions of neurosis that had preceded it; all were now included in the new catchall diagnosis.

In his monograph 'American Nervousness' Charles Beard applied the vasomotor hypothesis to the brain itself, and felt able to incriminate predisposing factors such as climate and sexual abuse, as well as releasing factors such as overwork to explain illnesses affecting the American businessman with various somatic symptoms but without demonstrable organic disease. (The tendency of American taxonomists to prefer large omnibus concepts to the nitpicking European approach has modern parallels in the case of 'major depressive episode', which tends to include many cases diagnosed as anxiety states and retarded depression using other systems [6, 7].)

The concept of neurasthenia was consecrated by Charcot in his *Leçons du mardi* towards the end of the century, so that hysteria and neurasthenia were now declared the two major neuroses affecting mankind. Although neurasthenia is no longer permitted in the country of its birth, in a great part of the globe it is still the commonest way of describing people with minor mental disorders: for example the Soviet Union, the People's Republic of China, and some parts of Japan.

The present century began with considerable confusion about the concept of neurosis. The last of the old-style neuroses, in the sense of a neurosis being related to abnormal reflex activity of the spinal cord, were the trophic neuroses: acrocyanosis, erythromelalgia and angioneurotic oedema. It was already obvious that there was a world of difference between a neurosis like angioneurotic oedema and one whose symptoms were wholly psychological. In an effort to distinguish them, Dubois called the latter the 'psychoneuroses'. Within a few years Freud had described anxiety states, phobias and obsessional illness from the universe of patients previously described as neurasthenics.

As our century has progressed psychiatrists have tended to confine themselves to the relatively small minority of patients with mainly psychological symptoms, while family doctors and physicians have continued to see those whose somatic symptoms cannot be accounted for by known organic disease. However, as psychiatrists are now tending to work more and more in primary care and hospital settings, they are once more encountering the problems that physicians struggled with throughout the 19th century.

As psychiatrists, our ideas of morbidity are determined by the clinical syndromes which GPs see fit to refer to us, so that during our training we become comfortable with a concept of neurosis that consists of patients with entirely psychological symptoms. Our concept of clinical reality is determined partly by the cases we happen to see, and partly by our current taxonomies. Psychiatrists who are unfamiliar with the clinical presentations in this setting have tended to pay more and more attention to the mood alterations which are so common in patients with minor illnesses, and to describe them as depressives.

CULTURAL ASPECTS OF SOMATISATION

Kleinman and Kleinman [5] report a survey of 100 patients in Hunan diagnosed by Chinese psychiatrists as 'neurasthenics'. When the patients were interviewed using the Schedule for affective Disorders and Schizophrenia (the 'SADS-L' interview), no fewer than 87% of these 'neurasthenic' patients fulfilled the criteria for 'major depressive disorder', and a further 6% fulfilled DSM-III criteria for other forms of depressive disorders. However, *all* the patients had sought medical help for somatic symptoms; 90% with headaches; 78% with insomnia; 73% with dizziness and 48% with pains in other parts of the body. Although all the patients experienced dysphoric symptoms only 9% articulated depression as one of their chief complaints. In keeping with the somatic idiom of their distress, the patients believed that their problems were primarily physical despite their knowledge that they were attending a psychiatric clinic. Dysphoric symptoms were thus suppressed: they are too close to the highly *stigmatised* 'mental illness'; and in China, expression of disturbed emotion is regarded as a potential cause of illness.

American psychiatrists, on being told of these findings, assumed that Chinese psychiatrists underdiagnose depressive illness. Anthropologists, on the other hand, assumed that the same distress syndrome was being construed in different ways in each culture: that the illness label used was a social construction of the same clinical reality. In each place, the doctors notice what the label encourages them to notice: weakness, irritability and fatigue to justify a diagnosis of neurasthenia; depression-related symptoms for major depressive disorder.

Kleinman defines somatisation as 'expression of personal and social distress in an idiom of bodily complaints with medical help-seeking'. Those familiar with papers written by psychiatrists from developing countries will know that on this if nothing else, all are agreed: from Bahrain to Borneo, the commonest way that psychiatric illness presents in developing countries is in the form of somatic symptoms [8]. Perhaps because they have stumbled upon it rather late in the day, psychiatrists assume that somatisation requires some special explanation: for example, alexithymia [9], atypical depression [10] or somatising personalities [1]. However, since somatisation goes back several centuries, and is world-wide in its distribution, perhaps it should be regarded as a basic mechanism which the human species has for responding to stress: we develop pains and discomforts in our bodies. Indeed, 'psychologisation' appears to be the more recent phenomenon, and it still seems to be relatively rare in many parts of the world. To the extent that it occurs at all in developing countries, it tends to affect Westernised élites. Perhaps we should ask why people psychologise, instead of looking for explanations for somatisation.

Psychologisation tends to occur in developed countries, with plentiful supplies of doctors; and it is to be expected in egocentric cultures, where there is a narcissistic idealisation of the self. Psychologisation, in this formulation, is characteristic of cultures given to introspection and glorifying the individual; whereas somatisation is seen wherever the individual tends to be submerged in the group. In this formulation, somatisation is the more basic cultural orientation all over the world.

An alternative formulation represents an iconoclastic inversion of this view, which came to us indirectly from a Tibetan monk [11]. In ancient Buddhist scriptures

psychologisation was regarded as the original, and most primitive, response to stress. It was regarded as primitive and maladaptive because it is difficult or impossible to meditate, and psychic pain is beyond the reach of medicines. In this formulation somatisation is regarded as an adaptive achievement of mankind, lessening psychic pain and exchanging it for physical pains for which there have always been treatments.

In the final part of our paper we will consider evidence that somatisation lessens psychic pain: but for the present we can observe that modern treatment for somatisation involves becoming more aware of one's psychological pain, and perhaps taking an anti-depressant. Pharmacology has come a long way since Buddha's time.

SOMATISATION AS A TRANSACTION

In Kleinman's study in Hunan, patients and their doctors had similar world views; both used biological models; both expected medical treatments. This indicates the transactional nature of somatisation: doctors sanction the patient's bodily idiom of distress.

Taxonomists tend to reify somatisation — as in the somatoform disorders of DSM-III — whereas actually it is a process, present in many diverse states, and sometimes apparently present when it is not possible to make any psychiatric diagnosis at all. Doctors often increase the likelihood of somatic symptoms forming the centre-piece of the medical interview by differentially reinforcing somatic symptoms: often because of their own fear of missing organic disease when it is present (i.e. making a 'type I error'). The high technology surrounding medical training encourages doctors to seek reductionist explanations of pain and distress.

As might be expected, some doctors thus produce more somatisation than others. In the Manchester somatisation study [12] Bridges came across the phenomenon which we called 'facultative somatisation'. This referred to patients who had somatised during their interviews with their family doctor, but who readily changed their attribution when interviewed by a sympathetic psychiatrist and were prepared to consider psychological factors as being relevant to their somatic symptoms.

There are of course factors which will encourage somatisation however sympathetically the patient is interviewed. We have mentioned the stigma of psychological disorder, and noted that some cultures stigmatise such disorders more than others. Even within a particular culture the degree of stigmatisation will vary with religious affiliation and between families. Patients tend to offer pains first because, by definition, pain hurts! Furthermore, while pain is often thought to presage serious organic disease, depressive phenomena are not perceived in the same way. The coexistence of depression and anxiety is likely to enhance sensitivity to pain and to lower the complaint threshold. The patient's fears about possible organic causes for his pain will be reinforced by physical investigations unless the negative results are fed back to him in a constructive way. Finally, attributions about the causes of pain will be influenced by depression, loneliness, and other people.

Patients and their doctors often agree in a way that surprises research psychiatrists. In our earlier study on the neurological wards in Manchester [13], 39% of the patients readily told Dr Bridges about their affective illnesses and their related life-problems. Yet few were dissatisfied with the treatment that they had received

from the neurologists, whom they appeared to regard as rather over-worked biological motor-mechanics: ‘. . . I was worried that my pain might have been caused by a brain tumour, and he reassured me that I hadn’t got one . . .’ — the neurologist had used the CAT scanner as an anxiolytic, and the patient was satisfied!

In England, and perhaps elsewhere as well, somatising patients had tended to remain with primary care physicians, although they make frequent forays into medical and surgical clinics in order to exclude organic disease. When this is not discovered they are typically referred back to their family doctors. Of course, the more they are investigated, the more convinced they become that there is some hidden physical illness present. However, they usually report mood disorders as well. Enter a research psychiatrist. It is these patients who record symptoms which lead him to make diagnoses of anxiety state, depressive illness, and mixed anxiety-depression. The research psychiatrist does this by ignoring the somatic symptoms; just as the other doctors have tended to handle them by confining their attention to the somatic symptoms. Each side has paid a heavy price for the divorce between psychiatry and general medicine.

THE MANCHESTER SOMATISATION STUDY

In recent years an extensive literature has grown up about somatisation, although we should perhaps emphasise that most authors are referring to chronic somatisers — as in the DSM-III ‘somatoform disorders’ — whereas our research is concerned with acute and subacute somatisation [4]. Thus, assertions which may be true of somatisers found in pain clinics or after referral through multiple general hospital clinics may not apply to those who are, as it were, at the beginning of their career as somatisers.

We carried out our survey in 15 family practices in the Greater Manchester area. We have described the procedure in greater detail elsewhere [12, 14]; but in essence we asked the family doctors to identify consecutive patients who were experiencing new illnesses, and selected cases with psychiatric illnesses diagnosable according to DSM-III criteria using a standard two stage case finding procedure. We found that although about one-third of our patients with new episodes of illness were diagnosable according to the DSM III system as having psychiatric disorders, only 5% had ‘entirely psychiatric’ illnesses, in the sense of being free of co-existing physical disorders. However, about 19% of patients with new illnesses fulfilled our research criteria for ‘somatisation’, in that they satisfied four operational criteria at psychiatric interview. These were first, that the patients were consulting for somatic symptoms; second, that the patient attributed these symptoms to physical disease, or took the view that the physical symptoms constituted the disorder; third, that a DSM-III psychiatric disorder was present; and fourth, that in the view of the research psychiatrist treatment of the affective disorder would reduce or eliminate the physical symptoms.

Family doctors were very much more likely to detect psychiatric disorders where the patient was ‘psychologising’ than when they fulfilled these criteria for ‘somatisation’: 95% of the former group were rated as ‘psychiatric cases’ by their doctor, against only 48% of the latter.

We collected groups of psychologists and somatisers and carried out extensive interviews including the administration of numerous personality and attitude scales. Whatever might be true of chronic somatisers seen in pain clinics in the United States, the subacute somatisers seen in primary care turned out to be remarkably similar to the psychologists seen in the same setting. Thus, we are unable to confirm that somatisers are low class, stupid, or sexually immature when compared with psychologists seen in the same settings. They describe their parents in a less abnormal way on the Parental Bonding Instrument, and their memories of childhood illnesses are the same. They have spouses who are neither more nor less sympathetic towards them when ill than psychologists, and their marital adjustment is the same. Psychologists are also just as likely as somatisers to have discussed their symptoms with other members of their family — so it does not look as though the function of somatisation is to assist in the provisional validation of the sick role.

However, our findings are not entirely negative. Somatisers were more likely to have received in-patient care as adults, and they were more likely to have achieved a physical examination from their doctors. On the differentiation of emotions test, somatisers had higher correlations between cognitive and somatic items than psychologists; for somatisers the correlation was almost +0.70 between scales, to be compared with only +0.36 for the psychologists.

Somatisers were more hostile to mental illness on various attitude scales, and asked to imagine themselves with symptoms of either depression, neurasthenia or panic declared that they would be less likely to consult a doctor, or, if they were seeing a doctor, would be less likely to mention such symptoms to him.

Psychologists, on the other hand, were significantly more likely to be depressed than somatisers, and had higher scores on both the GHQ-28 and the Present State Examination. They were just as anxious as somatisers, but tended to be more depressed. They were also significantly more dissatisfied with their social circumstances and significantly more stressed in their present lives. They reported more dependence on their relatives than somatisers did.

SOMATISATION AS BLAME AVOIDANCE

We are now in a position to see the adaptive advantage of somatisation, painful though it is in other ways: it is a great way for not seeing oneself as mentally ill, and not seeing oneself as responsible for the life predicament that one happens to be in. A man need not ask himself whether he has been a good husband, has done as well as might have done at his work, or made a mess of bringing up his children: it is enough that he is in pain. If anyone is to be responsible for his predicament, it is surely his doctor, who has either not made the correct physical diagnosis, or has at any rate not stopped his pain.

It is this blame-avoiding function of somatisation that seems to us to be its key feature, and perhaps explains why patients do not report such great levels of depression. They are in life predicaments just as bad as the psychologists, so we should not be surprised that they are hurting; and because they are hurting they are eligible for the secondary gains of the sick-role.

Blame can then be handled in one of three ways: projected outwards — as in the case of a Yoruba villager who attributes her abdominal pain to witchcraft;

internalised — as in the hypochondriacal countryman convinced that his abdominal pain is due to cancer; or a combination of both mechanisms — as in the patient who is convinced that the pain is due to some disease or other, but as the doctor hasn't found out what it was, it's all his fault.

If the advantage of somatisation is that patients are spared some of the self-blame and thus the intensity of depressive symptoms, a compensating disadvantage may be that somatised forms of psychiatric disorder tend to last longer than purely psychological disorders. It is possible that the failure to face up to one's problems by changing attribution is responsible for the greater tendency of these disorders to become chronic [15].

SUMMARY

We have tried to describe somatisation, not as a disease, but as a common and important human mechanism involving both doctor and patient. It is the single most common reason why psychiatric illness goes undetected in general medical settings, and it often occurs in conjunction with physical disease processes.

The association with dysphoric affect has been recognised at least since George Cheyne 250 years ago, and the reason for this is that both anxiety and depression serve to amplify pains. However, it seems likely that somatisation can occur in the absence of dysphoria.

Once it has been established, it is easy to see how it continues: it secures advantages from spouse, family and employers; and it tends to be encouraged by doctors — who differentially reward somatic symptoms.

But why does it occur in the first place? We have argued that it seems to have three functions:

First, it allows people who are unsympathetic to psychological illness, or who live in cultures where mental illness is stigmatised, to nonetheless occupy the sick-role while psychologically unwell.

Second, it is blame-avoiding: instead of being responsible for the mayhem, one is cast in the role of the suffering victim.

Finally, by reducing blame, it appears to save patients from being as depressed as they might otherwise have been.

IMPLICATIONS

There are three: involving taxonomy, further research, and training needs.

Taxonomy is probably the least important of these, since we must expect several years with ICD-10 before we can reasonably plan for ICD-11.

However, there are interesting precedents being set in the classificatory schemes being suggested for anxiety disorders by DSM-III-R. Here, instead of having to say whether a given patient is a case of panic, phobic disorder or agoraphobia, we are now allowed to say either 'uncomplicated panic disorder'; or 'panic disorder with limited phobic avoidance'; or 'panic disorder with agoraphobia'. In other words steak; steak and chips; or steak, chips and peas. In similar mode it is perhaps not too much to hope for a taxonomy that allowed uncomplicated depressive illness, or depressive illness with somatisation. Whether or not it was really worth doing

this would depend on research which has not yet been done, which deals with the optimal management of somatised forms of depression. To what extent is it necessary to alter the patient's attribution of their problem if they are to be helped, and to what extent is a good outcome mainly dependent on pharmacological treatment of their depressive illness? A straightforward 2 x 2 factorial design would be of very great interest.

We strongly suspect that such a study would show that it is necessary to change the patient's attribution: if this could be shown it would lead me to our final implication: how should family physicians be taught to alter patients' attributions? Interestingly, if the research concerned with optimal management is ever to take place, this educational research is actually the next step; until a group of family doctors can be taught to alter attributions in a reproducible way, the necessary research into management cannot really be done. If psychiatrists are to make a contribution to the work of family doctors in treating mental illness they must be prepared to acquaint themselves with what the problems actually are; and this will certainly involve them in assisting with the management of somatised forms of affective illness.

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