

## Correspondence

**Contents:** Somatisation in general practice/Declining incidence of schizophrenia/Maintenance therapy for schizophreniform disorder/Onset of the antidepressant effect of ECT/Psychosis and cannabis/Nasal decongestant and psychiatric disturbance.

### Somatisation in general practice

SIR: In describing the natural history of 'acute' somatisation in general practice, Craig *et al* (*BJP*, November 1993, 163, 579–588) address the least well understood aspect of this phenomenon. However, I would like to raise two concerns about the method of this study. Firstly, despite their concern to improve upon the least defensible criterion used by Bridges & Goldberg (1985), the authors omitted to describe their own method for assessing subjects' symptom attributions, arguably the *sine qua non* of somatisation (Lloyd, 1986). Secondly, although ostensibly a longitudinal study of the course of somatisation, it would appear that outcome data were ascertained retrospectively, from interviews two years after the index consultation. If so, such data would have been highly susceptible to both subject and observer bias.

While those working in secondary care prefer to define somatisation as "persistent consultation for medically unexplained somatic symptoms" (Creed *et al*, 1992), primary-care researchers conceptualise somatisation as "the somatic presentation of psychiatric disorder" (Bridges & Goldberg, 1985). This difference reflects the diversity of clinical phenomena subsumed within a single term (Kirmayer & Robbins, 1991), and the complex relationship between physical and psychiatric morbidity. As Craig *et al* point out, there is little evidence that the forms of somatisation seen in primary and secondary care are indeed part of a single spectrum. Nevertheless, these authors share the commonly held assumption that 'functional' somatic complaints can be distinguished from those which reflect 'genuine' organic pathology. Despite independently rating the likely 'organicity' of subjects' somatic symptoms, it was disappointing that (once again) no attempt was made to validate such judgements prospectively.

By concentrating on "one rather narrow view" of somatisation, Craig *et al* may have lost sight of the most important issue in the primary care of psychiatric disorder. The presentation of somatic

symptoms by the majority of patients with psychiatric morbidity results in low rates of psychiatric case detection by general practitioners, and contributes to prolonged morbidity, inappropriate (and costly) use of health service resources, and iatrogenic illness (Murphy, 1989). Craig *et al* found that only 44 out of 1220 consecutive attenders (3.6%) met their criteria for incident cases of somatisation. The practical implications of their findings are unclear, particularly since ten times this proportion (34.6%) were identified as probable cases of psychiatric morbidity using the General Health Questionnaire, of whom as many as two-thirds were likely to have presented only somatic symptoms to their doctor.

BRIDGES, K. W. & GOLDBERG, D. P. (1985) Somatic presentation of DSM-III psychiatric disorder in primary care. *Journal of Psychosomatic Research*, 29, 563–569.

CREED, F., MAYOU, R. & HOPKINS, A. (eds) (1992) *Medical Symptoms not Explained by Organic Disease*. London: Royal College of Psychiatrists and Royal College of Physicians of London.

KIRMAYER, L. J. & ROBBINS, J. M. (1991) Three forms of somatisation in primary care. *Journal of Nervous and Mental Disorders*, 179, 647–655.

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MURPHY, M. (1989) Somatisation: embodying the problem. *British Medical Journal*, 298, 1331–1332.

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SIR: With respect, I suggest that further pursuit of the concept of 'somatisation' is not worth the money or effort, because, like earlier obfuscatory labels, it offers no help to doctors who have the task of treating sick and suffering people.

Lipsitt (1973) described hypochondriasis as "a diagnosis in search of a disease", and added "our persistence in trying to retain such terms may lead not only to further inappropriate application . . . but worse to a stifling of investigations into complex conditions". However, by that time Parsons (1951) had added another term – in "the sick role", soon to be quoted by aspiring *cognoscenti* at scientific meetings as the latest in advanced thinking. "Illness behaviour" came next, coined by Mechanic in 1968, and "abnormal illness behaviour" followed (Pilowsky, 1969). Mayou's warning in 1986 was therefore wholly justified when

he said, "it is essential to realise that illness behaviour does not refer to a theory but is a portmanteau term".

In 1982 Wolf, while discussing the problems and pitfalls in behavioural approaches, made a pertinent observation. He said, "The epidemiological method, with its reliance on standard criteria and relatively large numbers, tends to blur characteristics of individuals that may be significant." He concluded that bio-behavioural studies had been "restricted to a Procrustean mould," unsuitable to people functioning in their social milieu.

If someone can show me that the concept of 'somatisation', like the other labels before it, has helped a single patient get better, I will gladly retract my criticism.

LIPSITT, D. R. (1973) Psychodynamic considerations of hypochondriasis. *Psychotherapy and Psychosomatics*, 23, 132-141.

MAYOU, R. (1986) Illness behaviour in cardiac patients. In *Proceedings of the 15th European Conference on Psychosomatic Research* (eds J. R. Lacey & D. A. Sturgeon), pp. 111-114. London: Libby.

MECHANIC, D. (1968) *Medical Sociology*. New York: Free Press.

PARSONS, T. (1951) *The Social System*. New York: Free Press.

PILOWSKY, I. (1969) Abnormal illness behaviour. *British Journal of Medical Psychology*, 42, 347-351.

WOLF, S. (1982) Psychological forces and neural mechanisms in disease: defining the question and collecting evidence. *Johns Hopkins Medical Journal*, 150, 95-100.

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**AUTHOR'S REPLY:** Weich and Paulley raise a number of issues that require a response.

First, it is said that we omitted to describe our method for assessing subjects' symptom attributions. This is not so, but perhaps the method could have been made more clear. In terms of the *subjects'* attributions, we followed the approach adopted by Bridges & Goldberg (1985) – somatising patients all attributed their somatic symptoms to physical rather than psychiatric disease, and had consulted their general practitioner with somatic complaints. We avoided any attempt to impose our own attributional theories. There were two reasons for this decision. First, we wanted to test the validity of the common clinical belief that the somatic symptoms of somatisers are attributable to psychiatric disorder. Second, any attributional judgements on our part would inevitably be influenced by our knowledge of independent variables. With these considerations in mind, we chose the potentially less biased approach of employing an independent physician to identify 'functional' disorder where there was no definite explanation for the symptoms in terms of known organic disease. We found a close association between the onset and course of psychiatric and

somatic symptoms only among subjects with these functional disorders, which is what one would hope to observe if the notion of somatisation is valid (p. 583 ff.). As we point out, this approach runs the risk of errors in classification, as some 'organic' explanations will be missed where laboratory tests are incomplete or presentations of symptoms are atypical. However, such errors will work against our main hypothesis (that there is an association between somatic and psychological symptoms in somatisers) and for an aetiological enquiry are preferable to classifications derived by researchers who are intimately familiar with the independent variables and might consciously or unconsciously bend the classification to fit the pattern of these data.

Secondly, Weich is correct in drawing attention to the risks of reconstructing events over a two-year retrospective period. In order to attempt to minimise these risks, we collected detailed information concerning the index disorder and associated consultation patterns at regular intervals throughout the study, and used this precisely dated information as anchor points in our retrospective interview. Again, there may well be errors of classification, but I do not believe these data would be "highly susceptible" to subject and observer bias. Observer bias was minimised by ensuring that research interviewers were blind to diagnostic group until all the data had been rated, and there was a close correspondence between subjects' reports of recovery and the clinical records.

Thirdly, both Weich and Paulley take us to task for the dubious applicability of this research. Weich also implies that our definition of somatisation is rather trivial given the wider problem of somatic presentations of psychiatric disorder in general practice. Weich muddles an incidence estimate for one manifestation of psychiatric disorder (somatisation) with a prevalence estimate for all psychiatric disorder that necessarily includes both incident and chronic cases. A better 'feel' for the magnitude of the problem of somatisation in general practice as reflected by this sample is the observation that of the 109 cases of recent-onset psychiatric disorder (as confirmed by Present State Examination), 91 presented with somatic symptoms and, of these, over half were somatisers. Furthermore, it was the somatisers who were at greater risk of chronicity of their physical symptoms, and thus of inappropriate diagnosis and excessive use of services. 'Mixed' cases, in contrast, recovered from their physical symptoms relatively rapidly, and consumed no more resources than their purely physically ill counterparts. None of this diminishes in any way the importance of being sensitive to the psychosocial treatment needs of our patients, whether or not these are 'somatised'.