

Table 2 Management of patients with functional neurological symptoms

Management stage	Suggested approach	References and further reading
Initial assessment	Demonstrate that you believe and are interested in symptom and severity	Craig, ⁴⁰ Williams and House, ⁴⁷
	Elicit history of other symptoms, previous contacts with health service	Page and Wessely, ⁶⁷
	Find out what patient has been told about his symptom by other doctors	Creed and Guthrie, ⁷¹
	Elicit patient's own beliefs about the symptom	Fink <i>et al</i> , ¹⁰⁶
	Screen for significant psychiatric disorder (especially depression and anxiety)	Morriss <i>et al</i> ¹⁰⁸
	Show interest in impact of symptoms on patients' life	
	Ask about life events	
	Obtain history from partner/relative/friend if possible	
	Review previous clinical records if possible	
	Arrange appropriate tests (if necessary)	
Communication of diagnosis	Admit uncertainty if investigations incomplete/inconclusive	Page and Wessely, ⁶⁷
	Clarify with the patient how structural disease has been excluded	Jackson and Kroenke, ⁷²
	(taking account of patient's specific health concerns)	Morriss <i>et al</i> , ¹⁰⁸
	Reframe symptoms ("I can see that since you lost your wife...")	Coia and Morley ¹¹³
	Give a positive explanation of the symptom	
	Convey the potential for substantial recovery	
	Be honest and direct with patients (copying clinic letters is a good way of reiterating important issues)	
Acute symptomatic therapy	Discuss potential acute/remote stressors	Richardson and Engel ¹¹⁴
	Suggest that symptoms are likely to improve	
	Encourage activity rather than rest/consider physiotherapy	
Psychiatric medication	Ask the patient's view (will they take the tablets?)	O'Malley <i>et al</i> , ⁹⁸ Soloff, ⁹⁹
	Consider antidepressants even in the absence of overt depressive/anxiety symptoms	Stone <i>et al</i> ¹⁰²

Referral for psychological/psychiatric assessment	<p>Explain length of treatment, possibly delayed effectiveness,</p> <p>lack of addictive potential</p> <p>Point out that reducing stress and learning ways of coping with symptoms are useful to all patients regardless of the nature of their symptoms</p> <p>Consider joint appointment</p> <p>Be optimistic but avoid raising expectations to levels which are likely to disappoint</p>	House ¹¹⁵
Psychological management options	<p>Consider patient held treatment plan, or patient held records</p> <p>Identify goals for treatment</p> <p>Work on identifying predisposing, precipitating, and perpetuating factors</p> <p>Look at potentially problematic patterns in interpersonal relationships</p> <p>Identify ongoing life stressors</p> <p>Identify and address patterns reinforcing abnormal behaviour</p> <p>Reframe and reattribute the links between psychological factors and symptoms</p> <p>Consider the use of specific psychotherapeutic techniques by those with appropriate training (for example, cognitive behavioural and analytical, interpersonal, behavioural psychotherapy)</p> <p>Use appropriate evidence based psychological interventions to treat anxiety and depression if present</p> <p>Discuss relapse prevention</p> <p>Consider goodbye letter to patients on completion of work</p> <p>reinforcing issues discussed and recording progress made.</p>	<p>Goldberg <i>et al</i>,⁷³ Guthrie,⁸⁵</p> <p>Bleichhardt <i>et al</i>,⁸⁷</p> <p>Sharpe <i>et al</i>,⁹⁶</p> <p>Roth and Fonagy,⁹⁷</p> <p>Fink <i>et al</i>,¹⁰⁶</p> <p>Morriss <i>et al</i>¹⁰⁸</p>

Behavioural interventions in the rehabilitation of acute v. chronic non-organic (conversion/factitious) motor disorders

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Background Repeated case series have documented the effectiveness of multidisciplinary in-patient behavioural treatment for conversion disorders. However, in the absence of controlled research, treatment success could be attributed to providing patients with a face-saving opportunity to get better.

Aims The present study contrasts two behavioural treatments to elucidate the factors underlying successful in-patient rehabilitation of this population.

Method Thirty-nine patients underwent a standard behavioural programme. Using a crossover design, patients who did not improve underwent a strategic-behavioural treatment in which they and their families were told that full recovery constituted proof of an organic aetiology whereas failure to recover was definitive proof of a psychiatric aetiology.

Results Chart review indicated that the standard behavioural treatment was effective for 8/9 'acute' patients but only for 1/28 'chronic' patients. Of the 21 patients with chronic motor disorder who then underwent the strategic-behavioural intervention, 13 were symptom-free at discharge.

Conclusions The strategic intervention was superior to standard behavioural treatment for patients with chronic motor disorder. Treatment components previously deemed critical for the effectiveness of behavioural treatment may be unnecessary.

Declaration of interest None.

Although two case series have documented the effectiveness of in-patient behavioural treatment for non-organic motor disorders (Trieschmann *et al*, 1970; Speed & Moonney, 1996) we (Shapiro & Teasell, 1997a) reported that this treatment was ineffective with patients with chronic motor disorders. However, the majority of these patients who had not progressed were discharged symptom-free after undergoing a strategic-behavioural intervention in which they and their families were told that although full recovery would constitute proof of a physical aetiology, failure to recover completely would constitute conclusive evidence of a psychiatric aetiology. Our results not only questioned the efficacy of behavioural treatment in patients with chronic conversion disorder but also suggested that the treatment components deemed critical from a behavioural perspective may be unnecessary. In the present study, the patients presented in our previous series (Shapiro & Teasell, 1997a) are combined with 15 additional cases to examine more closely the relative efficacy of standard behavioural *v.* strategic-behavioural treatment with acute *v.* chronic motor disorders and to elucidate further factors underlying treatment success.

METHOD

Participants

The participants were 39 patients admitted consecutively to the rehabilitation ward of the London Health Sciences Centre, University Campus, between 1 September 1987 and 31 October 1998. These patients were from an original sample of 130 patients referred to the Physical Medicine and Rehabilitation Unit by tertiary care specialists (generally neurologists) and examined by the second author (R.W.T.) as out-patients or during their in-patient admission to the Neurology Unit of this tertiary care facility. Although all 130

patients were referred with a (provisional) diagnosis of conversion disorder, 75 patients were subsequently diagnosed as having a chronic pain disorder with secondary, unexplained motor symptoms and were not deemed suitable for our programme (Shapiro & Teasell, 1997b; Teasell & Shapiro, 1997). Fifty-five patients were told that in-patient admission to our rehabilitation unit could 'get them functioning and walking normally'. Fourteen patients declined treatment.

Forty-one patients were admitted for an initial week of evaluation to rule out an organic basis for their symptoms. If not already performed, this evaluation included central nervous system imaging with magnetic resonance imaging and/or computed tomography, extensive blood testing and, in some cases, electromyography/visual and somatosensory evoked potential testing. The vast majority of patients had been assessed by more than one neurologist and many had been evaluated by multiple specialists. Two patients were subsequently diagnosed with organic disorders (transverse myelitis, renal phosphate-wasting osteomalacia). The diagnosis of non-organic motor disorder thus was based upon the presence of paralysis or paresis, atasia basia and/or ataxic-like symptoms with no apparent neurological or other organic disorder. Astasia basia is characterised by an unsteady gait (ataxia) with a bizarre lack of coordination, even though all leg movements can be performed normally while sitting or lying down. To confirm the diagnosis, during the evaluation period the remaining 39 patients were closely monitored by rehabilitation staff who observed and documented dramatic inconsistencies in symptom presentation between that demonstrated during formal examination *v.* informal (unobtrusive) observation when patients were engaged in distracting activities. We avoid using the diagnoses of conversion or factitious disorder. The criterion for distinguishing between them is whether patients are aware of intentionally producing their symptoms (i.e. whether symptoms are under conscious control). An observer must infer that which is conscious *v.* unconscious, an inference that is impossible to make definitively. This diagnostic issue is addressed more fully in the discussion section below.

Patient demographics and presenting symptoms are summarised in Table 1. Two groups were readily discernible on the basis of the length of time the symptoms

Table 1 Patient characteristics

	Acute motor disorder (n=9)	Chronic motor disorder (n=30)
Median age of symptom onset (years)	39	42
Male	11% (1)	20% (6)
Paresis	100% (9)	63% (19)
Paralysis	11% (1)	7% (2)
Astasia basia	0% (0)	40% (12)
Pain	33% (3)	70% (21)
Bladder/bowel dysfunction	0% (0)	27% (8)
Other secondary symptoms	11% (1)	60% (18)
More than one non-organic symptom	11% (1)	77% (23)

were present. Nine patients had acute motor disorder (onset within 2 months of admission) and 30 patients had chronic motor disorder (a symptom duration of more than 6 months). The primary symptoms were paresis and/or paralysis of one or more limbs and astasia basia. These symptoms were considered 'primary' because they were readily apparent to an observer and therefore critical for conferring the status of being disabled. The elimination of these observable symptoms was the focus of treatment. Patients frequently presented with secondary symptoms without a discernible organic basis. These included leg shaking, tremors, reports of pain, speech abnormalities and difficulty with bladder or bowel function. None of the patients reporting pain considered pain to be the primary reason for their disability. Inspection of Table 1 reveals that patients with chronic motor disorder more often presented with multiple symptoms.

Procedure

Prior to admission, patients were told that, regardless of the origin of their disorder, full recovery was possible with intensive in-patient rehabilitation. Among the 39 consecutive patients who agreed to treatment, 37 underwent standard behavioural treatment and two patients underwent the strategic-behavioural programme from the outset. If after 4 weeks of treatment there was no progress, the strategic protocol was implemented. Progress was defined as clearly observable improvements in gait and posture during physiotherapy. The first 20 patients were provided with individual counselling to 'help them through a difficult rehabilitation process'. As patients began to improve, they were encouraged to discuss concerns related to discharge.

The vast majority consistently maintained that they did not have emotional concerns (i.e. they failed to engage in a meaningful therapeutic process). Accordingly, individual counselling was rarely provided for subsequent patients. In anticipation of discharge, all patients were encouraged to consider follow-up counselling to help them adjust to the transition to a non-disabled status. Almost all declined. The few patients who initially agreed to follow-up counselling never attended the scheduled appointments.

Standard behavioural treatment

Patients were told that, regardless of the origin of their disorder, current symptoms were maintained by abnormal muscle patterns that had developed over time. They were told that therapies were designed to help them re-learn proper muscle functioning. Any reference to psychiatric terminology was avoided. Daily physiotherapy consisted of progressive gait and posture re-training along with flexibility and strength exercises. Therapies were structured in a manner similar to recovery from a neurological disorder. Secondary symptoms were interpreted as due to the same 'general muscle dysfunction'. Patients were assured that as they began to use their muscles in a more optimal fashion these related difficulties would normalise. Staff were instructed to praise successful performance and to encourage patients to try again if they failed to achieve a desired goal in therapy. In contrast to previously published case series of behavioural treatment of non-organic gait disorders (Trieschmann *et al*, 1970; Speed & Mooney, 1996), there was no attempt to eliminate all opportunities for symptoms to be reinforced by confining patients to wheelchairs or immobilising their affected limbs. Indeed, many patients were already wheelchair-dependent.

However, staff were instructed to respond to symptomatic behaviour in a matter-of-fact manner.

Strategic-behavioural treatment

The core element of this intervention involved telling patients that, although full recovery constituted proof of a physical aetiology, failure to recover constituted conclusive evidence of a psychiatric aetiology. This was communicated by the attending physician (R.W.T.) based upon a detailed script developed by the first author (A.P.S.) that also included instructions for all team members on implementing their part of the programme. Patients were told that although staff were pleased with their progress (which was minimal) they should be improving more quickly. It was explained that slower than expected progress could be due to only one of two factors: their disorder was not physical but a psychiatric problem called a conversion disorder; or there was an aspect of their disorder that required a modification in the treatment. It was explained that, once this necessary modification was made, progress would be rapid and recovery complete. However, if it was a conversion disorder they would not recover fully because of an 'unconscious need to remain disabled'. Hence they would: continue to make improvements in some areas but still experience significant problems and disability; improve in some or all areas but develop new problems for which there was no organic basis; fail to improve at all; make a complete recovery in hospital, only to develop the same or new problems some time after discharge from hospital; and/or request discharge before they recovered fully. It was explained that, if it turned out to be a conversion disorder, full recovery was possible with long-term psychiatric treatment.

The usual 'medical' explanation for lack of progress was that the 'muscle patterning' problem was causing excessive fatigue. This allowed for the use of 'deep rest' when patients failed to meet daily therapy goals. Deep rest involved immediately returning patients to their room to lay on their beds with their eyes closed and with no stimulation of any kind (television, telephone, reading or visitors) until their next scheduled therapy session (i.e. deep rest constituted an operant intervention whereby we withdrew all reinforcement for failure to progress). Observation of the first

three patients (Teasell & Shapiro, 1994) indicated that deep rest was unnecessary and therefore was removed from the strategic-behavioural protocol for the majority of patients. Instead a minor and inconsequential change in physiotherapy (e.g. changing the sequence of exercises) was made to address the 'muscle patterning' difficulty.

In a subsequent family session with the patient present, the exact same strategic 'script' was again presented. This family conference was scheduled just prior to discharge if symptoms resolved in response to the strategic intervention, or earlier if there was not sufficient improvement. In the latter case it was intended to overcome resistance. In both cases it was designed to prevent relapse. Thus, during this family conference the attending physician emphasised that even when patients recover fully in hospital there remains a possibility, albeit small, that the problem was always a conversion disorder. Accordingly, one only knows for certain if, after discharge, patients remain symptom free and do not develop new problems. If old symptoms should reappear or new non-organic symptoms develop, patients and their families are advised to seek psychiatric treatment.

When patient progress plateaued, the treating physician (R.W.T.) communicated his growing suspicion that the problem was psychiatric. Although usually effective, this intervention often needed to be repeated several times over the course of treatment. When this was not effective, presentation of the strategic script in a family conference often resulted in resumption of progress. Deep rest was instituted with several patients who failed to respond to both of these interventions and there was no impact (i.e. patients who did not respond to the strategic intervention ultimately remained treatment failures, whether or not this operant component was instituted).

Patients often maintained some minor sign of residual difficulty upon discharge in an apparent effort to communicate to others that despite their dramatic improvement they had a legitimate physical problem. For instance, a patient admitted who was wheelchair-dependent might be discharged with normal gait but insist on a one-point cane for walking distances. These subtle symptoms were allowed on a temporary basis. Thus, in the family conference patients were told that if the problem was physical, as their muscles continued to normalise, these minor

residual symptoms would completely disappear within 3 months. Failure to do so would constitute proof of a psychiatric aetiology.

Outcome measures

Outcomes were defined with respect to outward signs of disability. The usual index of improvement in rehabilitation – increasing functional independence – was not considered an appropriate outcome because patients readily improved in their ability to perform tasks independently while still exhibiting little or no change in the abnormal way they used their limbs. It was this apparent effort of patients to maintain control over the nature and extent of their improvement that prompted the development of the strategic protocol (Teasell & Shapiro, 1994). Outcomes were established from chart review by both authors independently and defined as follows.

- (a) *Complete/near complete improvement.* Patients displayed no overt signs of abnormal movement or posture suggestive of disability, nor did they complain of any symptoms that would render them disabled from the perspective of an outside observer. This included complete resolution of secondary symptoms. The only exceptions were very subtle residual signs such as the use of a one-point cane, but with normal posture and gait.
- (b) *Significant improvement.* Overt symptoms of disability were significantly reduced relative to admission status. However, an outside observer would still view the patient as disabled. Only one patient met this criterion – the first patient for whom we developed the strategic-behavioural protocol. She was admitted with quadriplegia and requested discharge once she progressed to fully independent paraplegia (Teasell & Shapiro, 1994). The strategic protocol was altered with subsequent patients so that failure to achieve *complete* resolution of symptoms was deemed proof of a psychiatric aetiology.
- (c) *Minimal/no improvement.* Outward signs of disability were not significantly reduced relative to admission.

RESULTS

Inspection of Table 2 reveals that the effectiveness of the standard behavioural intervention was a function of symptom duration. Eight out of nine patients with

acute motor disorder completely recovered. In contrast, 27/28 patients with chronic motor disorder were deemed treatment failures. The strategic protocol was then implemented for 22 (1 acute, 21 chronic) of these treatment failures, with six patients with chronic motor disorder discharged for the following reasons: one was admitted before the strategic approach had been developed; two spoke no English, which would have made the strategic protocol difficult to implement; and three were not deemed appropriate because we suspected a schizophrenic disorder in two patients and the third expressed suicidal ideation. As indicated in Table 3, among the 21 patients with chronic motor disorder who did not improve with standard behavioural treatment and then underwent the strategic intervention, 13 were discharged completely or almost completely symptom-free. Overall, the strategic-behavioural protocol was effective in 17/24 (71%) patients and was clearly superior to the standard behavioural approach.

DISCUSSION

This paper documents, via repeated case study, the potential utility of a strategic-behavioural approach in the rehabilitation of chronic non-organic motor disorders. It would be difficult to attribute the observed improvement in patients with chronic motor disorders to anything other than this intervention. The majority of these patients presented with symptoms of more than 2 years' duration and had undergone other interventions prior to admission without benefit. Moreover, the use of a crossover design in which 14 (13 chronic, 1 acute) of 21 patients who first failed the standard behavioural intervention were discharged completely, or almost, symptom-free following the strategic protocol lends further credence to the impact of this intervention. Other than the presentation of the strategic script, there was little difference between the standard behavioural and strategic protocols.

Behavioural treatment of conversion disorder is based upon the premise that these disorders represent maladaptive responses to stress that are maintained by positive support from others and successful avoidance, via disability, of stressful life situations. Although the standard behavioural programme was ineffective for patients with chronic motor disorder it was successful for patients with acute motor

Table 2 Standard behavioural treatment

	Complete/near-complete improvement	Minimal/no improvement
Chronic motor disorder (<i>n</i> =28)	1	27
Acute motor disorder (<i>n</i> =9)	8	1

disorder. This is despite the fact that there was no attempt to withdraw completely the reinforcement for disabled behaviour by confining patients to wheelchairs or otherwise immobilising the affected extremity, as was done in previous successful case series (Trieschmann *et al*, 1970; Speed & Mooney, 1996). The only attempt to withdraw systematically all reinforcement for failure to progress – deep rest – had no impact. Both behavioural and strategic-behavioural treatments failed to address systematically the hypothesised skill deficits in response to stress. Five of the nine patients with acute motor disorder did not receive psychotherapy but still recovered with the behavioural protocol. Among the patients with chronic and acute motor disorder who received counselling, few were observed to engage in the therapy process. Thus, the costly treatment components deemed critical for the success of behavioural interventions – complete control of environmental contingencies (requiring in-patient treatment) and individual psychotherapy – may be unnecessary. Thus, for patients with non-chronic motor disorder the conclusion by Bird (1979), based on a review of behavioural treatment of conversion, that treatment success could be due to having provided patients with ‘an adequately impressive excuse to get better’ may be equally applicable today. A randomised controlled trial that systematically varies the treatment components would be required to confirm this.

Methodological limitations

This paper is best viewed as a series of repeated case studies, with the many

associated methodological limitations. Arguably, the most significant flaw is the absence of systematic follow-up, thus precluding any conclusions about long-term outcome. The majority of patients came from a significant distance and were lost to follow-up. Based on an initial attempt to collect follow-up data on the first 20 patients, a conservative estimate of relapse among patients with chronic motor disorder would be 30–40%. The simulation of disability represents an extreme solution to life problems and most likely reflects the existence of certain variables – whether conceptualised as personality dimensions, skill deficits, unconscious conflict or family systems factors – that render patients at risk for retreating back into the sick role. In some patients, these predisposing variables are likely to exert relatively greater influence. Patients with the most severe form of factitious disorder, Munchausen’s syndrome, most likely fall into this category. Elsewhere (Shapiro & Teasell, 1997a) we suggest a more systematic and potentially more effective strategic intervention to require patients to continue to prove the legitimacy of their original disorder by remaining symptom-free post-discharge.

The absence of long-term follow-up is particularly problematic because patients diagnosed with conversion disorder may demonstrate a fluctuating course and thus it is conceivable that the observed improvement reflected this natural fluctuation. This is especially true of the patients with acute motor disorder, who might have remitted without any intervention. However, among our patients with chronic motor disorder almost all reported a history (typically

greater than 2 years) of non-remitting symptoms that, if anything, became progressively worse over time. Only one patient reported a history of temporary symptom remissions and this patient did not improve following the strategic intervention. It is also possible that among patients with chronic motor disorder the differential effectiveness of the strategic *v.* the behavioural intervention was a function of increased time in hospital. We believe that this is unlikely. For virtually every patient with chronic motor disorder who was eventually discharged symptom-free, introduction of the strategic script was immediately followed by improvements in physiotherapy that were qualitatively different from any improvements observed previously. Thus, patients would exhibit, for the first time, a more normal gait and/or posture whereas previous improvements were in the form of increased ability to accomplish daily tasks without any concomitant observable decrease in the highly abnormal ways they used their bodies.

Another potential methodological concern is that outcome was based upon retrospective chart review by the study authors. However, to be categorised as a treatment success the patients had to demonstrate virtually complete normalisation of what previously was a dramatically abnormal gait and/or posture. They also had to report resolution of any (non-pain-related) pseudo-neurological symptoms that were not readily observable – this included normalisation of bowel and bladder function. These outcomes were clearly documented in patients’ charts, as was the referral to psychiatry when patients continued to exhibit or report symptoms and were deemed treatment failures. This ‘all-or-none’ outcome criterion (notwithstanding the very subtle residual symptoms temporarily allowed) thus left little room for observer or investigator bias. Ultimately, definitive conclusions would be possible only after

Table 3 Strategic-behavioural treatment

	Complete/near-complete improvement	Significant improvement	Minimal/no improvement
Patients with chronic motor disorder who failed behavioural treatment (<i>n</i> =21)	13	1	7
Patients with chronic motor disorder who only received strategic treatment (<i>n</i> =2)	2	–	–
Patients with acute motor disorder who failed behavioural treatment (<i>n</i> =1)	1	–	–

replication of these findings using a randomised controlled design and incorporating rigorous outcome measures (*v.* chart review) and systematic follow-up. Given the inherent difficulties in identifying and recruiting this patient population and the deceptive aspect of the strategic protocol, few clinical researchers are likely to consider such an undertaking.

What factors maintain conversion/factitious disorders?

The strategic protocol was developed and refined based upon observations of the first three patients (Teasell & Shapiro, 1994) who appeared to try to control the nature and extent of their disabled status by maintaining the abnormal way they used their limbs. In designing this protocol, we were guided by strategic therapy's conceptualisations of behavioural disorders and its approach to patient resistance (Watzlawick *et al.*, 1974; Fisch *et al.*, 1982). Strategic therapy is, in turn, an outgrowth of the 1960s–70s family therapy movement and the pioneering work of Milton Erikson, Don Jackson and Jay Hayley (Hayley, 1973, 1976). For strategic therapists, life problems become disorders when, as a problem is not resolved, more of the same ineffective solution is applied. A vicious circle then ensues, with the problem escalating in size and nature to a point where it may have little apparent similarity to the original difficulty. We can speculate how this process might unfold and account for the differential impact of standard behavioural and strategic treatments.

In the case of most acute 'conversion' disorders, one would expect symptoms to resolve quickly as the precipitating stressor is no longer a factor and if patients are told that their symptoms will dissipate and do not require further attention. However, if symptoms persist the patients move into a sub-acute stage where further medical investigations rule out organic conditions and the possibility of a psychiatric aetiology often is raised. Once the spectre of the disorder being 'feigned' is raised, relinquishing the symptoms may be seen as confirming their non-organic nature. The more resources that have been provided to the patient in the form of time, finances and emotional support, the more frightening the anticipated reaction of family who have made these significant sacrifices to accommodate the patient's needs. Thus, although the original stressor may have become

inconsequential, a more immediate problem has emerged with potential for long-term, negative interpersonal consequences. The availability of a face-saving medical intervention at this sub-acute stage (e.g. in-patient behavioural intervention) may allow the patient to get better. However, if no such intervention is offered, and/or the precipitating stressor is still present, a safer response would be to maintain the symptoms. From the patient's perspective, lingering doubts about the veracity of symptoms may be preferred to confirming these suspicions by getting better. An even more effective response may be to develop more symptoms, which would be sure to generate a new round of medical investigations. The more seriously the patient is affected by these new symptoms, the more difficult it would be for others to question their veracity. The finding that patients with acute motor disorders typically presented with only one symptom whereas patients with chronic motor disorders evidenced multiple problems is consistent with this conceptualisation.

As symptoms become more chronic, simply providing 'an excuse to get better' may no longer suffice for the majority of patients. The longer the problem has continued and the more severely affected the patient, the greater the familial resources that have been devoted to the patient. The fear that symptom resolution would cast doubt on the veracity of the disorder would be even more pronounced at this stage. With chronicity comes a history of increasingly sophisticated investigations and an impressive array of specialists, thus requiring an even more impressive rationale and intervention before a patient can be confident that recovery would not cast doubt upon the nature of the disorder. The 'muscle patterning' explanation that accompanied our behavioural intervention likely was not sufficiently impressive for most patients with chronic motor disorders. The longer the symptoms persist, the greater the likelihood that additional issues emerge, making it even harder for patients to relinquish their symptoms. The longer the sick role has enabled patients to bypass many of life's difficulties, the greater the fear of being unable to function adequately outside this sick role. Over time, many relationships dissipate to give way to new relationships formed on the basis of the patients' disability. Recovery carries with it the possibility of significant isolation, especially if it casts doubt on the legitimacy

of the disability. Another factor is the admiration that most, if not all, of our patients received for apparently coping so well with their disability. Thus, patients usually appeared happy and well adjusted and were viewed as pillars of emotional strength. Undoubtedly, this view would be put to the test even if recovery did not cast doubt on the legitimacy of the disorder. With chronicity, most of our patients received long-term disability benefits, the potential loss of which may have been less anxiety-provoking than the prospect of having to function successfully in the workforce. Although the strategic intervention did not eliminate these barriers, it left patients with little choice but to accept the face-saving option of relinquishing their symptoms and confronting the feared consequences of being well.

Symptom duration was the critical factor predicting the outcome of the standard behavioural intervention. Patient characteristics associated with treatment failure in the strategic programme were less clear. However, clinical observation suggested that failure to recover most often occurred in patients whose families either would not believe or were relatively unaffected by the psychiatric diagnosis. For instance, one male patient who appeared unconcerned about a psychiatric diagnosis came from a strongly patriarchal culture in which the entire family readily accepted their duty to care for him. He likely also wielded sufficient influence over his family's understanding of his difficulties that he could successfully dismiss the strategic script presented in the family conference. In contrast, the strategic programme appeared to work especially quickly when one or more family members appeared both angry and sceptical about the nature of the symptoms. Thus, the effectiveness of the strategic protocol appeared to be a function of the degree to which a psychiatric diagnosis carried with it the potential for significant negative responses from family. Although all our patients had family with whom they were living, it may well be the case that patients living alone and without family support would not have benefited from this treatment.

Notwithstanding its methodological limitations, these results, along with the discussion of possible factors maintaining conversion/factitious disorders, suggest a relatively cost-effective approach to early intervention. Patients who do not initially respond to reassurance and a relatively simple medically oriented intervention

(e.g. out-patient physiotherapy) could be presented with an out-patient strategic protocol, thus increasing the likelihood that they will avail themselves of an early, face-saving opportunity to relinquish their symptoms.

Conversion v. factitious disorder

The foregoing discussion of the conditions under which patients will 'relinquish' their disability may leave the impression that patients maintain conscious control over their symptoms. Indeed, our original development of the strategic protocol (Teasell & Shapiro, 1994) was in response to what we viewed as patients' attempts to actively control the rehabilitation process by limiting the nature and extent of their improvement. The distinction between conversion and factitious disorder is that the former is not consciously produced whereas in the latter the patients are intentionally simulating their symptoms. Accordingly, it could be argued that our patients are more appropriately diagnosed as having a factitious disorder. The DSM-IV (American Psychiatric Association, 1994) indicates that the judgement that a symptom is intentionally produced should be based on direct evidence, as happens when a patient with haematuria is found to possess anticoagulants and blood studies are consistent with anticoagulant ingestion. However, for pseudoneurological symptoms such as paralysis or paresis, the DSM-IV provides no criteria to distinguish conscious from unconscious intent and thereby automatically relegates these patients to the conversion category. Accordingly, it was of no surprise that all our patients previously had been diagnosed as having conversion disorders. Ultimately, the observer must infer that which is conscious *v.* unconscious, an inference that is arguably impossible to make definitively, especially in the case of pseudoneurological symptoms. Miller (1988) argued that the criterion of whether patients are consciously aware of producing their symptoms should be dropped from the diagnosis of conversion disorder. This would eliminate the distinction between conversion and factitious disorders. Our experience with the in-patient rehabilitation of non-organic motor disorders also leads us to question the usefulness of this distinction.

Ethical considerations

The strategic protocol may be viewed as deceptive and manipulative. Patients signed

CLINICAL IMPLICATIONS

- In-patient behavioural treatment is ineffective for chronic conversion/factitious disorders.
- Factors maintaining conversion/factitious disorders change over time.
- A strategic-behavioural approach may be effective for chronic conversion/factitious disorders and if introduced at the sub-acute stage it may prevent chronicity.

LIMITATIONS

- Methodological limitations inherent in repeated case series preclude definitive conclusions.
- Failure to distinguish between conversion and factitious disorders is problematic.
- The strategic-behavioural treatment protocol raises significant ethical concerns.

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a standard consent form explaining that the multidisciplinary approach necessitates that patient information is shared both among team members and with patients' families. However, patients could not be informed about the exact nature of the programme. This raises ethical concerns related to informed consent and patient autonomy on the one hand, and undue medical influence and control on the other. Similar concerns were raised in the 1970s with the advent of behaviour therapy, particularly the use of contingency programmes, in institutional settings. Wachtel (1977) considered many of these same ethical issues in his classic text on the integration of psychoanalysis and behaviour therapy and his perspective is equally applicable to strategic therapy interventions. He started with the not uncommon view, at that time, that the use of reinforcement was a form of manipulation and coercion and antithetical to the psychotherapeutic process, which involved self-transcendence, a process of choice that originates within the person (Wheeler, 1973). Wachtel (1977) argued that the term 'manipulation' prejudices the issue and suggested that an alternative perspective is to

view 'the therapist as obligated to make a maximum effort to use his knowledge, skills, and understanding to help the patient free himself of a destructive cycle of events...' (p. 274).

Wachtel also argued that part of the difficulty is that reinforcement is somehow viewed as having an inexorable controlling effect that renders the patient incapable of choice. He points out that, even when being reinforced, patients are always making choices. The strategic protocol also provided patients with choice, the opportunity to save face and confront the fear of getting well or accept the psychiatric nature of the disorder and seek another form of treatment. There may be a concern that by pitting a medical diagnosis against a psychiatric diagnosis we accentuated the stigma associated with the latter, thus making it more difficult for patients to choose psychiatric treatment. However, when discussing the psychiatric diagnosis we were careful to define it as simply another type of illness, based on an 'unconscious' need and requiring treatment that is 'non-medical'. We emphasised that either diagnosis allowed for recovery, given the appropriate

treatment. Wachtel (1977) reminds us that influence is inherent in all human relationships, including psychotherapy. Regardless of orientation, therapists' actions ultimately are designed to enable the patient to act and feel differently. He argues that the therapists' aim 'is not to enable a pristine separation of outer from inner influences, but, rather, to enable the person to be more fully alive to the possibilities life offers' (p. 247).

In considering use of the strategic protocol, practitioners may be less concerned about exerting influence and control but uncomfortable with having to lie to patients and their families in order to do so. In strategic therapy, the manner in which the therapist 'frames' the problem is critical and typically involves lying (Fisch *et al*, 1982). This is particularly true of our intervention, which not only required the attending physician to lie but involved varying levels of deception on the part of the entire treatment team. Although some discomfort may be inevitable when presenting the strategic script, it is necessary to lie well (i.e. it is necessary to be unequivocal) – if a patient recovers completely and

permanently, it is and always was a physical disorder. One must resist the urge not to lie by leaving the question of causation ambiguous. If patients believe that recovery can still be interpreted as reflecting a psychological aetiology, they may view it as a 'no-win' situation and elect the safe alternative of remaining disabled and avoiding the feared consequences of becoming well.

REFERENCES

- American Psychiatric Association (1994)** *Diagnostic and Statistical Manual of Mental Disorders (4th edn)* (DSM-IV). Washington, DC: APA.
- Bird, J. (1979)** The behavioural treatment of hysteria. *British Journal of Psychiatry*, **134**, 129–137.
- Fisch, R., Weakland, J. H. & Segal, L. (1982)** *The Tactics of Change: Doing Therapy Briefly*. San Francisco, CA: Jossey-Bass.
- Hayley, J. (1973)** *Uncommon Therapy: The Psychiatric Techniques of Milton H. Erickson, M.D.* New York: Norton.
- Hayley, J. (1976)** *Problem-Solving Therapy: New Strategies for Effective Family Therapy*. San Francisco, CA: Jossey-Bass.
- Miller, E. (1988)** Defining hysterical symptoms. *Psychological Medicine*, **18**, 275–277.
- Shapiro, A. P. & Teasell, R. W. (1997a)** Strategic-behavioural intervention in the inpatient rehabilitation of non-organic (factitious/conversion) motor disorders. *NeuroRehabilitation*, **8**, 183–192.
- Shapiro, A. P. & Teasell, R. W. (1997b)** Misdiagnosis of chronic pain as hysterical. *NeuroRehabilitation*, **8**, 201–222.
- Speed, J. & Mooney, G. (1996)** Behavioural management of conversion disorder: retrospective study. *Archives of Physical Medicine and Rehabilitation*, **77**, 147–154.
- Teasell, R. W. & Shapiro, A. P. (1994)** Strategic-behavioural intervention in the treatment of chronic nonorganic motor disorders. *American Journal of Physical Medicine and Rehabilitation*, **73**, 44–50.
- Teasell, R. W. & Shapiro, A. P. (1997)** Diagnosis of conversion disorders in a rehabilitation setting. *NeuroRehabilitation*, **8**, 163–174.
- Trieschmann, R. B., Stolov, W. C. & Montgomery, E. D. (1970)** An approach to the treatment of abnormal ambulation resulting from conversion reaction. *Archives of Physical Medicine and Rehabilitation*, **51**, 198–206.
- Wachtel, P. (1977)** *Psychoanalysis and Behavior Therapy: Toward an Integration*. New York: Basic Books.
- Watzlawick, P., Weakland, J. H. & Fisch, R. (1974)** *Change: Principles of Problem Formation and Problem Resolution*. New York: Norton.
- Wheeler, A. (1973)** *How People Change*. New York: Harper and Row.

Cognitive Behavior Therapy for Hypochondriasis

A Randomized Controlled Trial

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HYPPOCHONDRIASIS IS DEFINED as a persistent fear or belief that one has a serious, undiagnosed medical illness. Occurring in as many as 5% of medical outpatients, it is a prevalent, disabling, and chronic condition that has generally been refractory to psychological and pharmacological treatment and costly for the health care system.¹⁻⁶ Until recently, there was no empirically validated treatment for the hypochondriacal patient's somatic symptoms, belief in the presence of an undiagnosed disease, health-related anxiety, and bodily preoccupation. Although limited by small samples, lack of randomization and control groups, the absence of long-term follow-up, limited generalizability, and the absence of validated outcome measures,⁷⁻¹² the existing treatment literature suggests a role for a variety of psychosocial interventions, although none has been conclusively validated. In the only large-scale, rigorous, randomized controlled trial to date, cognitive therapy and behavioral stress management were both more effective than a waiting list control condition.¹³

We have proposed that hypochondriasis can be understood as a self-perpetuating and self-validating disorder of cognition and bodily perception.¹⁴⁻¹⁶ In this formulation, personally threatening life events prompt

Context Hypochondriasis is a chronic, distressing, and disabling condition that is prevalent in ambulatory medical practice. Until recently, no specific treatment has been clearly demonstrated to be effective.

Objective To assess the efficacy of a cognitive behavior therapy (CBT) for hypochondriasis.

Design A randomized, usual care control group design, conducted between September 1997 and November 2001. The individual primary care physician was the unit of randomization, and all patients clustered within each physician's practice were assigned to the experimental treatment (individual CBT and a consultation letter to the primary care physician) or to the control condition. Subjects were assessed immediately before and 6 and 12 months after the completion of treatment.

Setting and Participants Participants were 80 patients from primary care practices and 107 volunteers responding to public announcements, all of whom exceeded a predetermined cutoff score on a hypochondriasis self-report questionnaire on 2 successive occasions.

Intervention A scripted, 6-session, individual CBT intervention was compared with medical care as usual. The CBT was accompanied by a consultation letter sent to the patient's primary care physician.

Main Outcome Measures Hypochondriacal beliefs, fears, attitudes, and somatic symptoms; role function and impairment.

Results A total of 102 individuals were assigned to CBT and 85 were assigned to medical care as usual. The sociodemographic and clinical characteristics of the 2 groups were similar at baseline. Using an intent-to-treat analytic strategy, a consistent pattern of statistically and clinically significant treatment effects was found at both 6- and 12-month follow-up, adjusting for baseline covariates that included educational level, generalized psychiatric distress, and participant status (patient vs volunteer). At 12-month follow-up, CBT patients had significantly lower levels of hypochondriacal symptoms, beliefs, and attitudes ($P < .001$) and health-related anxiety ($P = .009$). They also had significantly less impairment of social role functioning ($P = .05$) and intermediate activities of daily living ($P < .001$). Hypochondriacal somatic symptoms were not improved significantly by treatment.

Conclusion This brief, individual CBT intervention, developed specifically to alter hypochondriacal thinking and restructure hypochondriacal beliefs, appears to have significant beneficial long-term effects on the symptoms of hypochondriasis.

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predisposed individuals to suspect that they have become ill. This suspicion leads them to selectively attend to benign bodily sensations and health information that confirm their suspicion and to ignore disconfirmatory evidence. Benign bodily sensations are thereby amplified and misattributed to the putative disease, which further substantiates their disease convictions. We have developed a cognitive behavior therapy (CBT) that specifically targets the cognitive and behavioral amplifiers of benign bodily symptoms that propel this hypochondriacal cycle of disease conviction and symptom amplification. Patients are helped to correct faulty symptom attributions, restructure beliefs and expectations about health and disease, correct misunderstandings about the medical care process, modify maladaptive illness behaviors, and learn techniques of selective attention and distraction.

We hypothesized that the CBT intervention would alleviate the symptoms of hypochondriasis more effectively than medical care as usual.

METHODS

Study Design

A randomized, usual care control group design was used. To avoid a "contamination" effect, the individual primary care physician was the unit of randomization, and all patients clustered within each physician's practice were assigned to the experimental treatment (individual CBT and a consultation letter to the primary care physician) or to the control condition. Subjects were assessed immediately before and 6 and 12 months after the completion of treatment.

Subjects were recruited by screening with a self-report hypochondriasis questionnaire (composed of the Whiteley Index and Somatic Symptom Inventory), using a predetermined cutoff score selected to identify a sample, half of whom met *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)* criteria for hypochondriasis and half of whom had subthreshold (subdefinitional) hypochondriasis.

The precise cutoff score of 150 (score range, 52-258) was determined from previous studies^{3,17-19} and was chosen because we believed that many subthreshold individuals were sufficiently hypochondriacal to be in need of treatment and would be able to benefit from it. Signed informed consent was obtained in accordance with the Brigham and Women's Hospital Human Research Committee requirements. Subjects meeting initial eligibility criteria and exceeding the screening cutoff completed the baseline research battery, which included a second administration of the screening questionnaire and a determination of their medical morbidity.

Physicians were randomized immediately following the baseline research interview by a staff member not connected to the research, using a random numbers table. Randomization was stratified by physician seniority (house staff or attending staff) and by practice volume (part-time or full-time for attending staff, primary care program or traditional internal medicine program for house staff). All research data were collected by research assistants who were blind to the patient's treatment status, and the study therapists and investigators had no foreknowledge of treatment assignment. The therapists had no role in data collection. Subjects were compensated for each research interview, but not for undergoing treatment.

Subjects and Settings

Subjects were accrued from 2 sources: (1) successive patients attending primary care practices in 2 large, academic medical centers, and (2) volunteers responding to public announcements of a treatment study for "health anxiety and hypochondriasis."

The inclusion criteria were age older than 18 years, English fluency and literacy, having seen a primary care physician in the last 12 months, and exceeding the hypochondriasis screening cutoff score on both occasions. Exclusion criteria included major medical morbidity expected to worsen signifi-

cantly in the next 12 months; somatoform pain disorder; psychosis or suicide risk; and ongoing, symptom-contingent, disability determinations, workers' compensation proceedings, or litigation.

Treatment Conditions and Therapists

Cognitive behavior therapy was administered individually in six 90-minute sessions at weekly intervals. Each session was tightly scripted (manual available from the authors) and devoted to 1 of 5 factors that cause patients to amplify somatic symptoms and misattribute them to serious disease: attention and bodily hypervigilance, beliefs about symptom etiology, circumstances and context, illness and sick role behaviors, and mood. Each session consisted of educational information about the symptom amplifiers, an illustrative exercise, and a discussion to personalize the material presented. The 3 study therapists had master's or doctoral degrees and prior CBT experience. Treatment sessions were held in offices in the department of psychiatry.

Because it was considered essential that the patients' ongoing medical management be coordinated with the CBT to alter their understanding of symptoms, disease, and medical care, a standardized consultation letter was sent to each patient's primary care physician. This letter (available from the authors) contained the following 5 practical suggestions for medical management that were designed to augment the individual therapy: (1) make improved coping with somatic symptoms rather than symptom elimination the goal of medical management; (2) uncouple access to the physician from symptom status by scheduling regular appointments; (3) provide only limited reassurance; (4) explain the patient's symptoms using the model of cognitive and perceptual symptom amplification; and (5) be conservative in medical diagnosis and treatment, within the bounds of appropriate medical practice.

Treatment fidelity was assessed by auditing audiotapes of randomly se-

lected therapy sessions from all 3 therapists; adherence to the CBT manual was excellent. Receipt of the consultation letter was acknowledged by 96.8% of the primary care physicians.

Assessment

Outcome Variables. The primary outcome measure was the Whately Index, a widely used self-report questionnaire of hypochondriacal attitudes and beliefs, whose validity, reliability, and sensitivity to change have been demonstrated.^{20,21} Secondary outcomes included health-related anxiety, assessed with the Health Anxiety Inventory, a 14-item, self-report questionnaire that is minimally influenced by the presence of major medical illness and has good validity, internal consistency, and reliability.²² The frequency of hypochondriacal thoughts was assessed with the Hypochondriacal Cognitions Questionnaire, requiring the patient to rate how often each of 18 disease-related thoughts occurs. Hypochondriacal somatic symptoms were assessed with the Somatic Symptom Inventory.^{17,23} The reliability, internal consistency, and convergent validity of this 13-item questionnaire have been demonstrated previously.^{17,18,23-26} A clinical diagnosis of *DSM-IV* hypochondriasis was made by trained research assistants using the Structured Diagnostic Interview for Hypochondriasis, a highly structured interview shown in previous work to have concurrent, convergent, and discriminant validity.²⁷⁻²⁹

The tendency to amplify benign bodily sensation and experience it as noxious, unpleasant, and alarming was assessed with the Somatosensory Amplification Scale. This questionnaire has good reliability and validity and is sensitive to change as a result of attention training.^{14,30-33}

Role impairment and functional status were determined with the Functional Status Questionnaire.³⁴ This valid and reliable self-report questionnaire was developed for use in ambulatory medical populations.^{34,35} It includes subscales assessing intermediate activities of daily liv-

ing (eg, doing errands, working around the house) and social activities (eg, seeing friends, participating in community activities).

Covariates. Psychiatric comorbidity was assessed with the Hopkins Symptom Checklist-90.^{36,37} This widely used, 90-item instrument has excellent psychometric properties and provides an overall measure of generalized psychiatric distress.³⁷ Participant status indicated whether the subject was a patient accrued in the clinic or a volunteer recruited from the community. Aggregate medical morbidity was assessed in 2 ways: the Duke Severity of Illness Scale^{38,39} was used for patients accrued from primary care practices. This structured audit of the medical record was conducted by a research physician who was blind to treatment status. For study volunteers, their primary care physicians provided 2 ordinal ratings of aggregate medical morbidity.

Data Analysis

An intent-to-treat approach was used in all analyses. The last-observation-carried-forward approach was used to impute scores for missing data values. Although the physician was the unit of randomization, only 11 physicians had more than 1 subject. Thus, any approach to estimating clustering as an adjustment for physician effects would likely lead to unstable estimates of the within-cluster correlation. Therefore, to address the potential effect of clustering on the results, we selected 1 subject at random from each of these 11 physicians for analysis, resulting in the exclusion of 24 subjects ($n=163$). These results did not differ in any respect from those obtained with the full sample ($N=187$), which was therefore used for all subsequent analyses. The 2 groups were compared 6 and 12 months after treatment on the primary and secondary outcome variables. General linear modeling was used as the primary analytic approach, in which a series of univariate and multivariate repeated measures analysis of covariance (ANCOVA and MANOVA) models were used. This approach permitted the modeling of

both individual and sets of outcome measures as a function of treatment effects (between subjects), time of assessment (within subjects), and covariates (educational level, generalized psychiatric distress, and participant status [patient vs volunteer]). Hence, the interaction effect of treatment \times time was of most interest. In cases where the overall MANOVA was significant, or a priori contrasts were planned, only the ANCOVA results are presented, using the Greenhouse-Geisser adjustment. Individual, planned contrasts were calculated for each outcome measure comparing baseline and 6-month, and baseline and 12-month assessments, respectively. Effect sizes and threshold values for clinical significance were also derived for each analysis. All tests of statistical significance were interpreted with a criterion of $P<.05$. Statistical analyses were performed using SPSS, release 12.0.1 for Windows (SPSS Inc, Chicago, Ill).

RESULTS

Subject Accrual

The study was conducted between September 1997 and November 2001. A total of 6307 individuals completed the screening questionnaire, of whom 776 (12.3%) exceeded the cutoff score (FIGURE). Two hundred nineteen individuals declined to participate, 156 proved to be ineligible, and 214 could not be reached subsequently, resulting in a total of 187 subjects (30.2% of those eligible) who participated. A random sample ($n=191$) of the 589 nonparticipants was compared with those who participated. The nonparticipants were older (46.4 vs 42.3 years, $t_{376}=2.96$, $P=.003$), had less education (19.1% completed college vs 29.4%, $\chi^2_6=42.6$, $P<.001$), and were more likely to be male (38.9% vs 23.5%, $\chi^2_1=10.5$, $P<.001$).

Of the 102 patients in the treatment arm, 63 (61.8%) attended all 6 sessions, 13 (12.7%) attended 4 or 5 sessions, 12 (11.8%) attended 1 to 3 sessions, and 14 (13.7%) attended none. Six-month follow-up was obtained for 85 (83.3%) of the 102 treatment pa-

tients and for 76 (89.4%) of the 85 control patients (87% in person and 13% by telephone). Twelve-month follow-up was obtained for 92 (90.2%) of treatment patients and 78 (91.8%) of control patients (84.7% in person and 15.3% by telephone). These attrition rates do not differ significantly between groups. Six-month follow-up was obtained for 92% of treatment completers (those attending ≥ 4 sessions) and 57.6% of treatment dropouts. Twelve-month follow-up was obtained for 94.7% of treatment completers and 76.9% of treatment dropouts.

Treatment Groups at Baseline

The sociodemographic characteristics of the 2 treatment groups did not differ significantly (TABLE 1). They were predominantly women, middle-aged, and reported a history of hypochondriasis for approximately 11 years. Educational level and generalized psychiatric distress did not differ significantly in the 2 groups at baseline but were used as covariates in the analyses because of their established relationships to the outcome variables of interest. The treatment and control groups did not differ significantly in aggregate medical morbidity at baseline.

Eighty patients were recruited from primary care practices and 107 were volunteers. At baseline, volunteers were significantly more symptomatic and more disabled than patients on the major outcome measures of hypochondriacal symptoms ($P < .001$), health-related anxiety ($P < .001$), and intermediate activities of daily living ($P < .001$). Repeated measures of ANCOVA were performed on the Whiteley Index modeled as a function of participant status (patients vs volunteers), treatment (CBT vs usual care) and assessment point (baseline, 6 months, and 12 months), and all 2- and 3-way interaction terms. The 3-way interaction was significant ($F_{2,362} = 3.38$, $P = .04$), indicating that volunteers had higher Whiteley Index scores at baseline and experienced a larger treatment effect (difference between CBT and usual care) at 12 months than the patients

($F_{1,181} = 5.04$, $P = .03$), but not at 6 months ($F_{1,181} = 2.37$, $P = .13$). Consequently, participant status (patient or volunteer) also was included as a covariate in the models.

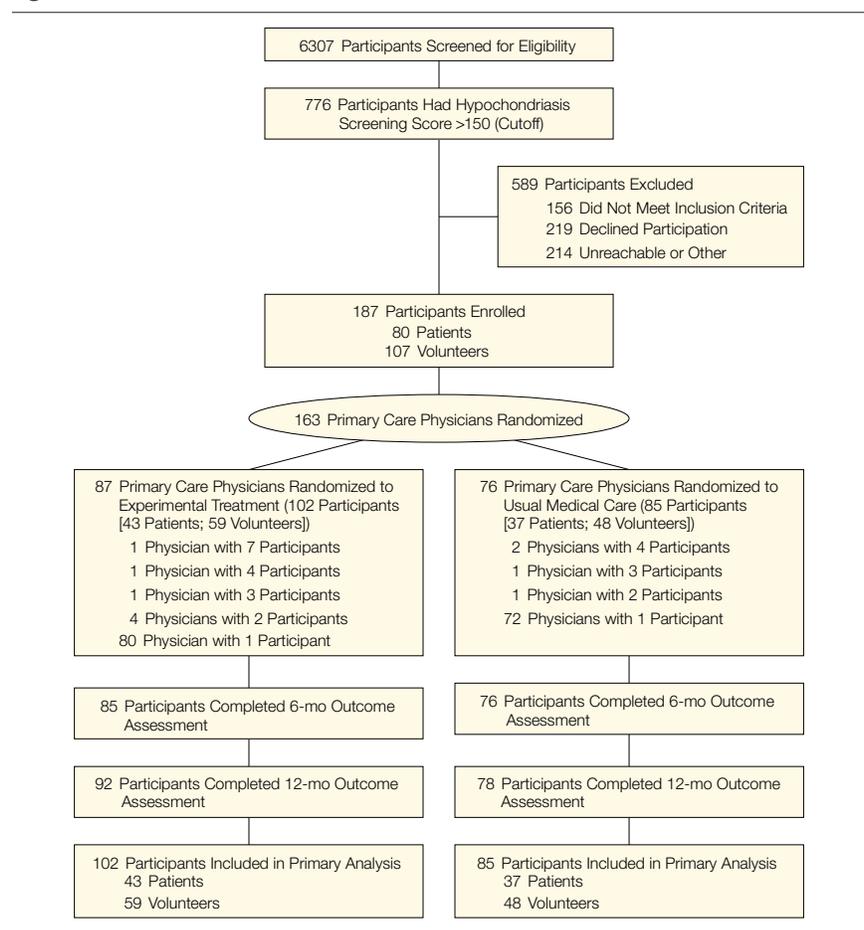
Treatment Outcomes

The results for the Whiteley Index, the primary outcome measure, are shown in TABLE 2, using repeated measures ANCOVA. There was a statistically significant interaction effect for group (treatment vs control) by assessment time (baseline, 6-month, and 12-month follow-up). Post-hoc tests of within-subjects contrasts disclosed a statistically significant improvement in the treatment vs control group at both 6-month ($F_{1,182} = 20.1$, $P < .001$) and 12-month follow-up, compared with baseline ($F_{1,182} = 14.2$, $P < .001$). The treat-

ment effect size for the Whiteley Index was $r = 0.31$ at 6 months and $r = 0.27$ at 12 months.

The secondary outcome measures of hypochondriacal symptoms were analyzed in the same manner as the Whiteley Index. For hypochondriacal thought frequency, health anxiety, and somatosensory amplification, the interactions between group and assessment were statistically significant, indicating a significant treatment effect on these measures (see Table 2). Post-hoc tests of within-subjects contrasts for hypochondriacal thought frequency revealed a significantly greater improvement in the treatment vs control group at 6-month ($F_{1,182} = 6.97$, $P = .009$), and at 12-month follow-up ($F_{1,182} = 5.64$, $P = .02$). For health anxiety, post-hoc tests of within-subjects contrasts re-

Figure. Profile of Randomized Controlled Trial



vealed a significantly greater improvement in the treatment vs control group at both 6 months ($F_{1,182}=6.73, P=.01$) and 12 months compared with baseline ($F_{1,182}=7.01, P=.009$). For am-

plification, individual tests of within-subjects contrasts revealed a significantly greater improvement in the treatment vs control group at 6 months ($F_{1,182}=8.47, P=.004$) and at 12 months

($F_{1,182}=7.70, P=.006$). Hypochondriacal somatic symptoms were not significantly improved by treatment.

The 2 treatment groups were compared on measures of functional status using MANCOVA as the omnibus test, with intermediate and social activities combined as dependent measures, again including baseline educational level, psychiatric comorbidity, and participant status as covariates. TABLE 3 indicates a statistically significant interaction effect for group by assessment time for intermediate activities ($F_{2,364}=12.32, P<.001$), but no statistical significance for social activities ($F_{2,364}=2.29, P=.10$). Post-hoc tests of within-subjects contrasts for intermediate activities of daily living revealed that there was a statistically significant improvement in the treatment vs control group at both 6 months ($F_{1,182}=18.44, P<.001$) and at 12 months ($F_{1,182}=18.30, P<.001$). The treatment effect size for intermediate activities at 12 months was $r=0.30$. For social activities, post-hoc tests of within-subjects contrasts revealed that there was a statistically significant improvement in the treatment vs control group at 12 months ($F_{1,182}=3.74, P=.05$), but no significance at 6 months ($F_{1,182}=3.06, P=.07$). The treatment effect size for social activities at 12 months was $r=0.23$.

No therapist effect was found, ie, when the patients treated by each of the therapists were compared, no significant differences were found for any of the outcome measures. Although the intervention did not include treatment for comorbid psychiatric disorder, subjects were free to obtain such treatment as they or their physicians saw fit. At 6-month follow-up, 20 CBT patients (19.6%) and 19 control patients (22.4%) had initiated a psychotropic medication or care with a mental health professional since inception. At 12 months, 6 CBT patients (5.9%) and 5 control patients (5.9%) had initiated a psychotropic medication or mental health care since their 6-month follow-up. These rates do not differ significantly between groups.

Table 1. Sociodemographic Characteristics

	Treatment Group, No. (%) (n = 102 [59 Volunteers, 43 Patients])	Control Group, No. (%) (n = 85 [48 Volunteers, 37 Patients])	P Value
Age, mean (SD), y	40.66 (12.60)	44.29 (13.75)	.06
Women	76 (74.5)	67 (78.8)	.49
Ethnicity			.23
White	72 (70.6)	63 (74.1)	
Black	17 (16.7)	11 (12.9)	
Hispanic	2 (2.0)	7 (8.2)	
Other	11 (10.7)	4 (4.8)	
Education			.12
Graduate/professional	29 (28.4)	17 (20.0)	
College graduate	34 (33.3)	21 (24.7)	
Some college	21 (20.7)	23 (27.0)	
High school graduate	11 (10.8)	19 (22.4)	
Grades 7-11	7 (6.8)	5 (5.9)	
Employed	70 (68.6)	53 (62.4)	.37
DSM-IV diagnosis of hypochondriasis	67 (65.7)	48 (56.6)	.20
Age of onset, mean (SD), y	30.54 (14.00)	32.65 (15.79)	.34

Abbreviation: DSM-IV indicates Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition.

Table 2. Hypochondriacal Symptoms (N = 187)

	Treatment Group, Mean (SE [95% CI])*	Control Group, Mean (SE [95% CI])*	P Value†
Whiteley In dex (score range, 1-5)‡			<.001
Baseline	3.58 (0.054 [3.47-3.68])	3.51 (0.060 [3.38-3.62])	
6-mo follow-up	2.82 (0.075 [2.68-2.97])	3.21 (0.083 [3.05-3.38])	
12-mo follow-up	2.65 (0.084 [2.48-2.81])	30.02 (0.093 [2.85-3.21])	
Hypochondriacal thought frequency (score range, 1-5)‡			.008
Baseline	2.09 (0.066 [1.98-2.21])	2.29 (0.064 [2.16-2.41])	
6-mo follow-up	1.75 (0.057 [1.63-1.86])	2.14 (0.063 [2.01-2.26])	
12-mo follow-up	1.62 (0.056 [1.51-1.73])	2.02 (0.062 [1.90-2.14])	
Health anxiety (score range, 1-4)‡			.004
Baseline	2.68 (0.047 [2.58-2.77])	2.71 (0.051 [2.61-2.81])	
6-mo follow-up	2.29 (0.047 [2.20-2.38])	2.51 (0.051 [2.41-2.61])	
12-mo follow-up	2.20 (0.051 [2.10-2.30])	2.44 (0.056 [2.33-2.55])	
Somatosensory amplification (score range, 1-5)‡			.003
Baseline	3.25 (0.067 [3.12-3.38])	3.04 (0.073 [2.90-3.19])	
6-mo follow-up	2.92 (0.068 [2.79-3.06])	2.96 (0.074 [2.81-3.11])	
12-mo follow-up	2.82 (0.070 [2.68-2.96])	2.87 (0.077 [2.72-3.03])	
Hypochondriacal somatic symptoms (score range, 1-5)‡			.08
Baseline	2.73 (0.048 [2.63-2.82])	2.81 (0.053 [2.70-2.91])	
6-mo follow-up	2.16 (0.056 [2.05-2.27])	2.42 (0.061 [2.30-2.55])	
12-mo follow-up	2.00 (0.056 [1.88-2.11])	2.24 (0.061 [2.18-2.36])	

Abbreviation: CI, confidence interval.

*Adjusted for baseline educational level, psychiatric comorbidity, and participant status.

†P value based on Greenhouse-Geisser adjustment for the interaction between group and assessment from analysis of variance (ANOVA) model covarying for educational level, psychiatric comorbidity, and participant status.

‡Higher scores indicate more severe symptoms.

COMMENT

This 6-session CBT, specifically targeting the cognitive and perceptual mechanisms thought to underlie hypochondriasis, appears to significantly improve a range of hypochondriacal symptoms, beliefs, and attitudes. These effects are evident at 6-month follow-up and persist at 12 months. The effects on role functioning are not consistently significant at 6 months but emerge at 12 months, the primary end point. These treatment effects are seen using an intent-to-treat analysis, after adjusting for psychiatric comorbidity, sociodemographic characteristics, and participant status (patient vs volunteer) at baseline. The findings are compatible with the only other major trial reported to date and expand on it by having a control group available for comparison at long-term follow-up.¹³

Though the magnitude of the treatment effect is modest, it is important to remember that hypochondriasis generally has been considered a refractory and chronic disorder (the mean duration of illness was 11 years in this study) for which there has been no empirically validated treatment. In addition, this CBT was brief (only 6 sessions) and included no follow-up “booster” sessions. Finally, patients were not treated in the study for comorbid psychiatric disorder, and the continued presence of these disorders likely moderated the treatment effect.

The study has several limitations. First, many eligible patients did not participate, limiting the generalizability of the findings. Those who did consent to participate might have been more receptive to a psychosocial approach and hence benefited more from it than those who did not consent. On the other hand, the study participants might be less severely hypochondriacal and more responsive to their medical physician's ministrations and/or more likely to improve spontaneously, which would tend to reduce the treatment effect. In addition, 25% of the subjects attended less than 4 treatment sessions, suggesting that future efforts must be directed toward reducing treatment dropout. This

Table 3. Functional Status and Role Impairment (N = 187)

	Treatment Group, Mean (SE [95% CI])*	Control Group, Mean (SE [95% CI])*	P Value†
Intermediate activities of daily living (0-100)‡			
Baseline	74.92 (2.01 [70.89-78.96])	74.99 (2.25 [70.57-79.43])	<.001
6-mo follow-up	82.54 (2.11 [78.39-86.71])	70.61 (2.31 [66.05-75.18])	
12-mo follow-up	83.42 (2.03 [79.43-87.42])	72.23 (2.22 [67.85-76.62])	
Social activities (0-100)‡			
Baseline	81.41 (2.35 [76.78-86.05])	81.61 (2.58 [76.52-86.69])	.10
6-mo follow-up	86.02 (2.20 [81.69-90.35])	80.55 (2.41 [75.80-85.30])	
12-mo follow-up	86.11 (2.34 [81.50-90.72])	79.26 (2.56 [74.21-84.32])	

*Adjusted for baseline educational level, psychiatric comorbidity, and participant status.

†P value based on Greenhouse-Geisser adjustment for the interaction between group and assessment from analysis of variance (ANOVA) model covarying for educational level, psychiatric comorbidity, and participant status.

‡0-100 (100 = maximal function).

is not unusual for such studies however; Kashner et al,⁴⁰ for example, found that 56% of somatization disorder patients randomized to group therapy failed to attend a single session.

Second, we lacked an “attention” control, ie, a generic psychosocial intervention providing nonspecific attention, support, concern, and positive expectation. This limits our ability to attribute the treatment effect to the specific cognitive and behavioral strategies of the intervention. However, the fact that the cognitive processes thought to underlie the disorder (eg, hypochondriacal cognitions, health beliefs, amplification) improved with treatment suggests that the treatment had a specific effect.

Third, considerable improvement occurred in the control group. This was likely due to the inadvertent inclusion of patients with transient hypochondriasis, probably because the 2 screening measures were too close to each other in time (approximately 3 weeks). Additionally, regression to the mean and the supportive effect of being enrolled in a longitudinal study contributed to the high rate of spontaneous improvement. A Hawthorne effect may also have occurred whereby control physicians, having learned of the study, made a greater effort to help their hypochondriacal patients.

Finally, study subjects came from 2 different sources. Participant status, however, was included as a covariate in all analyses, and the fact that these 2 groups differed at baseline confers some measure of generalizability on the findings.

Hypochondriacal attitudes and concerns improved more than somatic symptoms did. This finding, although it might seem counterintuitive, was actually expected: the treatment was intended to improve coping with symptoms rather than curing them outright (“care rather than cure”). This had both an empirical and a conceptual basis. Empirically, clinical experience and intervention trials for a variety of functional somatic syndromes suggest that the patients who do best are those who learn to compensate for, rather than attempting to eliminate, their somatic distress. Conceptually, hypochondriacal somatic symptoms cannot simply be stripped away with symptomatic treatment because they exist for underlying psychological and interpersonal reasons. This suggests that a realistic goal in treating hypochondriasis is amelioration of distressing fears and beliefs and improved coping, rather than the elimination of somatic symptoms per se.

We are unable to partial out the variance in treatment effect between the CBT and the physician consultation letter. That the latter may have been beneficial is suggested by 2 studies with somatization disorder patients in which a psychiatric consultation letter alone resulted in lower health care costs along with either improved or stable physical functioning.⁴¹⁻⁴³ This points to the critical importance of seamlessly integrating the psychosocial care of these patients with their medical care.

The treatment offered in this study was not attractive to many hypochon-

driacal patients, and only 30% of those eligible entered the trial. Hypochondriacal individuals are by definition convinced of the medical nature of their condition and therefore psychosocial treatment seems nonsensical to them. Although a major problem, this should not detract from the fact that those patients who did undergo treatment benefited from it. And since hypochondriasis is a prevalent problem in ambulatory medical practice,^{6,44} this fraction of hypochondriacal patients still represents a sizeable population. The

treatment must be made more attractive in the future by seamlessly integrating it into the primary care process and conducting it in the medical setting (as our treatment was not). The treatment effect could also be strengthened by increasing the number of sessions to 8 and by adding booster sessions.

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Study concept and design: Barsky, Ahern.

Acquisition of data: Barsky, Ahern.

Analysis and interpretation of data: Barsky, Ahern.

Drafting of the manuscript: Barsky, Ahern.

Critical revision of the manuscript for important intellectual content: Barsky, Ahern.

Statistical expertise: Ahern.

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REFERENCES

- Kellner R. *Somatization and Hypochondriasis*. New York, NY: Praeger; 1986.
- Noyes R, Kathol RG, Fisher MM, Phillips BM, Suelzer M, Woodman CL. One-year follow-up of medical outpatients with hypochondriasis. *Psychosomatics*. 1994;35:533-545.
- Barsky AJ, Fama JM, Bailey ED, Ahern DK. A prospective 4-5 year study of DSM-III-R hypochondriasis. *Arch Gen Psychiatry*. 1998;55:737-744.
- Lipsitt DR, Starcevic V. *Hypochondriasis: Modern Perspectives on an Ancient Malady*. New York, NY: Oxford University Press; 2001.
- Kellner R, Abbott P, Pathak D, Winslow WW, Umland BE. Hypochondriacal beliefs and attitudes in family practice and psychiatric patients. *Int J Psychiatry Med*. 1983;13:127-139.
- Barsky AJ, Wyshak G, Klerman GL, Latham KS. The prevalence of hypochondriasis in medical outpatients. *Soc Psychiatry Psychiatr Epidemiol*. 1990;25:89-94.
- Warwick HM, Clark DM, Cobb AM, Salkovskis PM. A controlled trial of cognitive behavioural treatment of hypochondriasis. *Br J Psychiatry*. 1996;169:189-195.
- Avia MD, Ruiz A, Olivares ME, et al. The meaning of psychological symptoms: effectiveness of a group intervention with hypochondriacal patients. *Behav Res Ther*. 1996;34:23-31.
- Warwick HMC, Marks IM. Behavioural treatment of illness phobia and hypochondriasis: a pilot study of 17 cases. *Br J Psychiatry*. 1988;152:239-241.
- Stern R, Fernandez M. Group cognitive and behavioral treatment for hypochondriasis. *BMJ*. 1991;303:1229-1231.
- Papageorgiou C, Wells A. Effects of attention training in hypochondriasis: a brief case series. *Psychol Med*. 1998;28:193-200.
- Bouman TK, Visser S. Cognitive and behavioural treatment of hypochondriasis. *Psychother Psychosom*. 1998;67:214-221.
- Clark DM, Salkovskis PM, Hackmann A, et al. Two psychological treatments for hypochondriasis. *Br J Psychiatry*. 1998;173:218-225.
- Barsky AJ, Wyshak G. Hypochondriasis and somatosensory amplification. *Br J Psychiatry*. 1990;157:404-409.
- Barsky AJ. Somatosensory amplification and hypochondriasis. In: Lipsitt DR, Starcevic V, ed. *Hypochondriasis: Modern Perspectives on an Ancient Malady*. New York, NY: Oxford University Press; 2001:223-248.
- Barsky AJ. A 37-year-old man with multiple somatic complaints. *JAMA*. 1997;278:673-679.
- Barsky AJ, Wyshak G, Klerman GL. Transient hypochondriasis. *Arch Gen Psychiatry*. 1990;47:746-752.
- Barsky AJ, Wyshak G, Klerman GL. Hypochondriasis: an evaluation of the DSM-III criteria in medical outpatients. *Arch Gen Psychiatry*. 1986;43:493-500.
- Barsky AJ, Cleary PD, Sarnie MK, Klerman GL. The course of transient hypochondriasis. *Am J Psychiatry*. 1993;150:484-488.
- Pilowsky I. Dimensions of hypochondriasis. *Br J Psychiatry*. 1967;113:89-93.
- Pilowsky I. A general classification of abnormal illness behaviours. *Br J Med Psychol*. 1978;51:131-137.
- Salkovskis PM, Rimes KA, Warwick HMC, Clark DM. The Health Anxiety Inventory: development and validation of scales for the measurement of health anxiety and hypochondriasis. *Psychol Med*. 2002;32:843-853.
- Weinstein MC, Berwick DM, Goldman PA, Murphy JM, Barsky AJ. A comparison of three psychiatric screening tests using receiver operating characteristic (ROC) analysis. *Med Care*. 1989;27:593-607.
- Barsky AJ, Wyshak G, Latham KS, Klerman GL. Hypochondriacal patients, their physicians, and their medical care. *J Gen Intern Med*. 1991;6:413-419.
- Kroenke K, Spitzer RL, deGruy FV, Swindle R. A symptom checklist to screen for somatoform disorders in primary care. *Psychosomatics*. 1998;39:263-272.
- Barsky AJ, Wyshak G, Klerman GL. Medical and psychiatric determinants of outpatient medical utilization. *Med Care*. 1986;24:548-560.
- Noyes R, Happel RL, Yagla SJ. Correlates of hypochondriasis in a nonclinical population. *Psychosomatics*. 1999;40:461-469.
- Noyes R, Kathol R, Fisher M, Phillip B, Suelzer M, Holt C. The validity of DSM-III-R hypochondriasis. *Arch Gen Psychiatry*. 1993;50:961-970.
- Barsky AJ, Cleary PD, Spitzer RL, Williams JBW, Wyshak G, Klerman GL. A structured diagnostic interview for hypochondriasis: a proposed criterion standard. *J Nerv Ment Dis*. 1992;180:20-27.
- Speckens AEM, Spinhoven P, Sloekers PPA, Bolk JH, van Hemert AM. A validation study of the Whiteley Index, the Illness Attitude Scales, and the Somatosensory Amplification Scale in general medical and general practice patients. *J Psychosom Res*. 1996;40:95-104.
- Gramling SE, Clawson EP, McDonald MK. Perceptual and cognitive abnormality model of hypochondriasis: amplification and physiological reactivity in women. *Psychosom Med*. 1996;58:423-431.
- Wise TN, Mann LS. The relationship between somatosensory amplification, alexithymia, and neuroticism. *J Psychosom Res*. 1994;38:515-521.
- Barsky AJ, Wyshak G, Latham KS, Klerman GL. The Somatosensory Amplification Scale and its relationship to hypochondriasis. *J Psychiatr Res*. 1990;24:323-334.
- Jette AM, Davies AR, Cleary PD, et al. The Functional Status Questionnaire: reliability and validity when used in primary care. *J Gen Intern Med*. 1986;1:143-149.
- Cleary PD, Jette AJ. Reliability and validity of the Functional Status Questionnaire. *Qual Life Res*. 2000;9:747-753.
- Derogatis LR. *The SCL-90-R: Administration, Scoring, and Procedures Manual II*. Towson, Md: Clinical Psychometric Research; 1983.
- Derogatis LR. *SCL-90-R: Administration, Scoring, and Procedures Manual*. 3rd ed. Minneapolis, Minn: National Computer Systems Inc; 1994.
- Parkerson GR Jr, Michener JL, Wu LR, et al. Associations among family support, family stress, and personal functional health status. *J Clin Epidemiol*. 1989;42:217-229.
- Parkerson GP, Broadhead WE, Chin-Kit JT. Quality of life and functional health of primary care patients. *J Clin Epidemiol*. 1992;45:1303-1313.
- Kashner TM, Rost K, Cohen B, Anderson M, Smith GR. Enhancing the health of somatization disorder patients: effectiveness of short-term group therapy. *Psychosomatics*. 1995;36:462-470.
- Rost K, Kashner TM, Smith GR Jr. Effectiveness of psychiatric intervention with somatization disorder patients: improved outcomes at reduced costs. *Gen Hosp Psychiatry*. 1994;16:381-387.
- Smith GR, Rost K, Kashner TM. A trial of the effect of a standardized psychiatric consultation on health outcomes and costs in somatizing patients. *Arch Gen Psychiatry*. 1995;52:238-243.
- Smith GR Jr, Monson RA, Ray DC. Psychiatric consultation in somatization disorder: a randomized controlled study. *N Engl J Med*. 1986;314:1407-1413.
- Escobar JI, Gara M, Waitzkin H, Cohen Silver R, Holman A, Compton W. DSM-IV hypochondriasis in primary care. *Gen Hosp Psychiatry*. 1998;20:155-159.

Paradoxical Therapy in Conversion Reaction

Paradoxical therapy consists of suggesting that the patient intentionally engages in the unwanted behaviour such as performing compulsive ritual or wanting a conversion attack. In this study, the subjects were selected by the emergency unit psychiatrist from patients who were admitted to the emergency unit with pseudoseizure. The diagnoses was based on DSM-IV criteria. Paradoxical intention was applied to half of the 30 patients with conversion disorders; the other half were treated with diazepam in order to examine the efficiency of the paradoxical intention versus diazepam. In both groups the differences of the anxiety scores at the beginning of the study were found to be insignificant ($z=1.08$, $p=0.28$). Of the 15 patients who completed paradoxical intention treatment, 14 (93.3%) responded favorably to paradoxical intention. On the other hand of 15 patients who completed diazepam therapy, 9 (60%) responded well to therapy and 6 patients carried on their conversion symptoms at the end of 6 weeks. Paradoxical intention-treated patients appeared to have greater improvements in anxiety scores ($z=2.43$, $p<0.015$) and conversion symptoms ($t=2.27$, $p=0.034$) than the diazepam-treated patients. The results of the present study are encouraging in that paradoxical intention can be effective in the treatment of conversion disorder.

Key Words : *Psychotherapy; Intention; Conversion Disorder; Diazepam*

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INTRODUCTION

Paradoxical intention (PI) is a technique that was described and developed by Frankl (1), originally in the context of logotherapy. It can be defined as those interventions in which the therapist apparently promotes the worsening of problems rather than their removal (2). Over the last decade, PI has been started to be used as a popular technique by a variety of therapists who have incorporated the technique into their existing clinical practices.

Behavioral researchers have reported a number of case studies supporting the efficacy of PI in the treatment of emotional, behavioral and psychiatric problems. The paradoxical approach has been reported to be successful with symptoms such as obsessive behavior and thinking, insomnia, migraine headaches, anorexia nervosa, phobic neurosis and psychotic states (3-7). However, despite the widespread application of PI to anxiety related problems, we have not found any report on conversion disorder, an anxiety disorder which is known to be more prevalent in lower sociocultural classes and counts up to more than 55% of the psychiatric diagnoses in our emergency unit. One of the application criteria for paradoxical approaches defined by Rohrbaugh et al. is as follows; "Where opposition is low and symptoms are seen by the patient as outside of control" (8). In conversion disorder, there is no patient opposition to symptoms, and symptoms are outside of the patients control.

There are a variety of paradoxical techniques employed in therapy. Perhaps the most common and best known paradoxical technique is symptom prescription (9). It is applied as a positive or negative intention. In positive intention, the patient is advised or instructed to continue or exaggerate the symptoms and associated behaviors. For an anxious patient, this intention may look like this: "Try to be as nervous as you can" or as used in this study "Try to experience the sensations just before you pass out" and the patient is encouraged to have a conversion attack.

Benzodiazepines are well known to be effective on anxiety (10-13). Wardle and colleagues reported a study on the effects of 5-15 mg/day diazepam on subjects with agoraphobia. In this study, PI was applied to half of the patients with conversion disorders; the other half were treated with diazepam in order to compare the efficiency of paradoxical intention versus anxiolytics in conversion disorder.

MATERIALS AND METHODS

The subjects were selected by the emergency unit psychiatrist from patients who were admitted to the emergency unit with pseudoseizure. The diagnoses were based on DSM-IV criteria (14). All patients with an abnormal EEG, organic disease, axis I or II disorder, previous psychiatric treatment were

Table 1. Sociodemographic variables of the paradoxical intention and drug groups

	PI group	Drug group
Age (range) (yr)	23 (16-30)	27 (18-35)
Sex		
Women	15	14
Man	-	1
Education		
Illiterate	5	3
Primary school	10	11
High school	-	1
Treatment duration (days)	42	48

excluded. Finally, thirty patients (29 women and 1 man), diagnosed as conversion disorder were randomly divided into two groups by means of a computer.

PI group consisted of 5 illiterates and 10 primary school graduates. The patients' mean age was 23 yr (ranging from 16 to 30). Of the patients who were in the diazepam-treated group, 3 were illiterates, 11 were primary school graduates, and 1 from high school. The patients' mean age was 27 yr (ranging from 18 to 35). The overall mean duration of conversion disorder was 42 days (mean 34 days for the PI group, 48 days for the diazepam-treated group) (Table 1).

All patients were assessed by a psychiatrist who was undisclosed to the subjects' group throughout the study. The anxiety score was measured for each patient before and after the treatment, using the Hamilton Rating Scale for anxiety (HRSA) (15). The frequency (number of attacks within the past week) of the conversive attacks were noted for each patient, and changes in these scores were converted to percentages. After the six week treatment period, changes in the above scores were analyzed.

All patients were treated by another psychiatrist. Patients treated with diazepam were offered appointments at the days 10-20-30-45 of treatment to review their progress, to reinforce the use of diazepam, and to regulate the dosage of diazepam. This group consisting of outpatients was treated with diazepam in a dosage of 5-15 mg. At the end of the treatment period, patients treated with diazepam were assessed for anxiety and conversion symptoms.

The patients in the PI group were informed about the nature of the treatment, what was expected of them, and approximately how long the treatment would last. The relationship between anxiety and conversion disorder was discussed with each patient. After obtaining their written consent, they were hospitalized. The patient and family members were interviewed separately. During the history-taking, special attention was given to anxiety provoking situations and experiences specific to the patient. Two sessions were planned for each day. During the session the patient was encouraged to imagine an anxiety provoking situation and/or experience. In most cases the patient was successful to carry out the session without much help from the therapist. In some cases who exhibited resistance, phrases such as "Imagine yourself in that same place

Table 2. The difference of anxiety scores in both groups

	Drug (n=15)	PI (n=15)	z*	p
Anxiety score before treatment	25.60±4.27	27.60±5.00	1.08	.280
Anxiety score after treatment	18.20±3.47	14.47±5.36		
Difference	7.27±4.56	13.13±5.67	2.43	.015
Z [†]	3.24	3.41		
p	.0012	.0007		

*Mann-Whitney U test; [†]Wilcoxon matched pairs test.

with the same person... Think of this as it is happening right now.... try to experience the same emotions you did then..... It's all right if you pass out (or whatever the symptom specific to the patient)" were helpful. In conclusion, we helped the patients to re-experience their specific traumatic events and promoted the patients to try to have conversion attacks. At the end of the three-week period, patients were discharged. We invited the patients to visit us three weeks later, and changes in clinical anxiety scores and conversion were assessed. Family members were interviewed for verification.

Statistical analyses

The baseline anxiety scores of two groups were compared using Mann-Whitney U test. The differences of anxiety scores of each group from baseline to the end were compared using Wilcoxon matched-pairs test. At the end of the study, the differences of anxiety scores of two groups were compared to each other using Mann-Whitney U test. The percentage of recovery from conversion disorder was assessed and the results were analyzed by t-test.

RESULTS

In both groups the differences of the anxiety scores at the beginning of the study were found to be insignificant ($z=1.08$, $p=0.28$).

The scores of the HRSA at the beginning of the study were decreased significantly at the end of the treatment in both diazepam-treated and ($z=3.24$, $p=0.0012$), PI groups ($z=3.41$, $p=0.0007$).

In both groups, the differences in anxiety scores found at the end of the study were compared to one another, and in the PI group, the decrease in anxiety scores were found to be more significant than the diazepam-treated group ($z=2.43$, $p=0.015$) (Table 2).

Patients who had no conversive symptoms within the past 2 weeks of the last control were considered as well-responders to the treatment. Of the 15 patients who completed PI treatment, 14 (93.3%) responded favorably to PI at the end of 6-week therapy, only one patient did not respond well to the PI

and at the end of 6 weeks this patient carried on her conversion symptom. On the other hand of 15 patients who completed diazepam therapy, 9 (60%) responded well to therapy and 6 patients carried on their conversion symptoms at the end of 6 weeks. In the PI group, the recovery rate was more significant than in the diazepam-treated patient group ($t=2.27$, $p=0.034$).

DISCUSSION

Our findings confirm the fact that PI is applicable to the conversion disorder. Although PI is found to be more effective than diazepam which is accepted to be a valuable therapeutic agent in the treatment of conversion disorder, it is not easy to make a complete explanation to the varying rates of improvement between the two treatment groups. This can be related to many factors, e.g., patients receiving diazepam were not hospitalized. They lived in their previous environment. This situation may provide a perpetuity of the symptom-context relationship and secondary gains.

The efficiency of PI may also be related to our paradoxical method. Since the patients were asked to behave symptomatically in an unusual surrounding, the relationship between the context and symptom disappeared. Also, the symptoms lost their surrounding supports and secondary gains.

Patients observed their own symptoms from other patients; this provided the patients with an insight into their diseases. After 3 to 4 days, some of the patients acquired a humorous view to their own conversion. When we asked, "Why can't you be ill anymore?" some of them replied "I find my illness funny". As we encouraged the patients frequently to try to have very severe conversion attacks which are related to the reexperience of a specific traumatic event (at least twice a day; in the morning and in the evening), patients may have acquired desensitization to their anxiety-related problems and satisfaction of their symptoms. This may bring about a change of attitude towards the symptoms which enables the patients to place themselves at a distance from the symptoms.

For example, a 32-yr old patient started having conversive symptoms (passing out without full loss of consciousness, intact sensation to verbal or painful stimuli but unable to respond, and intact memory to this period) when she learned that her husband was about to marry another woman. These symptoms reoccurred whenever she recalled this "traumatic event". She was suggested to frequently recall the fact that her husband had intended to marry another woman. During the first five days, she remembered the traumatic event each time she had a conversive attack. On the sixth day, she had no symptoms despite remembering the traumatic event. On asking her why, she answered that passing out was only a game she played to avoid the real challenge, and it would be better to solve the problem by talking to her husband face to face.

According to psychoanalytic theory, conversion disorder is

caused by the repression of unconscious psychological conflict which arises anxiety and the conversion of the anxiety into a physical symptom. The psychological conflict is in the patient's unconscious, and the physical symptom is not under voluntary control (14). From this point of view, it seems reasonable that the best approach to this situation would be an insight therapy where the unconscious material is recovered, the patient gains insight to the primary conflict and the necessity to utilize conversion, as a defence mechanism is no longer required. In clinical practice, however, there are certain drawbacks of such an approach. Most of the time, psychoanalytic therapies require long periods of time and are expensive. On the other hand, as mentioned above, conversion disorder is more prevalent in the lower socioeconomic classes in which the patient can not afford the expenses or the time necessary for such a therapy. Another problem arises when the secondary gains become so systematized that the patient presents a "La belle indifference" which is an indicator of resistance to therapy. Maybe the most important of all, patients who suffer conversive symptoms are so convinced that they have organic diseases, e.g., paralysis, epilepsy, this concern alone produces an intolerable anxiety which promotes conversion. Once this vicious circle is established, the patient is most likely to resist psychotherapy and seek help in other fields.

PI is an inexpensive short term psychotherapy. Although it does not remove the primary conflict or challenge the secondary gains, it provides an invaluable insight to the anxiety arising at the second half of the vicious circle. Once the patient eventually perceives the close relationship between the occurrence of the symptoms and anxiety, it is much more easier to establish a self confidence and defeat resistance.

It is sometimes unavoidable to prescribe anxiolytics to patients who exhibit clinical anxiety. This type of treatment is much less expensive than any form of psychotherapy, including PI. However, we have observed that our patients coming from rural areas have important refill problems. Also the risk of dependence or drug abuse are important drawbacks for anxiolytics. Most important of all, patients who are treated with drugs alone are more likely to underestimate the importance of facing the real life problems, since the drug is doing it for them. PI, on the other hand, emphasizes these problems and provides a sense of control and confidence over them. As a matter of fact, paradoxical interventions are much helpful to enable the patient to acquire a sense of detachment towards his/her neurosis by developing a humorous view. Paradoxical intention is the clinical application of Allport's statement; "The neurotic who learns to laugh at himself may be on the way to self-management, perhaps to cure" (19).

REFERENCES

1. Frankl VE. *Paradoxical intention: A logotherapeutic technique. Am J Psychother* 1960; 14: 520-35.

2. Cade B. *Paradoxical techniques in therapy*. *J Child Psychol Psychiatry* 1984; 25: 509-16.
3. Adshead G, Drummond LM, Mercer S. *Paradoxical intention and anti-exposure in a non-compliant, obsessive-compulsive ritualiser*. *Br J Psychiatry* 1988; 153: 821-3.
4. Bootzin RR, Perlis ML. *Nonpharmacologic treatments of insomnia*. *J Clin Psychiatry* 1992; 53 (Suppl): 37-41.
5. Skorzevska A, Lal S. *Spasmodic torticollis and phobic neurosis*. *Neuropsychobiology* 1990; 24: 8-11.
6. Purdon C, Clark DA. *Suppression of obsession-like thoughts in non-clinical individuals: impact on thought frequency, appraisal and mood state*. *Behav Res Ther* 2001; 39: 1163-81.
7. Fehm L, Margraf J. *Thought suppression: specificity in agoraphobia versus broad impairment in social phobia?* *Behav Res Ther* 2002; 40: 57-66.
8. Rohrbaugh M, Tennen H, Press S, White L. *Compliance, defiance and therapeutic paradox: guidelines for strategic use of paradoxical interventions*. *Am J Orthopsychiatry* 1981; 51: 454-67.
9. Omer H. *Integrating paradoxical interventions in the normal course of therapy: nonspecific approach*. *Am J Psychother* 1986; 40: 572-81.
10. Wardle J, Hayward P, Higgitt A, Stabl M, Blizard R, Gray J. *Effects of concurrent diazepam treatment on the outcome of exposure therapy in agoraphobia*. *Behav Res Ther* 1994; 32: 203-15.
11. Hayward P, Wardle J, Higgitt A. *Benzodiazepine research: current findings and practical consequences*. *Br J Clin Psychol* 1989; 28: 307-27.
12. Posternak MA, Mueller TI. *Assessing the risks and benefits of benzodiazepines for anxiety disorders in patients with a history of substance abuse or dependence*. *Am J Addict* 2001; 10: 48-68.
13. Rickels K, DeMartinis N, Aufdembrinke B. *A double-blind, placebo-controlled trial of abecarnil and diazepam in the treatment of patients with generalized anxiety disorder*. *J Clin Psychopharmacol* 2000; 20: 12-8.
14. Guggenheim FG. *Somatoform disorders*. In: Kaplan HI, Sadock BJ, editors, *Comprehensive textbook of psychiatry*, 6th ed. Williams & Wilkins, Baltimore 1995; 1251-70.
15. Fresquet A, Sust M, Lloret A, Murphy MF, Carter FJ, Campbell GM, Marion-Landais G. *Efficacy and safety of lesopitron in outpatients with generalized anxiety disorder*. *Am J Pharmacother* 2000; 34: 147-53.

Psychological Assessment and Treatment of Somatization: Adolescents with Medically Unexplained Neurologic Symptoms

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Somatization Disorder: A disorder characterized by recurrent, multiple, physical complaints and symptoms for which there is no organic cause. The condition typically occurs in adolescence or in the early adult years and is rarely seen in men. The symptoms vary according to the individual and the underlying emotional conflict.

Mosby's Medical, Nursing,
and Allied Health Dictionary⁴⁵

Adolescent patients who report physical symptoms that are unexplained by physical disease or pathophysiologic processes are prevalent in health-care settings.^{4,7,8,17,33–35,44} Physical symptoms with no notable physical pathology are often referred to by primary care physicians (PCPs) as medically unexplained symptoms (MUS).^{34,40,53} Patients who present with MUS often have accompanying impairments in emotional, social, family, and educational or occupational functioning. The nature of their problems can negatively impact the overall quality of the doctor-patient relationship.^{34,54} In addition, many patients with MUS have poor treatment outcomes, higher health-care utilization rates, and a propensity to consume a disproportionate amount of health-care resources.^{31,32,58,56} Failure to recognize MUS may lead to unnecessary diagnostic testing and the use of po-

tentially harmful medications. In the past few years, an emerging literature has stressed the importance of developing more effective techniques for identifying and treating patients with MUS to reduce these associated costs.^{6,11,12,26,43,46,49}

The majority of adolescent patients who experience somatic-like physical symptoms will interpret them as normal bodily discomfort; however, a small subgroup may erroneously interpret the symptoms as being positive signs of underlying physical disease.^{43,55} Fortunately, many of the somatic symptoms presented by adolescent patients are reduced or eliminated by simple reassurances and explanations from the PCP. In cases in which these techniques are not effective in eliminating or reducing the presence of somatic symptoms, it may be essential for the PCP to recognize the role of psychological factors in the etiology or maintenance of the unexplained physical symptoms and to adopt alternative management techniques. Specifically, it may be necessary for physicians to evaluate the presence of a somatoform disorder.^{34,40,44} As outlined in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) the somatoform disorders include conversion disorder, somatization disorder, pain disorder, hypochondriasis, body dysmorphic disorder, undifferentiated somatoform disorder, and somatoform disorder not otherwise specified.¹

Although patients with MUS likely would benefit from psychological or psychiatric treatments, many adolescent patients and their parents are reluctant to accept referrals for these adjunctive services^{32,29,35,56,60}; therefore, the onus often falls on physicians to appropriately screen for such problems and to make cost-effective and appropriate referrals. This chapter provides some guidelines for physicians to assist in this assessment, intervention, and referral process. Specifically, we address issues related to the assessment of psychological factors that may contribute to the development or maintenance of unexplained medical symptoms among adolescent patients and present recommendations regarding the management of such patients. Given that symptoms of depression and anxiety frequently occur among patients with MUS, assessment and treatment issues relevant to these psychological problems are emphasized. We present an overview of the advantages in integrating psychological screening practices into the evaluation process and provide recommendations regarding potential psychological assessment strategies. In addition, several psychologically based treatment interventions that are relevant to addressing the psychosocial issues in adolescents with MUS are reviewed.

IMPORTANT TERMINOLOGY

Historically, a wide variety of descriptive terms have been used to refer to somatic-like physical symptoms. *Somatization* and *MUS* are some of the more frequently encountered terms in the empirical literature. Additional terms include *neurosis*, *functional somatic symptoms*, *hypochondriasis*, and *psychosomatics*.⁵³ The lack of consistent terminology attests to the fact that the etiology of these physical symptoms is poorly understood.³⁵ Many of these terms imply that the patient's symptoms are produced by an underlying medical condition, whereas others suggest that they arise primarily from a psychiatric or psychological disorder. Often the aforementioned terminology has the propensity to make patients feel that their symptoms are not real, are "all in their mind," or that there is nothing medically wrong with them.⁵² None of these terms explicitly acknowledge that physical symptoms and psychological factors often coexist and are interrelated in complex ways.⁵³

Given the lack of consistent and agreed-upon terminology, we have chosen to define MUS as neurologic-like symptoms that, after the completion of appropriate medical and laboratory examinations, cannot be attributed to physical disease or pathophys-

iologic processes alone. This definition takes into account the fact that some adolescent patients may have a verifiable neurologic condition that is further exacerbated by unexplained factors or by clear psychological factors. In addition, the definition includes cases of medical syndromes that are by definition unexplained, including chronic fatigue syndrome, pseudoseizures, fibromyalgia, and chronic headaches. Finally, the proposed definition of MUS accounts for patients who have clear psychiatric disorders (e.g., anxiety, depressive, or somatoform disorders) that are accompanied by unexplained medical or neurologic complaints as well as those patients who report MUS in the absence of any identifiable physical or psychiatric disorder.⁴⁶

EPIDEMIOLOGY AND DEMOGRAPHICS

The prevalence rates of unexplained somatic symptoms among children and adolescents fluctuate depending on symptom focus, terminology, clinical setting, and the patient sample studied. Some of the more commonly studied MUS include headaches, abdominal pain, back and chest pain, low energy levels, dizziness, fatigue, numbness and tingling sensations in the limbs, and gastrointestinal symptoms.^{13,14,18,20,23,48,67} Prevalence rates of these symptoms among children and adolescents range from 10–25%.^{7,18,67} Somatization rates increase during the adolescent years. Furthermore, MUS tend to be more prevalent among adolescent females and are associated with lower socioeconomic levels.^{3,13,47}

Adolescent patients with somatic complaints are a heterogeneous population, which makes it difficult to establish a precise etiology. Although it is not clear how factors combine to perpetuate the development of somatic symptoms in adolescents, there is strong evidence to suggest that certain contributory factors play a significant role in maintaining the symptoms.^{19,21} Adolescent patients with a history of MUS are more likely to be diagnosed with a psychiatric or psychological disorder, including anxiety, depressive, and somatoform disorders.^{28,32,64} Other psychosocial factors are also associated with MUS. Adolescents under a great deal of life stress may be at greater risk for developing unexplained physical complaints. For example, family discord (e.g., divorce or parental conflict), history of physical and sexual abuse, peer conflicts, school-related problems (e.g., transfer to a new school or unrealistic academic expectations), and drug abuse and dependence are all associated with higher rates of MUS.^{2,14,19,65} The presence of verifiable physical illness or a strong preoccupation with physical illness within the family system may lead the adolescent to focus more intently on bodily symptoms, resulting in more frequent health-care utilization to rule out the possibility of actual physical disease.^{8,19}

DIAGNOSTIC ISSUES

It goes without saying that all medical complaints should be attended to with gravity and caution. The foremost requirement in diagnosing and treating MUS is to rule out potential disease and other pathophysiologic processes that may have initiated or maintain the presenting physical complaints. The most important diagnostic concern in MUS is the exclusion of neurologic and other general medical conditions. Failure to appropriately diagnose real physical pathology can have serious, deleterious consequences.

In the diagnostic process there is always risk of making one of two types of classification errors.³⁹ Type I errors, or false positives, occur when a physician identifies existing pathology where none actually exists. With respect to diagnosing patients with MUS, type I errors occur when a patient's medical complaints are attributed to physical pathology rather than to psychiatric, psychological, or other factors that may better ac-

count for the complaints. In contrast, type II errors, or false negatives, occur when the physician fails to identify physical pathology when it actually exists. For example, a patient's physical complaints are attributed to psychological or psychiatric factors when, in fact, the symptoms are the result of underlying physical pathology. The likelihood of making classification errors is further complicated in situations where the symptoms can be characteristic of both psychological disorders and neurologic disease or other medical conditions.^{30,39} The risk of making a type II error is exemplified in the following hypothetical case-scenario:

Case

A 15-year-old high school student originally sought neurologic help for symptoms of fatigue, double vision, and numbing and tingling sensations in the limbs on the left side of his body. After taking a full history, performing a brief physical examination, and making a referral for a neurologic evaluation, it was concluded that there was no substantial evidence of organic disease. On finding no evidence of organic disease, the patient was referred to a psychiatrist for further evaluation and treatment. The patient's adoptive mother, who herself had an extensive history of health problems, was quite concerned about her son's health and continued to press for a medical explanation for his physical symptoms. By the end of the first year following the onset of the symptoms, a comprehensive team consisting of a primary care physician, neurologist, psychiatrist, psychologist, and neuropsychologist had already evaluated the patient.

The patient's past history of anxiety, depression, and substance abuse and his tendency to experience the symptoms only in the classroom environment and when he had an exam or a major project due led to widespread agreement among the health-care team that his symptoms were better accounted for through psychological and other psychosocial factors. Neuropsychological and laboratory testing results also supported this diagnosis. When his symptoms dissipated after participating in psychological interventions (i.e., individual therapy, relaxation training, and stress management), all of the doctors who were involved in the case were relieved to learn of these positive treatment outcomes. During a routine physical examination after several symptom-free months, however, his primary care physician performed a Quick Neurological Screening Test (QNST) and a Mini Mental Status Exam (MMSE) and noticed some cognitive difficulties and motor and sensory impairments on the left side of his body. The doctor immediately referred him for additional laboratory testing. An abnormal MRI revealed a small right frontotemporal brain lesion. During surgery, it was found that the tumor was inoperable.

Several neurologic disorders found in adolescent populations are being reviewed elsewhere in this issue. These disorders include, but are not limited to, seizure disorders, migraine headaches, consciousness disorders (e.g., vertigo or syncope), multiple sclerosis, Guillain-Barré syndrome (GBS), and cerebral palsy. One of the more commonly found neurologic disorders in adolescents is multiple sclerosis (MS). The presenting symptoms in the early stages of this medical condition (e.g., weakness of limbs, double vision, numbness) are often vague and transient. Therefore, diagnostic procedures often fail to detect the presence of the disease while not necessarily implying that the etiology of the patient's symptoms is entirely psychological. In other cases, patients may display psychiatric or psychological symptoms that are very similar to or that closely mimic symptom patterns found in many organic neurologic conditions. All of these scenarios suggest the necessity of comprehensive, differential diagnostic procedures.

Further complicating the picture, many adolescent patients who have been diagnosed with a demonstrable medical condition also may begin to develop and experience

additional MUS. For example, adolescent patients diagnosed with an organic seizure disorder may be at risk for developing a medical syndrome known as pseudoseizures.³⁶ Concluding that such unexplained symptoms are entirely accounted for by psychological factors has obvious implications. As Lezak states, “many serious and treatable neurological diseases [MS] often present with vague, often transient symptoms that can worsen with stress and temporarily diminish or even disappear altogether with symptomatic or psychological intervention.”³⁹ One obvious implication is that physicians fail to intervene with appropriate medical care and rely instead on psychosocial treatments to address the symptomatology.

Although a full history, complete physical examination, and appropriate laboratory procedures often resolve these diagnostic dilemmas, there are other situations in which it is much more difficult to make an appropriate diagnosis. Fortunately, the advent of advanced laboratory techniques (e.g., MRI, PET, CT, EEG) has significantly reduced many difficulties associated with differential diagnosis.³⁹ Many of these instruments have been quite effective in localizing brain and other types of neurologic pathology, which has ultimately reduced the risk of missing underlying physical disease.

However, when patients are in early developmental stages of a pathologic condition, many of the available laboratory instruments may not be sensitive enough to detect the presence of an organic condition.³⁹ It may be particularly difficult to make a precise determination of the etiology of the symptoms unless the patient begins to develop “hard core” signs of an underlying organic condition.^{39,43} After several months or years, it may be discovered that the patient is indeed experiencing a pathologic condition as symptoms become more chronic and severe and less transient.³⁹

The differential diagnosis task for physicians is indeed complicated. In order to make appropriate medical diagnoses and to understand underlying etiology of presenting symptoms, the PCP often will conduct a full medical history, a thorough physical examination, and additional necessary laboratory procedures. These assessment practices are generally sufficient in either detecting physical disease should it exist or in reassuring patients that nothing is wrong with them. In fact, more recent research has shown that many patients who present with physical complaints will report a reduction in physical symptoms following the diagnostic process, especially if they have a healthy physician–doctor relationship, have been properly evaluated, and have been closely followed up in subsequent visitations.^{11,12}

That being said, there are other circumstances where psychological factors play the sole or primary role in the development and maintenance of MUS. Often in patients with MUS, physician reassurance is not effective in allaying concerns or diminishing physical complaints. Although it is imperative to rule out physical pathology, strict adherence to the traditional assessment practices described previously result in subjecting these patients to unnecessary diagnostic testing, unwarranted referrals to specialists, and other costly medical treatments while offering negligible clinical benefit for the individual patient. In cases where patients’ MUS appear to be more closely related to psychiatric and psychological factors, alternative assessment practices and resulting treatment recommendations are required.

Use of psychological instruments may aid PCPs in identifying relevant psychopathology and contributing psychosocial stressors that may better explain the presenting symptomatology. Although such assessment can be accomplished by referring these patients to a psychologist or psychiatrist, it may be more efficient and cost-effective to directly assess the patient in the context of the primary care setting, especially if the multidisciplinary team includes a clinician trained in evaluating and treating psychological problems.

PSYCHOLOGICAL ASSESSMENT IN PRIMARY CARE SETTINGS

It is well established that PCPs represent de facto behavioral health care systems in the United States.^{42,50,62} In fact, empirical research has demonstrated clearly that approximately one half of patients with clinically significant behavioral health disorders (e.g., depression, anxiety, somatoform disorders, or substance abuse) will seek treatment from their PCP in the primary care setting.^{42,59,61,63} Unfortunately, the process of identifying and treating these patients with significant behavioral health disorders in primary care settings is inadequate in many respects. It has been suggested that approximately one third to one half of patients with behavioral health problems fail to be identified by primary care providers.⁴² Furthermore, in cases in which patients have been diagnosed with a psychiatric disorder, PCPs often under-appreciate the seriousness of patients' problems. In fact, one study reported that 50–75% of patients diagnosed with major depression and were actively suicidal had consulted with their PCP shortly before they committed suicide.^{9,42}

Given that primary care settings are typically the first point of entry into the health care system, it becomes imperative to include identification of behavioral health disorders (i.e., psychiatric problems) in the assessment process. Specifically, in order to effectively intervene with patients with MUS, it is essential to incorporate psychological or psychiatric assessment into a comprehensive evaluation. One strategy is for PCPs to integrate efficient and effective psychological screening instruments into the evaluation process.

Over the past few years, psychological testing has gained support and momentum and is now recognized as an important tool for primary care physicians to add to their professional armamentarium.⁴¹ Currently, a number of different screening instruments are available for the PCP to use in the primary care setting. Some of these instruments are time consuming, comprehensive, and require a qualified clinician to administer and score. However, others are less time consuming, can be administered by most primary medical care staff, and are relatively easy to administer within the context of primary care settings.

Although it is beyond the scope of this chapter to provide detailed information about all of the available psychological screening instruments, the authors offer a brief review of several psychological instruments that may be used by the PCP to identify the presence of psychiatric disorders or other psychosocial problems. In addition, Table 1 outlines a more comprehensive list of several commercially available instruments that may be useful to physicians. For a more detailed description of available psychological screening instruments and tests please see reference 41.

Prime-MD

The Primary Care Evaluation of Mental Disorders (Prime-MD) is a two-stage diagnostic instrument that is helpful in identifying psychological disorders relevant to adolescent patients with MUS. The Prime-MD is specifically designed for the primary care physician and consists of two major components: the "screening/case finding" component and the "diagnostic" component.²⁵ The screening/case finding component is a self-report patient questionnaire (PQ) that the patient can complete while waiting in the reception area prior to seeing the PCP. Although the PQ is primarily designed to be a paper-and-pencil self-report questionnaire, it can also be administered orally in interview format. The PQ consists of 25 yes and no questions and assesses the following five categories of psychological disorders: mood disorders, anxiety disorders, somatoform disorders, alcohol abuse and dependence, and eating disorders. If the PQ component suggests that the patient needs additional evaluation for the possible presence of a psychological disorder, the PCP can administer the second diagnostic component, the Clinician Evaluation Guide (CEG). The CEG is a physician-administered interview and is based

TABLE 1. Psychological Instruments That Can Be Administered and Scored in the Primary Care Setting

Instrument	Description	Administration Time	Use	Ownership
Reynolds Adolescent Depression Scale (RADS)	Self-report instrument; designed to identify depressive symptoms in adolescents (5th–12th grades)	5–10 minutes	S	Psychological Assessment Resources, Inc., P.O. Box 998, Odessa, FL 33556-9908; 813-968-3003; http://www.parinc.com
Beck Depression Inventory-II (BDI-II)	Self-report instrument; designed to measure severity level of depression; for ages 13 and older	5–10 minutes	S	The Psychological Corporation, 555 Academic Court San Antonio, TX 78204-2498; 800-211-8378; http://www.hbem.com
Beck Anxiety Inventory (BAI)	Self-report instrument; designed to measure anxiety; for ages 17 and up	5–10 minutes	S	The Psychological Corporation (see contact information above)
Beck Hopelessness Scale (BHS)	Self-report instrument; designed to measure level of hopelessness; for ages 17 and up	5–10 minutes	S	The Psychological Corporation (see contact information above)
Mini-Mental State Examination (MMSE)	Clinician-administered instrument; designed to determine level of cognitive functioning	5–10 minutes	S	Psychological Assessment Resources, Inc. (see contact information above)
Prime-MD	Clinician-administered and self-report instrument; designed to measure depression, anxiety, somatoform, alcohol, and more	15 minutes	S,D	Pfizer, Inc. http://www.pfizer.com
Symptom Checklist-90-Revised (SCL-90-R)	Self-report instrument; designed to measure depression, anxiety, alcohol, somatization, psychosis, and more	15–20 minutes	S	National Computer Systems 5605 Green Circle Dr., Minnetonka, MN 55343; 800-627-7251 x5151
Brief Symptom Inventory (BSI)	Self-report measure; designed to measure depression, anxiety, alcohol, and more	5–15 minutes	S	National Computer Systems (see contact information above)
Suicidal Ideation Questionnaire (SIQ, SIQ-Jr., ASIQ)	Self-report instrument; designed to measure relative risk for suicidal ideation; for grades 7th to college	5–10 minutes	S	Psychological Assessment Resources (see contact information above)
State-Trait Anger-2 Inventory (STAXI-2)	Self-report instrument; designed to identify for the presence of anger problems; for ages 16 and older	5–10 minutes	S	The Psychological Corporation (see contact information above)
State-Trait Anxiety Inventory (STAI)	Self-report instrument; designed to measure anxiety; for high school and college students	5–10 minutes	S	Psychological Assessment Resources (see contact information above)

Table continues on next page.

TABLE 1. Psychological Instruments That Can Be Administered and Scored in the Primary Care Setting (*Cont.*)

Instrument	Description	Administration Time	Use	Ownership
Child Behavior Checklist (CBCL)	Parent self-report instrument; designed to measure social competence and behavioral problems; for ages 4–18	15–20 minutes	S	Thomas M. Achenbach, Ph.D., Department of Psychiatry, University of Vermont, Burlington, VT 05401-3456
Conflict Behavior Questionnaire (CBQ)	Parent self-report instrument; designed to measure level of parent–adolescent conflict; for adolescents	5–10 minutes	S	Psychological Assessment Resources (see contact information above)
Quick Neurological Screening Test (QNST)	Clinician-administered instrument; designed to screen for neurological impairments; for ages 5–adult	15–20 minutes	S	Academic Therapy Publications, 20 Commercial Boulevard, Novato, CA 94949-6191; 415-883-3314
Parenting Stress Inventory (PSI)	Parent self-report instrument; designed to measure level of parenting stress	15 minutes	S	Psychological Assessment Resources (see contact information above)
Adolescent Drinking Index	Self-report instrument; designed to measure the severity of problems with drinking; for ages 12–18	5–10 minutes	S	Psychological Assessment Resources (see contact information above)
Beck Scale for Suicide Ideation	Self-report instrument; designed to measure severity of suicidal risk	5–10 minutes	S	Psychological Assessment Resources (see contact information above)
Brief Neuropsychological Cognitive Examination (BNCE)	Clinician-administered instrument; designed to assess for cognitive impairments; for ages 18 and older	30 minutes	S	Psychological Services, Inc., 100 West Broadway, Suite 1100, Glendale, CA 91210; 818-244-0033

Abbreviations: S = screener; D = diagnostic

on DSM-IV criteria; it is divided into five different modules for each of the five categories of psychological disorders listed above. Based on the results of the PQ, the physician decides which of the five modules should be administered to the patient.²⁵

Mini-Mental Status Examination

The Mini-Mental Status Examination (MMSE) is a screening instrument that was developed to assess problems in cognitive functioning.¹⁶ The MMSE is a widely used instrument, takes approximately 5–10 minutes to administer, and provides a quantitative score of the severity of cognitive impairment. The MMSE consists of 11 items that are designed to examine functions such as memory, attention, and orientation. It also investigates abilities in naming, following both a verbal and written command, writing a complete sentence, and copying two geometric figures that intersect with each other.¹⁶ A patient who scores a 23 or below may be experiencing a significant cognitive or neurologic impairment. It is important to note that the MMSE is not designed to replace more extensive neurologic and neuropsychological instruments. Patients who obtain a significant score can be referred for more extensive neuropsychological testing.

Beck Anxiety Inventory

The Beck Anxiety Inventory (BAI) is an instrument designed to measure symptoms associated with anxiety disorders, which is one of the most prevalent behavioral health disorders in the United States.⁵¹ The BAI consists of 21 items and can be administered quickly in the primary care setting. The majority of the items on the BAI assess objective physiologic symptoms such as numbness, tingling, heart racing, difficulty in breathing, and dizziness. The remaining items examine more subjective symptoms, including fear of losing control or of something negative happening in the future. The BAI produces a quantitative score that ranges from 0 to 63, with higher numbers indicating greater symptom severity.¹⁵ Because many MUS often coexist with anxiety disorders, the BAI may be quite helpful in identifying adolescent patients who may be suffering from some type of anxiety disorder.

TREATMENT STRATEGIES FOR PATIENTS WITH MUS

The majority of the empirical research on the treatment of somatization has focused on specific medical syndromes (e.g., irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome, abdominal pain, or chronic pain) and other psychiatric disorders, such as the somatoform, anxiety, and depressive disorders.^{5,10,43,66} The aim of the present section is to discuss an existing model of treatment that has been developed to deal more effectively with patients who present with MUS in the primary care setting.^{22,37,38} This treatment model is called the **retribution model** and has been specifically designed to help in the identification and treatment of patients suffering from somatic symptoms. The model consists of the following stages: (1) “feeling understood”; (2) “broadening the agenda”; and (3) “making the link.”²² Influenced by the work of Lesser,^{37,38} Gask²² developed this consultation model to help PCPs learn more effective techniques for identifying and treating patients presenting with somatic symptoms in primary care settings. This model aims to gradually encourage the patients to shift from a medical to a psychological standpoint regarding the etiology of presenting symptoms. It should be noted that the three-stage model does not recommend that intervention be completed in one visit, but rather suggests that additional consultations are required. In addition to providing an overview of this three-stage treatment model, we offer additional treatment guidelines that we view as important in the treatment of adolescent patients with somatic symptoms.^{19,21,22,52}

Stage 1: Feeling Understood

Because physical symptoms and psychological problems often coexist, the consultation process should involve a discussion of how physical and psychological factors interrelate and interact with each other.^{22,24} In order to develop a healthy and productive doctor–patient relationship, it is imperative that PCPs educate and explain to adolescent patients and their parents that both physical and psychological factors are important considerations in the diagnostic and treatment process.

One important task in this first stage is to take a full history. Because many adolescent patients and their parents typically are seeking a medical explanation for presenting symptoms and are quite concerned about the possibility of overlooking an underlying physical disorder, the assessment should focus initially on exploring the patient’s past medical history, which allows the PCP to make a well-informed decision as to whether or not additional laboratory examinations are needed to rule out pathophysiologic processes.¹⁹ PCPs should avoid subjecting patients to unnecessary diagnostic and laboratory investigations, unless the patient’s medical history and physical complaints clearly suggest hard core signs of physical disease.⁵⁷

TABLE 2. Sample of Suggested Questions for Adolescents with MUS

Have you recently witnessed or been exposed to any form of violence in the home or school environment?	Are you currently using any drugs, alcohol, or tobacco?
Are you having any difficulties in your interpersonal relationships?	Do any of your friends use drugs, alcohol, or tobacco?
Are any of your friends struggling with any serious emotional, social, or family problems?	Are you involved with the legal system?
Are you currently experiencing any suicidal thoughts, attempts, or plans?	Are your parents currently employed?
Are you having any school-related problems?	How are your parents getting along?
Do you have any school attendance problems?	Do you get along with your family members?
Have you noticed any changes in your academic or social functioning?	Are your parents divorced?
Do you get along with your teachers?	Is your family having any financial problems?
Have you noticed any changes in your mood?	Does your family have any history of substance use or abuse?
Are you currently experiencing any significant anxiety?	How many times have you moved over the years?
Have you had any arguments or fights with your parents or girlfriend/boyfriend?	Have your parents divorced recently?
	Have there been any recent deaths, illnesses, or injuries in your family?

The assessment should not only aim at including specific details of previous medical investigations but also explore contributing behavioral, emotional, and psychosocial factors (Table 2). Substance abuse, depressive and anxiety disorders, school-related problems, and other types of psychosocial problems may play an important role in the etiology of somatic symptoms. Assessment of significant health problems within the family, family beliefs about what might be causing the symptoms, and family conflicts is also recommended.^{19,22} Integrating psychological screening instruments (see Table 1) into this stage of the assessment process will aid PCPs in eliciting specific and reliable information regarding the presence of psychiatric or psychosocial problems.

A second task in this first stage is to conduct a brief, focused physical examination on the area of the body where the reported symptoms are believed to originate.^{22,57} Although the PCP may want to conduct only a brief physical examination to rule out the possibility of physical disease, it will also offer reassurance to the patient and parents that the PCP is taking the symptoms seriously.^{22,24} If the doctor suspects at this point that the patient's symptoms are not physical in origin and are better accounted for through a psychiatric diagnosis or other psychological factors, the PCP should avoid making definite statements that suggest that nothing is medically wrong or that the symptoms are related to mental health problems. The process of shifting the patient from a medical to a psychological perspective may not be possible in one office visit and may require several brief, regularly scheduled visits.^{22,57} The organization and timing of these consultations likely will depend on the results of the history and physical examination or whether additional laboratory tests are needed.

Stage 2: Broadening the Agenda

The main purpose of the second stage is to begin to shift gradually or broaden the agenda by encouraging the patient to recognize the interaction of physical symptoms and psychologic factors.²² One important step at this stage involves providing specific feedback

on the results of the physical examination. Based on the results of the investigation, the PCP will more than likely be able to classify the patient into one of three categories: (1) patients experiencing a verifiable physical disorder; (2) patients experiencing a demonstrable psychiatric or psychological disorder; and (3) patients experiencing a mixed-condition involving a combination of both physical disease and a psychological disorder. Although the latter group may unduly complicate the consultation process, the information presented herein is still applicable to these patients.²² Not only should feedback regarding the results of the examination be clearly presented and described to the patient, but PCPs should gradually begin to explain that many physical and bodily symptoms do not necessarily imply the presence of significant pathology; however, it is important that PCPs not insinuate that patients' symptoms are not real or that nothing is physically wrong with them.

Another crucial step in stage 2 involves the gradual process of reframing patients' complaints by linking physical symptoms, psychological factors, and other relevant factors (e.g., social, family, or emotional) identified during stage 1. For example, a patient who complains of chronic headaches, visual disturbances and memory disturbances *and* acknowledges alcohol abuse, anxiety symptoms, and school-related difficulties may be gently encouraged to consider the possibility that his or her physical symptoms are connected in some way to the psychological and psychosocial issues. As Gask points out, it is essential for PCPs to provide patients with the opportunity to discuss their viewpoints openly regarding acceptance of this multifaceted explanation for their presenting complaints.²² In order to encourage patients to entertain alternative explanations, PCPs should continue to acknowledge that patients' symptoms are real while also respecting their viewpoints regarding the nature of their complaints. After broadening the agenda, PCPs will be in a better position to shift the patient's viewpoint toward considering a psychological perspective.

Stage 3: Making the Link

The final stage of the reattribution model involves making the link between physical symptoms and psychological factors. Although many patients may have already made the link from previous office visits, others may require additional explanations and demonstrations to understand more clearly how these factors are interrelated. One simple method of making the link for patients involves providing the patient with information. For example, PCPs may explain how alcohol dependence can lead to physiologic reactions (e.g., grand mal seizures, memory impairments, transient visual illusions, or hand tremors); how an anxiety disorder can cause dizziness, paresthesias, and cognitive impairments; or how major depressive disorder can lead to psychomotor retardation and poor concentration.

With patients who have a more difficult time understanding the interrelatedness of physical and psychological symptoms, it may be necessary to provide more specific demonstrations during the office visit.²² One effective technique that can be used in the primary care setting demonstrates how hyperventilation can cause physiologic changes. For example, by facilitating over-breathing or spinning the individual in a chair, patients with panic attacks may see how their attacks are linked to many of the physiologic symptoms that they tend to experience. Alternatively, PCPs may ask patients to keep a written symptom diary to see if the symptoms are related to any environmental events or psychosocial stressors (Fig. 1). The data collected from these behavioral records may reveal that symptoms occur only during particular times, settings, or when the patient is experiencing a significant amount of stress or is thinking in a particular way. A final technique may involve exploring family background information collected from the full history. For example, it may be discovered that the patient has modeled the behavior of a family member who has struggled with a significant health-related disorder.

Record Sheet of Physical Symptoms, Thoughts, and Feelings
 Name: _____

Date	Time	SITUATION				Symptoms Rate Severity: 0-100	Automatic Thoughts	Moods Rate Severity: 0-100
		Who?	What?	When?	Where?			
3/8/02	1 PM	With friends and teacher	Completing math exam	After lunch	At school in Math Class	Dizziness (90) Numbness (85) Blurred Vision (100) Tingling in arms (70)	- I'm going to faint - I'm going to fail my exam - I'm having a stroke - My friends are going to think I am crazy - I'll never graduate from high school	Anxious (96) Depressed (50)

FIGURE 1. Sample Symptom Monitoring Form

ADDITIONAL TREATMENT CONSIDERATIONS

Location of Treatment

The location of treatment for patients with MUS depends to a great extent, on available resources. Most adolescent patients will realize that somatic symptoms are temporary and should not be interpreted as signs of serious physical disease; however, a small group of patients will continue to experience the symptoms and will actively seek reassurance and treatment from their PCP. Depending on the availability of resources, some MUS patients can be treated successfully and managed in the primary care setting by the PCP or other behavioral health-care professionals (i.e., psychiatrist, psychologist, or social worker) and will not require a referral for specialized services. However, access to adjunctive mental health services may not be readily available in many primary care facilities. In these cases, patients with a suspected psychiatric disorder or who continue to complain of recurrent somatic symptoms may require a referral for more specialized psychosocial services.

When to Make a Referral

The PCP and other members of the health-care team can manage the majority of the patients with MUS by following the management techniques reviewed in this chapter (Fig. 2). These treatment techniques should be sufficient in reducing or eliminating MUS that are perpetuated by clear psychological factors. When patients continue to report

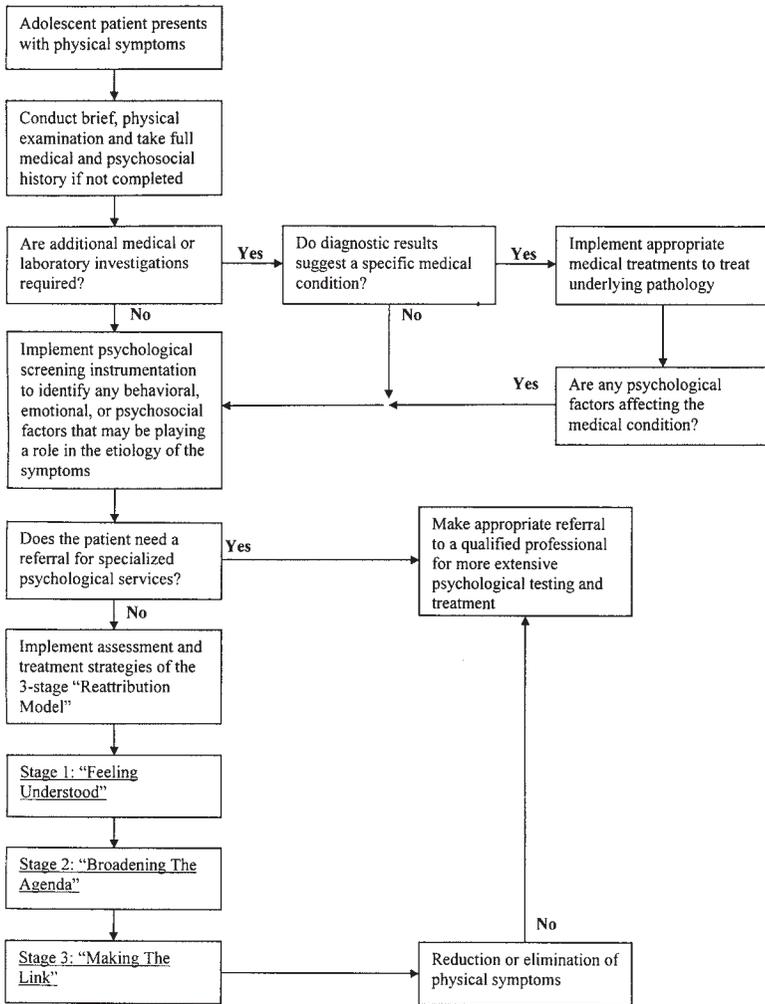


FIGURE 2. Potential flow chart of treatment process for patients with MUS.

neurologic-like symptoms with relatively little evidence of the presence of psychological factors, a referral to a neurologist or neuropsychologist for a differential diagnosis is needed. This referral will provide a more in-depth examination to rule out specific pathophysiologic conditions and other various impairments in neuropsychologic or cognitive functioning. Patients experiencing an obvious psychiatric disorder (e.g., psychiatric disorder or suicidal behavior) or other significant psychosocial problems should be referred to a qualified professional with more extensive and specialized training in treating these problems. Similarly, a referral also may be needed for those patients with verifiable medical conditions that are further exacerbated by unexplained factors or by clear psychological factors. The treatment techniques reviewed herein may help reduce MUS in individuals with verifiable medical conditions. Depending on available resources and time restraints, the PCP also may determine that a referral to a psychiatrist or psychologist is required.

A variety of specific psychological treatments are available for patients with MUS. These types of treatments may be a useful adjunct to the treatment techniques previously discussed. They also may be appropriate for patients with MUS that are exacerbated by a specific medical condition or syndrome. Behavioral and cognitive techniques have been shown to be most effective in the treatment of these groups of patients. Many of the treatments offer specific techniques, including stress management, relaxation training (e.g., deep breathing or progressive muscle relaxation), and coping skills training. Other treatment strategies may include pharmacologic agents (e.g., antidepressants), group therapy, and more individualized and explorative psychotherapy.

Doctor–Patient Relationship

As many authors point out, it is imperative to establish a high quality doctor–patient relationship with patients with MUS.^{52,57} The doctor–patient relationship plays an important role in the treatment process with these types of patients. In order to build effective rapport, it is important for PCPs to develop collaborative relationships with adolescent patients and their parents. Within the context of a collaborative relationship, PCPs will be in a better position to listen sympathetically, to take patients' symptoms seriously, and to develop a clearer understanding of what patients need. It also allows clinicians to explore psychological factors and other fears, thoughts, and beliefs that might play a significant role in the etiology of the symptoms.

CONCLUSION

Adolescents who present with unexplained neurologic symptoms in the primary care setting may be suffering from a clinically significant behavioral health disorder or some other form of psychological distress. If no adequate medical cause can be found to explain the patient's presenting symptomatology, it is important for the PCP to conduct a careful assessment of the patient's psychosocial functioning. Various psychological factors and other psychosocial problems found in adolescent patients may help explain the presence of somatic physical symptoms.

Although experienced PCPs can perform comprehensive assessments for both the presence of physical disease and the presence of significant psychopathology, it also may be beneficial to include psychologists or social workers as part of an interdisciplinary team.²⁷ Adolescent patients who have a comorbid psychiatric disorder, are actively suicidal, or are under a great deal of psychological distress should be referred for a more comprehensive psychological evaluation and treatment. Many patients and their parents are seeking a medical diagnosis for their physical symptoms rather than a diagnosis of mental illness; furthermore, they are often quite reluctant to accept a referral for psychological or psychiatric services. It is, therefore, essential for PCPs to adopt alternative assessment and treatment practices that help the patient understand the interrelatedness of physical symptoms and psychological factors.

References

1. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed, text revision. Washington DC, American Psychiatric Association, 2001.
2. Aro H: Life stress and psychosomatic symptoms among 14- to 16-year old Finnish adolescents. *Psychol Med* 17:191–201, 1987.
3. Aro H, Paronen O, Aro S: Psychosomatic symptoms among 14–16 year old Finnish adolescents. *Soc Psychiatry* 22:171–176, 1987.
4. Bass CM: *Somatization: Physical Symptoms and Psychological Illness*. Oxford, Oxford University Press, 1990.

5. Bass C, Mayou R: Chest pain and palpitations. In Mayou R, Bass C, Sharpe M (eds): Treatment of Functional Somatic Symptoms. Oxford, Oxford University Press, 1995, pp 328–349.
6. Brill SR, Patel DR, MacDonald E: Psychosomatic disorders in pediatrics. *Indian J Pediatr* 68:597–603, 2001.
7. Campo JV, Fritsch SL: Somatization in children and adolescents. *J Am Acad Child Adolesc Psychiatry* 33:1223–1234, 1994.
8. Campo JV, Jansen-McWilliams L, Comer DM: Somatization in pediatric primary care: Association with psychopathology, functional impairment, and use of services. *J Am Acad Child Adolesc Psychiatry* 38:1093–1101, 1999.
9. Cole S, Raju M, Barrett J, et al: The MacArthur Foundation Depression Education Program for primary care physicians: Background, participant's workbook, and facilitator's guide. *Gen Hosp Psychiatry* 22:299–358, 2000.
10. Creed F: Psychological treatment of irritable bowel syndrome and abdominal pain. In Mayou R, Bass C, Sharpe M (eds): Treatment of Functional Somatic Symptoms. Oxford, Oxford University Press, 1995, pp 255–270.
11. Crimlisk HL, Bhatia KP, Cope H, et al: Slater revisited: Six year follow up study of patients with medically unexplained motor symptoms. *BMJ* 316:582–586, 1998.
12. Crimlisk HL, Bhatia KP, Cope H, et al: Patterns of referral in patients with medically unexplained motor symptoms. *J Psychosom Res* 49:217–219, 2000.
13. Eminson M, Benjamin S, Shortall A, et al: Physical symptoms and illness attitudes in adolescents: An epidemiological study. *J Child Psychol Psychiatry* 37: 519–528, 1996.
14. Faul C, Nicol AR: Abdominal pain in six-year-olds: An epidemiological study in a new town. *J Child Psychol Psychiatry* 27:251–260, 1986.
15. Ferguson RJ: Using the Beck Anxiety Inventory in primary care. In Maruish ME (ed): Handbook of Psychological Assessment in Primary Care Settings. Mahwah, NJ, Lawrence Erlbaum Associates, 2000, pp 509–535.
16. Folstein MF, Folstein SE, McHugh PR: Mini-mental state: A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res* 12:189–198, 1975.
17. Fritz G, Fritsch S, Hagino O: Somatoform disorders in children and adolescents: A review of the past 10 years. *J Am Acad Child Adolesc Psychiatry* 36:1329–1338, 1997.
18. Garber J, Walker LS, Zeman J: Somatization symptoms in a community sample of children and adolescents: Further validation of the Children's Somatization Inventory. *Psychol Assess J Consult Clin Psychol* 3:588–595, 1991.
19. Garralda ME: Practitioner review: Assessment and management of somatisation in childhood and adolescence: A practical perspective. *Child Psychol Psychiatry* 40:1159–1167, 1999.
20. Garralda ME: Somatisation in children. *J Child Psychol Psychiatry* 47:13–33, 1996.
21. Garralda ME: The management of functional somatic symptoms in children. In Mayou R, Bass C, Sharpe M (eds): Treatment of Functional Somatic Symptoms. Oxford, Oxford University Press, 1995, pp 42–65.
22. Gask L: Management in primary care. In Mayou R, Bass C, Sharpe M (eds): Treatment of Functional Somatic Symptoms. Oxford, Oxford University Press, 1995, pp 391–409.
23. Goodman JE, McGrath PJ: The epidemiology of pain in children and adolescents: A review. *Pain* 46:247–264, 1991.
24. Greydanus DE, Pratt HD: Emotional and behavioral disorders of adolescents: Part I. *Adolesc Health Update* 7:1–8, 1995.
25. Hahn SR, Kroenke K, Williams JBW, et al: Evaluation of mental disorders with the PRIME-MD. In Maruish ME (ed): Handbook of Psychological Assessment in Primary Care Settings. Mahwah, NJ, US Lawrence Erlbaum Associates, 2000, pp 191–253.
26. Hartz AJ, Noyes R, Bentler SE, et al: Unexplained symptoms in primary care: Perspectives of doctors and patients. *Gen Hospital Psychiatry* 22:144–152, 2000.
27. Homnick DN, Pratt HD: Respiratory diseases with a psychosomatic component in adolescents. *Adolesc Med* 11:547–565, 2000.
28. Hotopf M, Mayou R, Wadsworth M et al: Temporal relationships between physical symptoms and psychiatric disorder: Results from a national birth cohort. *Br J Psychiatry* 173:255–261, 1998.
29. Hotopf M, Wadsworth M, Wessely S: Is somatisation a defense against the acknowledgment of psychiatric disorder? *J Psychosom Res* 50:119–124, 2001.
30. Howlerson DB, Lezak MD: The neuropsychological evaluation. In Yudofsky SC, Hales RE (eds): American Psychiatric Press Textbook of Neuropsychiatry. Washington, DC, American Psychiatric Press, 1992, pp 127–150.
31. Jackson JL, Kroenke K: Difficult patient encounters in the ambulatory clinic. *Arch Intern Med* 159:1069–1075, 1999.
32. Katon WJ, Lin E, von Korff M et al: Somatization: A spectrum of severity. *Am J Psychiatry* 148:34–40, 1991.

33. Katon WJ, Walker EA: Medically unexplained symptoms in primary care. *J Clin Psychiatry* 59:15–21, 1998.
34. Kellner R: *Psychosomatic Syndromes and Somatic Symptoms*. Washington, DC, American Psychiatric Press, 1991.
35. Kroenke K, Mangelsdorff D: Common symptoms in ambulatory care: Incidence, evaluation, therapy, and outcome. *Am J Med* 86:262–266, 1989.
36. Lancman ME, Asconpe JJ, Graves S, et al: Psychogenic seizures in children: Long-term analysis of 43 cases. *J Child Neurol* 9:404–407, 1994.
37. Lesser AL: The psychiatric and family medicine: A different training approach. *Med Educ* 15:398–406, 1981.
38. Lesser AL: Problem-based interviewing in general practice: A model. *Med Educ* 19:299–304, 1985.
39. Lezak MD: *Neuropsychological assessment*. New York, US Oxford University Press, 1995.
40. Lipowski ZJ: An inpatient program for persistent somatizers. *Can J Psychiatry* 33:275–280, 1988.
41. Maruish ME: *Handbook of Psychological Assessment in Primary Care Settings*. Mahwah, NJ, US Lawrence Erlbaum Associates, 2000.
42. Maruish ME: Introduction. In Maruish ME (ed): *Handbook of Psychological Assessment in Primary Care Settings*. Mahwah, NJ, US Lawrence Erlbaum Associates, 2000, pp 3–41.
43. Mayou R, Bass C, Sharpe M: *Treatment of Functional Somatic Symptoms*. Oxford, Oxford University Press, 1995.
44. Mayou R, Bass C, Sharpe M: Overview of epidemiology, classification, and aetiology. In Mayou R, Bass C, Sharpe M (eds): *Treatment of Functional Somatic Symptoms*. Oxford, Oxford University Press, 1995, pp 42–65.
45. *Mosby's Medical, Nursing, and Allied Health Dictionary*, 3rd ed. St. Louis, Mosby, p 1097.
46. Nimnuan C, Hotopf M, Wessely S: Medically unexplained symptoms: An epidemiological study in seven specialities. *J Psychosom Res* 51:361–367, 2001.
47. Oster J: Recurrent abdominal pain, headache and limb pains in children and adolescents. *Pediatrics* 50:429–436, 1972.
48. Rauste-von Wright M, von wright J: A longitudinal study of psychosomatic symptoms in healthy 11–18 year old girls and boys. *J Psychosom Res* 25:525–534, 1981.
49. Reid S, Whooley D, Crayford T, et al: Medically unexplained symptoms: GPs' attitudes towards their cause and management. *Fam Pract* 18:519–523, 2001.
50. Regier DA, Narrow WE, Rae DS, et al: The de facto US mental and addictive disorders service system: Epidemiologic Catchment Area prospective 1-year prevalence rates of disorders and services. *Arch Gen Psychiatry* 50:85–94, 1993.
51. Robins LN, Helzer JE, Weissman MM, et al: Lifetime prevalence of specific psychiatric disorders in three sites. *Arch Gen Psychiatry* 41:949–958, 1984.
52. Sharpe MC, Bass C, Mayou RM: An overview of the treatment of functional somatic symptoms. In Mayou R, Bass C, Sharpe M (eds): *Treatment of Functional Somatic Symptoms*. Oxford, Oxford University Press, 1995, pp 66–86.
53. Sharpe MC, Mayou RM, Bass C: Concepts, theories, and terminology. In Mayou R, Bass C, Sharpe M (eds): *Treatment of Functional Somatic Symptoms*. Oxford, Oxford University Press, 1995, pp 3–16.
54. Sharpe MC, Mayou RM, Seagroatt V, et al: Why do doctors find some patients difficult to help? *QJM* 87:187–193, 1994.
55. Sharpe MC, Peveler R, Mayou R: The psychological treatment of patients with functional somatic symptoms: A practical guide. *J Psychosom Res* 36:515–529, 1992.
56. Shaw J, Creed F: The cost of somatization. *J Psychosom Res* 35:307–312, 1991.
57. Smith GR: Treatment of patients with multiple symptoms. In Mayou R, Bass C, Sharpe M (eds): *Treatment of Functional Somatic Symptoms*. Oxford, Oxford University Press, 1995, pp 175–187.
58. Smith GR: Somatization disorder: Defining its role in clinical medicine. *J Gen Intern Med* 6:168–175, 1991.
59. Smith GR, Rost K, Kashner TM: A trial of the effect of a standardized psychiatric consultation on health outcomes and costs in somatizing patients. *Arch Gen Psychiatry* 52:238–243, 1995.
60. Speckens AEM, van Hemert AM, Spinhoven P, et al: Cognitive behavioural therapy for medically unexplained physical symptoms: A randomised controlled trial. *BMJ* 311:1328–1332, 1995.
61. Spitzer RL, Williams JB, Kroenke K, et al: Utility B, Kroenke K, et al: Utility of a new procedure for diagnosing mental disorders in primary care: The PRIME-MD 1000 study. *JAMA* 272:1749–1756, 1994.
62. Strosahl K: Confessions of a behavior therapist in primary care: The odyssey and the ecstasy. *Cognit Behav Practice* 3:1–28, 1996.
63. Tiemens BG, Ormel J, Simon GF: Occurrence, recognition, and outcome of psychological disorders in primary care. *Am J Psychiatry* 153:636–644, 1996.

64. Van Hemert AM, Hengeveld MW, Bolk JH, et al: Psychiatric disorders in relation to medical illness among patients of a general medical out-patient clinic. *Psychol Med* 23:167–173, 1993.
65. Walker LS, Garber J, Greene JW: Psychosocial correlates of recurrent childhood pain: A comparison of pediatric patients with recurrent abdominal pain, organic illness, and psychiatric disorders. *J Abdom Psychol* 102:248–258, 1993.
66. Wessely S, Sharpe M: Chronic fatigue, chronic fatigue syndrome, and fibromyalgia. In Mayou M, Bass C, Sharpe M (eds): *Treatment of Functional Somatic Symptoms*. Oxford, Oxford University Press, 1995, pp 285–312.
67. Zuckerman B, Stevenson J, Bailey V: Stomachaches and headaches in a community sample of preschool children. *Pediatrics* 79:677–682, 1987.

APPENDIX A

Population and Need-Based Prevention of Unexplained Physical Symptoms in the Community

*Charles C. Engel, Jr., and Wayne J. Katon**

SYNOPSIS

How might military medicine respond to existing research on the epidemiology, burden, natural history, and management of medically unexplained physical symptoms (MUPS) in primary care and the general population? This review of extensive published research suggests that MUPS are pervasive and contribute substantially to physical, social, occupational, and organizational impairment, psychosocial distress, unnecessary health care utilization and expenditures, and adverse health care outcomes. These studies suggest that the natural history of MUPS is influenced by a number of predisposing, precipitating, and perpetuating factors and that certain prognostic factors may help clinicians and policy makers estimate the outcomes and population needs.

We use the epidemiology of MUPS and the basic principles of population-based health care to construct an efficient MUPS prevention strategy that emphasizes a continuum of care. In the absence of randomized trial evidence of efficacy for any single multifaceted continuum of MUPS care, the prevention program suggested is conservative and reasonably achievable, lends itself to subsequent evaluation and improvement, and calls for a multifaceted, well-integrated, stepped care management approach involving

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The views expressed by Doctor Engel in this article are his own and do not reflect the official policy or position of the Department of the Army, the Department of Defense, or the U.S. Government.

- broad-based and low-intensity educational interventions delivered to every member of the military services and perhaps their family members;
- primary care-based collaborative and interdisciplinary practice teams that aim to improve short- and long-term health behaviors using a variety of behavioral strategies including education;
- information systems that use expert systems to process and feed back data obtained by using a health care-based health information system and a population survey-based health data monitoring system;
- specialized, multimodal services available for the intensive multidisciplinary management of disabling and otherwise treatment-refractory MUPS; and
- development of a “center of excellence” to lead clinical, research, and educational efforts related to MUPS in the military.

We suggest that future improvement efforts target military clinicians, military health care delivery, the military work environment, and existing methods for compensating and returning ill personnel to work.

No matter the overall process and structure of care provided for individuals with MUPS, physicians are urged to practice “person-centered” rather than “disease-centered” care. They cannot ignore their place as consultants to real people in real predicaments who are attempting to make difficult decisions potentially affecting their future health, career, relationships, and status. Hadler has stated that the role of physicians, “should be more than that of concerned citizens or even of patients’ advocates; [to that] we can add the perspective of students of the human predicament.”⁵⁸ The expanded notion of ill health as a human predicament is especially apropos in occupational and military medicine settings. Occupational and military physicians treat diseases, but of equal import is their obligation to study and prepare the workplace so those workers with illness-related work limitations can eventually make a successful return to productivity. Eventually, we are impressed that military medicine’s innovations in this area may provide an important model for civilian health care organizations seeking solutions to the difficult challenge of MUPS.

UNDERSTANDING MEDICALLY UNEXPLAINED PHYSICAL SYMPTOMS

The absence of a discerned cause for physical symptoms is best viewed through the lens of the scientific uncertainty necessarily involved in any one-to-one doctor–patient visit. We will use “MUPS” in reference to *health care use for physical symptoms that are not clinically explained by a medical etiology*. MUPS can be broken down into a four-part process. First, an individual must *experience* the symptom. In a simplified way, this might be viewed as the biological part of the process. Presumably, for one to perceive a symptom, some neurophysiological event must bring it to awareness. The second step is *cognitive*, or related to how we think about the symptom. The person perceiving a

symptom overlays some knowledge, biases, or beliefs that he or she has about the symptom and its cause, assigning it a level of medical importance. We do not seek care for most of the symptoms we experience, partly because we assign them some relatively low level of medical significance. When we seek care, we are taking a third and *behavioral* step that is mediated by our belief in the symptom's significance.

The fourth and final step is the purview of the clinician: he or she must decide the extent to which symptoms are explained by the patient's medical diagnoses. This is one of the most problematic aspects of MUPS. There is a clear potential for doctor-patient conflict in this formulation. Differing clinician and patient explanations for MUPS may be one of the most important contributors to the frustration that these symptoms create for clinicians^{61,97,154} and the dissatisfaction with care that many affected patients describe. Add some reason for doctor-patient mistrust, and the relationship can become outwardly adversarial and result in mutual rejection.¹²⁰

In occupational settings like the military, clinicians must provide care within the context of competing and sometimes unacknowledged obligations. The clinician is committed to the welfare of the employer, who is both paying the clinician's salary and providing medical benefits for the patient. This same clinician has a simultaneous duty to the health and well-being of the patient. Under these circumstances, the patient may fear that the clinician is being coerced to deny the reality of the medical problem in service to the employer's financial or political interests. The patient may feel that the clinician is more interested in keeping the patient on the job than in providing treatment. Alternatively, the clinician may suspect that the patient is exaggerating health concerns to obtain benefits. Conflicts such as these heighten doctor-patient mistrust, dampen rapport, and diminish the chance of a productive clinical encounter.

Symptom-based disorders are diagnoses based upon patient-reported physical symptoms rather than specific findings on clinical examination or diagnostic testing. Symptom-based disorders seldom offer clinicians and patients more than a label. In most instances, the prognosis, treatment, and factors that determine disability are remarkably similar across different symptom-based disorders. Observed differences are typically small and are attributable to differences in severity, the number of other symptoms involved with the syndrome, or differences in loss of functioning due to symptom location (e.g., lower-extremity joint pain impedes walking, whereas headache pain does not). The names of symptom-based disorders are usually based on hypothesized etiology (e.g., chronic Lyme disease), putative triggers (e.g., multiple chemical sensitivity), a central descriptive feature (e.g., chronic fatigue syndrome), or body region (e.g., temporomandibular disorder). Labels often use complicated terminology (e.g., fibromyalgia or myalgic encephalomyelitis) that suggests to patients, doctors, and the public that the syndrome is better understood than it actually is. Therefore, we will use the term *symptom-based disorder* to signify syndromes that are clinically diagnosed almost exclusively by using patients' verbal descriptions. Table A-1 displays some common examples of symptom-based disorders and illustrates that clinicians in nearly

every specialty encounter them. Symptom-based disorders overlap extensively, manifest remarkably similar pathophysiology, risk factors, clinical course, and prognosis, and respond to similar rehabilitative treatment approaches.^{17,23,54,67,118,158} Historically, physicians have tended to categorize MUPS and symptom-based disorders as psychiatric symptoms on the basis of exclusion. It seems most logical that only some MUPS are psychiatric in their origin.

TABLE A-1 Some Symptom-Based Diagnoses and the Specialties that Commonly Diagnose and Encounter Them

Specialty	Clinical Syndrome	Specialty	Clinical Syndrome
Orthopedics	Low back pain Patellofemoral syndrome	Dentistry	Temporomandibular dysfunction
Gynecology	Chronic pelvic pain Premenstrual syndrome	Rheumatology	Fibromyalgia Myofascial syndrome Siliconosis
Ear-Nose-Throat	Idiopathic tinnitus	Internal Medicine	Chronic fatigue syndrome
Neurology	Idiopathic dizziness Chronic headache	Infectious Diseases	Chronic Lyme disease Chronic Epstein-Barr virus Chronic brucellosis Chronic candidiasis
Urology	Chronic prostatitis Interstitial cystitis Urethral syndrome	Gastroenterology	Irritable bowel syndrome Gastroesophageal reflux
Anesthesiology	Chronic pain syndromes	Physical Medicine	Mild closed head injury
Cardiology	Atypical chest pain Idiopathic syncope Mitral valve prolapse	Occupational Medicine	Multiple chemical sensitivity Sick building syndrome
Pulmonary	Hyperventilation syndrome	Military Medicine	Gulf War Syndrome
Endocrinology	Hypoglycemia	Psychiatry	Somatoform disorders

EPIDEMIOLOGY OF SYMPTOMS AND SYMPTOM-BASED DISORDERS

Prevalence in the Community and Primary Care

Review of the epidemiology of unexplained physical symptoms necessarily involves discussion of the epidemiological literature on somatization and the somatoform disorders (e.g., conversion disorder, somatization disorder, or pain disorder). The central feature in the somatoform disorders, however, is the presence of MUPS. The absence of test abnormalities or objective physical examination findings means that a psychiatric etiology is presumed but that the actual etiology is a matter of debate. We advocate an atheoretical, nonetiologic, and phenomenological understanding of MUPS since this formulation is intellectually honest and maximally acceptable to those affected.

Population-based surveys have shown that 85 to 95 percent of community respondents experience at least one physical symptom every 2 to 4 weeks although relatively few of these symptoms are reported to physicians.¹⁶¹ The population-based Epidemiologic Catchment Area Study examined 13,538 respondents from four U.S. communities and found that 25 percent reported chest pain, 24 percent reported abdominal pain, 23 percent reported dizziness, 25 percent reported headache, 32 percent reported back pain, and 25 percent reported fatigue.⁹¹ Thirty-one percent of symptoms were medically unexplained, and the type of symptom was unrelated to the absence of explanation. Eighty-four percent of symptoms caused respondents to seek health care, take a medicine, or curtail activities.⁹¹ Over 4 percent of people had a lifetime history of multiple, chronic, unexplained symptoms and an exacerbation within the past year.^{38,142}

Other studies have shown that MUPS are associated with a high proportion of populationwide disability and health care utilization, largely because they are so common.^{39,74} For example, the 1989 National Ambulatory Medical Care Survey estimated that physical symptoms account for 57 percent of all U.S. ambulatory care visits including some 400 million clinic visits per annum.¹²⁷ Kroenke and Mangelsdorff⁹⁰ reviewed the medical records of 1,000 primary care-internal medicine patients over a 3-year period and determined the incidence, diagnostic findings, and outcomes of 14 common symptoms. At least one common symptom was present in 38 percent of patients, and only 16 percent of symptoms were felt to have an organic cause. Symptomatic patients were monitored for an average of 11 months, and for 47 percent of patients the symptom persisted throughout the follow-up period. Two-thirds of symptoms were evaluated beyond the initial history and physical examination, but only approximately 1 in 10 evaluations resulted in an organic diagnosis not apparent at the index visit. Subsequently, Kroenke et al.⁸⁸ completed an office-based survey of 410 primary care-internal medicine patients to determine the prevalence and adequacy of therapy for 15 common symptoms. Eighty-two percent of patients had one or more symptoms, and in 77 percent one or more of these symptoms had been reported to patients' physicians. However, only 39 percent of patients with fa-

tigue, dyspnea, dizziness, insomnia, sexual dysfunction, depression, and anxiety reported any noticeable response to treatment. Most other primary care research suggests that etiologies are unknown for at least 25 to 30 percent of patients with either painful or nonpainful physical symptoms.^{87,92,93}

An extensive scientific literature has shown that MUPS are strongly and consistently associated with psychosocial distress, psychiatric disorders, decreased quality of life, and increased health care utilization.^{6,18,25,38,39,56,76,90,92,129,135} Depression and anxiety are consistently associated with MUPS across many studies that have used wide-ranging methodologies including cross-sectional,¹³⁵ case-control,^{73,82,140,152,156} and longitudinal designs.¹⁵⁰ Some evidence suggests that associated high health care utilization leads to more harm and patient dissatisfaction than benefit.^{86,145}

Natural History of MUPS

MUPS are characteristically chronic and intermittently relapsing, although the natural history is reasonably variable in severity and periodicity. Factors responsible for variability in clinical outcomes may be classified as predisposing, precipitating, and perpetuating factors.

Predisposing factors are characteristics of individuals that render them more vulnerable to MUPS and related morbidity. Important predisposing factors are heredity;^{136,162} neurophysiological, neurotransmitter, and autonomic nervous system factors;^{4,31,44,52,55,83,144} early life adversity (e.g., child maltreatment),^{3,26,68,85,98,152,153,155} chronic medical illness,^{2,12,66,121,147} or chronic distress or mental illness.^{34,70} Predisposing factors may be either intrinsic (i.e., innate to the individual) or acquired (i.e., obtained during lifetime exposure or experience).

A *precipitating factor* is essentially a “straw that breaks the camel’s back,” initiating an acute episode of MUPS and related morbidity. Factors that precipitate MUPS include biological stressors,^{15,134} psychosocial stressors,^{27–29} acute psychiatric disorders,¹¹¹ and epidemic health concerns.^{14,21,24,62,69,139}

Perpetuating factors are those that maintain, exacerbate, or prolong symptoms, distress, and disability after they occur. Perpetuating factors may occur independently of the original precipitants. They include harmful illness beliefs (beliefs that lead to a maladaptive response to the symptoms),¹³² labeling effects (i.e., the adverse effects associated with viewing oneself as ill),^{40,60,63,106} misinformation,^{1,7,16,100,130,133} workplace and compensation factors,^{11,59,128,141} and social support factors.¹⁰⁷

Prognostic Factors: Prediction of Outcomes and Assessment of Future Needs

MUPS occur along a spectrum of severity and prognosis⁷⁴ ranging from mild and transient to chronic and disabling. *Prognostic factors* are individual, environ-

mental, or population characteristics that may be used to predict symptom outcomes and estimate future treatment and resource needs. The prognostic spectrum of MUPS includes acute, recurrent, and chronic subtypes. *Acute MUPS* occurs in the absence of a previous pattern or history of MUPS and lasts a few months at most, and associated disability is often temporally associated with an acutely stressful life event. *Recurrent MUPS* is characterized by alternating symptomatic, asymptomatic, and mildly symptomatic periods. *Chronic MUPS* is a pattern of persistent unexplained physical symptoms associated with chronic disability, high health care utilization, and persistent problems with coping.

Empirically evaluated prognostic indicators for MUPS include (1) prior level of health care use, (2) psychiatric factors, (3) physical symptom factors, and (4) factors related to functioning. A high level of previous health care use suggests that a poor long-term outcome characterized by chronic MUPS is relatively likely.^{78,136} A large number of prospective studies have consistently found that the presence of stressors, distress, and psychiatric disorders, especially when they are chronic, predict persistent MUPS and related disability.^{9,13,22,29,57,65,105,109,119,126} A higher number of comorbid physical symptoms (“symptom count”)⁵³ and longer symptom duration^{13,22,89,95,148} also predict a poor outcome. Past poor functioning including occupational functioning suggests a poor prognosis.^{37,50,94} A patient’s historical level of functioning can serve as a marker for a myriad of issues that diminish the amount of reserve that an individual can muster when symptoms worsen.

PREVENTION OF SYMPTOMS AND SYMPTOM-BASED DISORDERS

The epidemiology of MUPS suggests that those individuals afflicted with the mysterious “Gulf War Syndrome” may represent only the most disabled, symptomatic, and distressed of ill Gulf War veterans. For each veteran who seeks care for Gulf War-related health concerns, there may be several others with fewer physical symptoms. In a less protean manner, perhaps, these individuals’ symptoms are reducing their capacity to function, increasing their use of health care, and heightening their health-related worries. Left unmanaged, these milder syndromes may become subject to the adverse influences of the previously described predisposing, precipitating, and perpetuating factors.

Is it possible to prevent MUPS? Resources are limited, and the scope of the problem is wide. The success of any program of prevention will depend on the degree of effectiveness of existing interventions and the resources required to deliver them. It may be feasible to significantly reduce the organizational impact of MUPS among military personnel by using a coordinated combination of population-based and need-based strategies. We recommend the adoption of a “population-based health care” model that uses a stepped-care approach (Figure A-1) to achieve maximum overall efficiency and effectiveness.

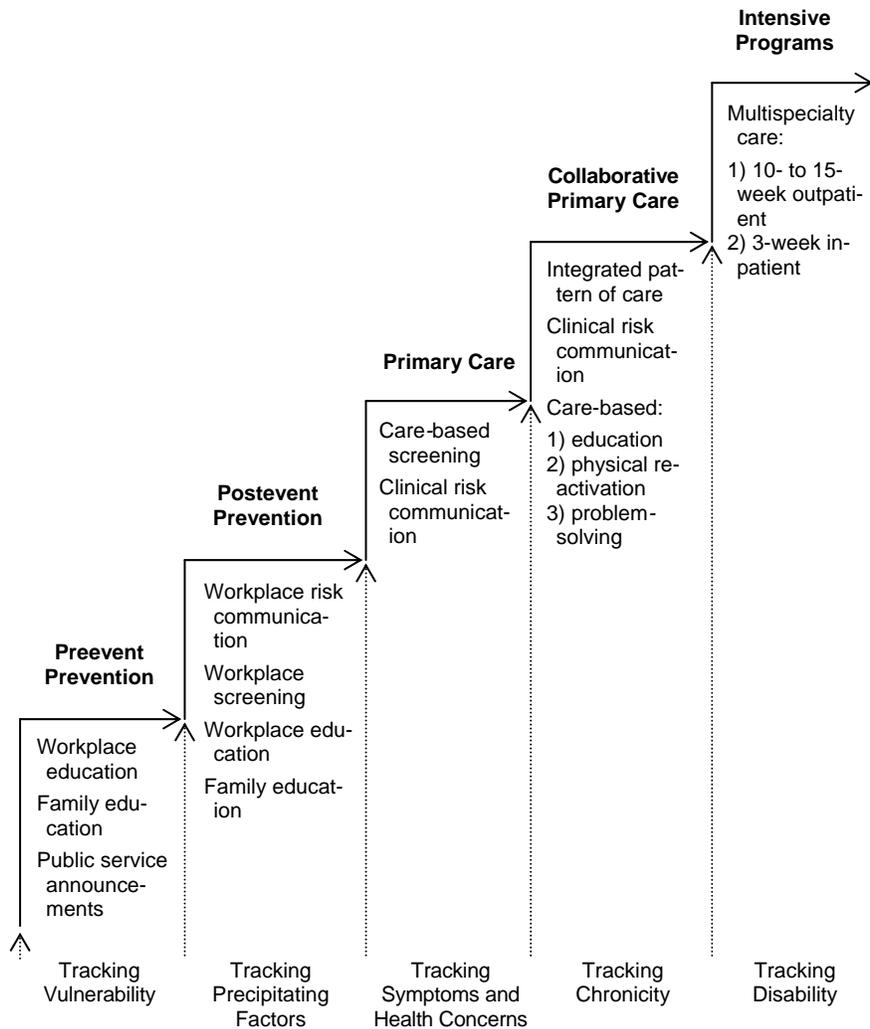


FIGURE A-1 A stepped-care approach to the population management of medically unexplained physical symptoms.

Advantages of Population-Based Intervention

Rose¹²³ has noted, “a large number of people exposed to a small risk may generate many more cases than a small number exposed to a high risk” (p. 24). Similarly, a large number of people exposed to a low-intensity preventive intervention can have a very large population effect (i.e., the effect of prevention summed across every person experiencing the intervention). Figure A-2 uses

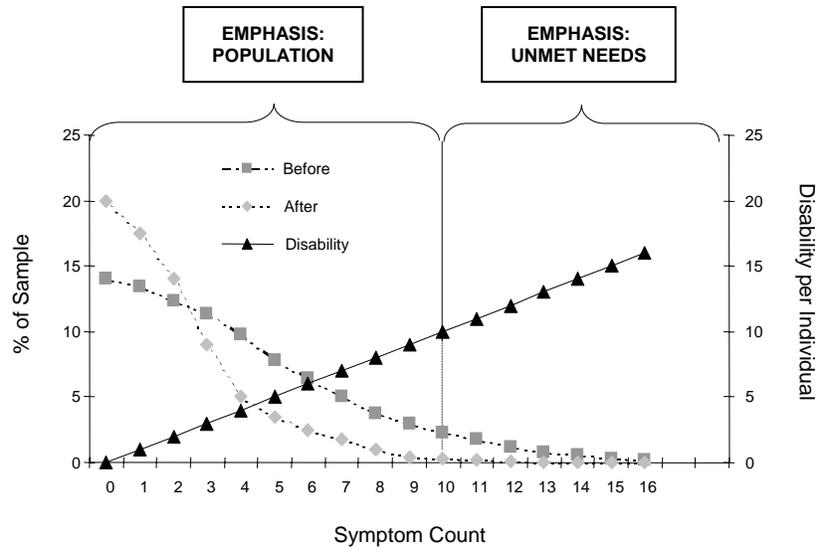


FIGURE A-2 Contrasting the population-based and needs-based approaches to reducing morbidity related to medically unexplained physical symptoms. Since disability (right vertical axis) is closely related to symptom count, population interventions that reduce symptoms a small amount per individual (“Before” = before intervention; “After” = after intervention) can prevent extensive disability when benefits are summed across the population. More intensive needs-based interventions can assist the relatively few individuals with repeated health care visits, multiple symptoms, and high levels of disability. Units of disability are imaginary and are hypothetical.

hypothetical data to illustrate that there is a graded and threshold-free relationship between symptom count and disability. Therefore, even among relatively healthy individuals, a small intervention benefit results in a small average individual improvement in functional status. Figure A-2 also shows that most of the population experiences relatively few symptoms and consequently little disability related to MUPS. When small reductions in individual disability occur across an entire population, the resulting societal benefits may be large and meaningful.

For the majority of people, MUPS come and go, usually without so much as a physician consultation. If these people are encouraged to seek health care for MUPS, it may increase the chance of long-term disability. This increase in disability may occur via mechanisms such as unnecessary worry, unnecessary avoidance of physical and social activities, unnecessary treatment, adverse effects of treatment, and provider errors.⁴² “Medicalization” of otherwise minor and transient symptoms may also occur. This is a process similar to labeling, wherein the act of visiting a doctor for a symptom imbues the symptom with catastrophic meaning, thereby setting up a self-fulfilling expectation of future disability.

In sum, population-based approaches to MUPS have the advantages of universal exposure to an intervention and summation of the benefit per individual across an entire population. Since many individuals who would never have become ill necessarily receive intervention, population-based interventions must have a lower potential for harm than most interventions employed for the sick.

Advantages of Need-Based Intervention

Interventions that target the whole population can seldom address the unmet needs of the important minority suffering from many symptoms and extensive disability. Rose¹²³ described health care-based preventive approaches as “the high risk strategy” because the effort is to identify individuals at especially high health risk or with especially great need for health care. The time-limited nature of clinical practice requires that providers rapidly recognize patients who require special attention. In essence, the clinician must identify and dichotomously delineate people lying along the continuum of disability severity as either ill or not ill. The point at which people are deemed ill is more or less arbitrary but necessary to operationalize so that the process of care can proceed unhindered. Using the hypothetical data from Figure A-2, for example, the “cutoff point” for identification of individuals in need of clinical care is set at 10 symptoms.

This artificial dichotomy leads to the specific advantages and disadvantages of health care-based prevention strategies. The primary advantage is that intervention can be matched to the unique needs of a relatively few seriously ill individuals, an approach that is attractive and sensible to both ill patients and their providers. Another advantage is that intervention aimed at the ill is minimally intrusive or harmful for those who are not ill. Riskier, more intensive, or more invasive interventions may be justified for “high risk” or ill individuals because of the comparatively large potential for individual benefit and the reduced societal cost conferred by limiting the intervention to a few.

On the other hand, clinical strategies contribute disappointingly little to any overall reduction of population disability. This is because only a very small proportion of society is ever exposed to a clinically based intervention that targets an ill or needy population. For example, Figure A-2 suggests that relatively few individuals have 10 or more symptoms, and many who have fewer than 10 symptoms will manifest significant disability and unmet needs that would not be addressed by a clinical intervention.

In sum, the population-based and need-based prevention approaches both offer important advantages and suffer from unique limitations. The best approach to the prevention of MUPS therefore involves some combination of population-based and need-based prevention, intervention, and management.

Population-Based Care: Matching Resources to Needs

Population-based care aims to improve health outcomes through carefully structured clinical services linked through primary care to a population-based prevention plan. Population-based care is the development and implementation of a detailed plan that covers all people in a defined population who, despite population-based prevention, have developed a chronic or recurrent health condition or concern. Important symptoms are identified, a mechanism to track outcomes is devised, and a deliberate matching of appropriate resources to patients with unmet needs occurs.¹⁵¹

Katon and colleagues⁸¹ have described how population-based care can reduce the prevalence of depression, and we advocate an analogous approach for MUPS. Critical is an understanding that various health care settings see different clinical populations with contrasting levels of MUPS severity and duration. More severely ill populations are encountered as the setting shifts from the community into higher levels of health care (e.g., tertiary care and inpatient hospital).

This is clearer when one considers the dynamics of illness in populations. Consider that the point prevalence (P) of some illness (i) is roughly equal to its incidence (I_i) times its average duration (D_i): $P_i \cong I_i * D_i$.¹²⁵ For intermittently relapsing illnesses such as MUPS, the duration of symptomatic illness can be approximated as the number of symptom episodes (N) times the average duration per symptom episode (D_e). Given some assumptions (beyond the scope of this discussion), the following can be shown:

$$P_i \cong I_i * D_e * N_i$$

This equation predicts that groups with more frequently episodic MUPS or MUPS of longer episode duration are overrepresented in populations because these characteristics elevate prevalence. The incidence of brief, nonrecurrent MUPS (e.g., acute back pain with a rapid resolution) may be relatively high compared with that of chronic MUPS. Even so, the long symptom duration and large number of episodes among those few individuals with an incident case of MUPS who develop chronic MUPS ensure that those with chronic MUPS are disproportionately represented in the population at any point in time. This overrepresentation of those with chronic and recurrent MUPS versus those with brief and acute MUPS is greater in specialty care than primary care and greater in referral facilities than local facilities. This occurs because local care and lower-intensity levels of care serve to “filter out” healthy and transiently ill individuals. Hence, the prevalence of chronic and recurrent illness is least in the general population, the greatest in specialty and tertiary referral settings, and intermediate in local and primary care settings.

The equation presented above suggests that the societal or organizational burden of MUPS may be reduced in at least three ways:

- incidence reduction or prevention of illness onset (primary prevention),

- duration reduction (secondary prevention), and
- relapse prevention (secondary prevention).

A fourth method of MUPS prevention (tertiary) targets the important morbid consequences of chronic MUPS: psychosocial distress, psychiatric disorders, and disability. From the equation, we would expect that the first three strategies might reduce the population prevalence of MUPS. The fourth approach may not alter the prevalence of MUPS but may still reduce the population burden of MUPS.

Implementing and Improving Population-Based Care

Wagner and coworkers¹⁵¹ have described how to implement and improve population-based care. They describe three distinct organizational thrusts: information systems, practice design, and patient education.

Information Systems

Information systems (ISs) are computer-based systems used to capture data that can be used to inform clinicians regarding patient status, assist clinicians and medical executives interested in monitoring and improving the quality of care, and guide policy makers attempting to assess population needs and determine appropriate staffing levels. An IS for MUPS should use three components: a *health information system* (HIS) (a passive automated health surveillance system), a survey-based *health monitoring system* (HMS) (an active health surveillance system), and *expert computer systems* (ESs) (automated systems that generate useful reports for the identification of high-risk patients and evaluation of care, population health status, and clinical outcomes).

The schematic in Figure A-3 shows the interrelationship of IS components to various tools that may enhance the population-based care of MUPS. The HIS can record medical problem lists and measures of health care utilization (outpatient, inpatient, and pharmacy services and various procedures), health care costs, and presenting symptoms. These data, combined with HMS-based data on patient-reported physical symptoms, may be used to define MUPS for tracking purposes and to identify high-, intermediate-, and low-risk groups. Katon and colleagues⁸¹ have suggested that the following elements are integral to any HIS that supports evidence-based interventions within a population-based health care system:

- regularly updated information on patients' primary care physician, place of care, and other contact information;
- current information on health care use including medication fills, procedures, laboratory results, primary care visits, and specialty care visits;
- a prioritized medical problem list; and

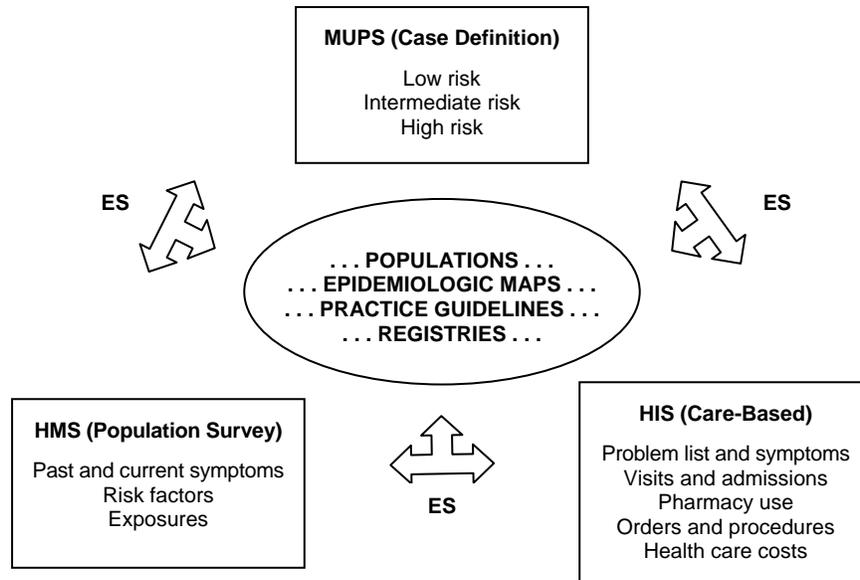


FIGURE A-3 Components of an IS used in population-based health care for MUPS and their relationship to one another. ES = expert computer systems, HIS = health information system, and HMS = health monitoring system

- other information relevant to establishing condition-specific patient registries for tracking and monitoring.⁸¹

The IS uses ESs to process raw data obtained with HMS and HIS, prepare these data for various uses, and deliver cleaned and collated data to appropriate users. ESs are programmed to generate tools that aid clinical management, patient follow-up, and treatment and policy decisions. Examples of ES tools include reports, reminders, clinical indicators, feedback systems, and guideline recommendations. ESs may be used to create registries, identify from a practice team panel patients who are likely to meet case criteria or who require intervention, monitor outcomes, compare outcomes for individual patients to those for groups of similar patients, and track the progress and relative prognosis of particular high-risk patients. An appropriate ES for MUPS might identify high-risk MUPS patients (for example, those with frequent visits or certain diagnostic codes from the International Classification of Diseases), remind clinicians of applicable guidelines and algorithms, identify relevant patient and family education tools, and implement screening scales or standard questions for consistent outcomes monitoring.⁸¹ Eventually, it will become possible to compare the relative impact of primary care, specialty care, and quality of care on MUPS outcomes.

In the future, linking of the HIS and HMS with *administrative information systems* (AISs) (e.g., military personnel files containing dates of promotion, disci-

plinary actions, awards, deployments, and evaluations of performance) may allow careful empirical evaluation of whether risk factors and interventions alter militarily relevant MUPS outcomes. The combined use of ESs, HISs, HMSs, and AISs may provide for careful longitudinal tracking of the health status of individuals with MUPS who have recently deployed. Eventually, extensive empirical experience and understanding regarding the course of MUPS after deployments may be gained. IS data may be used to create population-based case registries and epidemiological maps showing the population distribution of people meeting case criteria. These individuals may be tracked for outcomes of potential interest such as long-term health care costs and service utilization, absenteeism, activity limitations recorded on military medical profiles, length of military career, rates of active duty reenlistment, promotion rates, and misconduct rates. Over time, refinements may be made to the existing case definition of MUPS so that it identifies individuals and groups at low, intermediate, and high risk of poor outcomes from MUPS. These data may also inform efforts to generate, implement, and evaluate pertinent clinical practice guidelines and best clinical practices.

Practice Design

Many have argued that the biggest barrier to quality clinical practice is the manner in which medical care is delivered.¹⁶¹ Ambulatory care involves patients seeking care for a myriad of poorly understood psychosocial and medical reasons. In the traditional acute care approach, a physician quickly narrows to an often oversimplified “chief complaint,” assesses only the most urgent medical needs, and then triages the patient to an appropriate level of care. Physicians managing acute medical problems are seldom practiced, skilled, or inclined to deliver preventive behavioral measures (e.g., dietary counseling, smoking cessation, and exercise prescription).

This approach fails to address the broad and often behaviorally based needs of people with chronic health conditions like MUPS. These individuals require systematic assessments, effective and targeted education, and sustained psychosocial support and follow-up aimed at maximizing long-term health and well-being. Their medical status may not become life threatening or severe enough to require acute medical attention until late in life or course of illness. By then, the opportunity to provide effective preventive measures has largely been lost.

The following are other barriers to the primary care management of MUPS:

- time restrictions and patient defensiveness;¹¹³
- high level of concern and low level of patient trust of military health care providers potentially responding to an organizational allegiance when caring for patients with MUPS after a deployment;
- reimbursement approaches that favor the use of invasive medical procedures over more behaviorally oriented rehabilitative care;³³

- clinician perceptions of MUPS patients as frustrating, noncompliant, and undesirable;^{61,97,112,154}
- inadequate coordination of care between primary and specialty care;¹¹³
- excessive reliance on physicians as the primary clinical facilitators of medical and behavioral change;
- disproportionate physician and media interest in disease-centered care featuring new technologies rather than patient-centered care stressing health behavior change; and
- an unwillingness or inability on the part of physicians to delegate crucial behavioral and educational aspects of the patient encounter that are best addressed by clinicians from nonmedical disciplines (e.g., nurses, psychologists, social workers, nutritionists, exercise physiologists, physical therapists).¹⁵¹

Improving primary care management of patients with MUPS requires far-reaching alterations in the culture, incentives, structure, and process of medical care as it is currently delivered. Given the demands on primary care, it seems unrealistic to expect that primary care physicians alone will comprehensively and intensively meet the diverse medical, educational, behavioral, and psychosocial needs of all MUPS patients. A more achievable goal is to develop a proximate, structured, collaborative, interdisciplinary, and multimodal process of primary care capable of reducing the burden of MUPS on primary care physicians. If primary care physicians can achieve success within the context of a reorganized clinic process, they may eventually find that behavioral management of MUPS and related distress and disability is rewarding and worthwhile.

Therefore, we recommend the development, implementation, and use of structured and carefully monitored health care programs that use *primary care practice teams*. Practice teams employ a wide range of nonphysician and physician providers collaborating together in a coordinated process of care. The team meets regularly to improve clinical coordination and intensify care-based efforts to inform patients about MUPS, prevent relapse of MUPS, increase physical activation, improve treatment adherence, respond to patient support needs, and hasten return to work.

Patient Education and Clinical Risk Communication

The range of patient education options is rapidly expanding. Carefully designed patient education materials are particularly important for those experiencing MUPS after deployments. Appropriate education materials can address harmful illness beliefs, the health effects of individual deployments, self-help strategies, the importance of managing disability and distress, the risks and limitations of extended diagnostic testing in “low-yield” clinical situations, and the ubiquitous nature of MUPS. Modalities available for disseminating patient information include brochures, mailings, books, videotapes, audiotapes, and waiting-room computers using self-guided learning approaches, as well as Internet-

based learning technologies. Nonphysician specialists trained in patient education strategies and information technologies may assist patients with their questions in a manner that fosters trust and reduces distress regarding unlikely causes of symptoms. They may help patients troubleshoot attempts to initiate regimens of regular physical activity, take their medicines regularly, and so on.

Health risk communication is a discipline that addresses methods of enhancing bilateral communication in “low-trust, high-concern” situations. We have already described the insidious impact of the physician’s competing and frequently unacknowledged obligation to the employer on the provider-patient encounter in occupational and military medicine. To date, risk communications experts have focused primarily on community-based methods of disseminating information and keeping communication constructive. However, risk communication approaches may be modified and applied to the low-trust, high-concern clinical encounter that occurs in occupational and military medicine settings. Risk communication imperatives are to carefully design and empirically test the impact of health risk messages. In clinical settings, we might ask: (1) Does a particular waiting room brochure foster patient trust in their physician? (2) Is there a way to restructure the clinical encounter that enhances communication between providers and patients under these tense situations? (3) What is the most effective way for a military physician to tell someone postdeployment that the person’s symptoms are medically unexplained without fostering fear of a progressive illness due to some poorly understood military-related toxic exposure? *Clinical risk communication* might be defined as the application of health risk communication approaches in the interest of enhancing the overall effectiveness of occupational, military, and analogous medical encounters.

Stepped-Care Approach to Population MUPS Management

A critical focus of population-based care involves matching intervention intensity to the severity, duration, disability, and psychosocial needs of patients. The stepped administration of specific interventions (i.e., administration from least to most intensive) ensures that the individuals with the greatest need receive the most intensive and costly treatments. Figure A-1 summarizes the stepped approach that we currently envision. It employs five basic steps: preevent prevention, postevent prevention, routine primary care, collaborative primary care, and intensive multidisciplinary care. Note that a high level of clinical certainty and rigorous empirical evidence is not required to initiate this care model. The approach that we describe may be and should be incrementally updated and revised as necessary research is completed.

Step One: Preevent Primary Prevention

Currently, the primary prevention of MUPS is poorly understood, and resource-intensive attempts to implement unproven primary prevention strategies seem premature and unnecessarily costly. Nonetheless, populationwide primary preventive efforts to prevent the onset of MUPS as well as associated distress and disability are deserving of further attention and research. For example, “step one” approaches such as organizational policies and regulations or community- or workplace-level education involving literature, television, or other media segments require study and may have significant value. Unfortunately, the effectiveness of such efforts for MUPS is anecdotal and largely unknown. The routine administration of high-intensity step one prevention is likely to overextend costly resources to the majority of individuals who will never develop health concerns, making feasibility a major concern. Therefore, large resource expenditures may be difficult for policy makers to justify in the absence of experimental evidence supporting the efficacy of preevent prevention.

One promising primary prevention modality is education and related programs. For example, Symonds and colleagues¹⁴³ found that a low-intensity workplace intervention for back pain prevented subsequent sick leave. The intervention involved reattribution of back pain by use of an educational program. Pamphlets were distributed to all workers regardless of back pain history. The pamphlet highlighted the benign nature of low back pain and the importance of activity maintenance and early return to work as ways to successfully reduce morbidity. The investigators also found the program shifted worker beliefs about the causes of back pain. Similarly, military personnel, their families and significant others, their leaders, and health care personnel may benefit from brief, simple, education-oriented efforts that provide appropriate information regarding MUPS and their relationship to distress and treatable psychiatric disorders.

One potential way of narrowing the scope, increasing the feasibility, and reducing the cost of intensified step one prevention is to inform them by using IS technology. For example, smaller groups with predisposing MUPS factors may respond to a targeted intervention. ISs may help narrow the focus of intensified efforts to mitigate the impacts of these factors on subsequent development of MUPS and related morbidity.

Step Two: Postevent Primary Prevention

We suggest narrowing the focus of postevent prevention to specific units and associated families that have recently deployed or faced other events that might precipitate subsequent health concerns. Within these units smaller groups at especially elevated risk of MUPS may be identified on the basis of the presence or absence of past MUPS or other predisposing factors. The “real-time” availability of IS data has the potential to focus preventive efforts at identified points of organizational vulnerability.

Several candidates for postevent preventive efforts deserve further attention and evaluation. Workplace-based briefings may teach recently deployed personnel the associated possible or known health risks. Leadership efforts to normalize the workplace through an early return to work routines and previously scheduled activities may maximize postevent productivity. A feeling of chaos and loss of control are common immediately after a tactical deployment or a catastrophic event. A rapid return to routines may provide personnel with a familiar and predictable environment and a feeling of productivity. The availability of support meetings and meetings open to some larger community (so-called town hall meetings) may provide a forum for military and community leaders to learn of event-related community and family concerns. Similarly, town hall meetings offer opportunities for personnel and significant others to articulate and even ventilate important event-related health concerns. If the event or deployment involved sufficiently large numbers, telephone hot lines may be useful, too, providing personalized contact for people with questions, concerns, or previously undiscovered events or exposures.

A large anecdotal literature often promotes large-scale postevent debriefings. However, randomized trials of critical incident debriefings (CIDs) have shown limited efficacy, and at least one study has suggested that CIDs may actually increase the risk of postevent psychological distress.¹⁵⁹ A CID uses a structured debriefing format often led by mental health professionals with various levels of experience and expertise. Those exposed to the “critical incident” are encouraged to review the event in detail, focusing on current emotions and emotions during the incident. Efforts are made to inform people of the signs and symptoms of psychological trauma. CID is difficult and costly to successfully implement on any wide scale, may set up self-fulfilling expectations of subsequent psychological symptoms and disability, and is empirically unsupported from the experimental trials completed to date.

As in step one, caution is necessary when considering relatively high-intensity preventive measures for people who have yet to develop MUPS. A commonly considered step two approach is populationwide postevent screening. These efforts may positively reinforce or “medicalize” what are otherwise normal transient symptoms following such events. Even given IS data regarding predisposing and precipitating factors, it may be difficult to accurately predict who will develop MUPS and even harder to know who among individuals with MUPS will then develop disability and distress. Singling high-risk individuals out for a psychosocial intervention before the onset of symptoms and disability may unnecessarily and unfairly stigmatize or prematurely label many individuals. Most of those labeled immediately postdeployment will not develop symptoms or their symptoms will be time limited. Therefore, primary care-based screening for MUPS, tracking of outcomes of MUPS, and intensification of treatment for those with suboptimal outcomes is the most practical and least costly approach.

Step Three: Routine Primary Care

As noted, feasible primary prevention strategies for MUPS are, unfortunately, of a low intensity; therefore, we can expect that new cases of MUPS will regularly occur even after relatively successful population-based prevention programs. Virtually all individuals with MUPS will encounter primary health care. Therefore, a key to secondary prevention may involve early primary care recognition and timely management of MUPS to reduce the impact of precipitating and perpetuating factors on physical symptoms, emotional distress, and disability. IS technologies may remind primary care physicians which of their patients are most symptomatic, most concerned about their health, and most distressed regarding undiagnosed illness. Once these patients are identified, there are several ways that clinicians may mitigate the impacts of precipitating and perpetuating factors in an effort to prevent a chronic course. These are now reviewed.

Routine Primary Care Physician Management *First, do no harm.* Most patients with MUPS have had extensive diagnostic evaluations. Often, clinicians are aware at the time of initial history and physical that diagnostic testing offers a low yield or that anxiety or depression are important exacerbating factors. Studies suggest, however, that for patients with MUPS, clinical awareness is not well integrated into physicians' diagnostic and treatment practices.⁸ As we have described, "shotgun" diagnostic testing under these circumstances can be harmful. Ordering unnecessary tests sends the wrong message to patients and promotes a passive patient mindset (e.g., "the doctor's in charge" and will "find it and fix it") that is counter to achieving behavioral activation goals and shifting some responsibility for wellness to the patient. Physicians are notoriously poor at making patients aware of the tests that they order, the rationale for ordering them, and the eventual results. One alternative to running new tests is for doctor and patient to carefully review past testing together, an approach that promotes clinician-patient collaboration and patient understanding. Sometimes, however, new diagnostic testing is necessary. A good rule of thumb for testing in patients with MUPS is to test only for classic constellations of symptoms or new objective signs.

Clinicians must take care not to present medications as a substitute for person-centered care for MUPS aimed at addressing health concerns and reducing disability. Although medical explanations for physical symptoms are often lacking, physicians often still place the patient on medications, even though medications are a relatively small part of the overall management of MUPS and unintended adverse effects often outweigh medication benefits. Sedatives are usually inappropriate unless insomnia is acute, stress related, and expected to abate within a short time. Narcotic analgesics usually do more harm than good, since they slow thinking, cause sedation, and reduce overall functioning. Both of these medication groups usually have adverse impacts on efforts to activate patients. Chronic administration of other central nervous system depressants such as so-called muscle relaxants is unadvised for similar reasons. Antidepressants, how-

ever, reduce the occurrence of MUPS among patients with chronic pain, panic disorder, dysthymic disorder, and major depressive disorder. In addition, reductions in depression and anxiety are critical to behavioral activation. It is important to carefully explain the rationale for antidepressants, or else patients will assume they were prescribed because the doctor thinks that the symptoms are “in the head,” causing the patients to discontinue the medicine or see another doctor. All patients with MUPS should receive a complete and careful explanation of medication side effects, so that if they occur the clinician’s credibility is enhanced and the chances of continued adherence is maximized.

Cure rarely; comfort always. Seldom is it possible to cure any chronic illness, and MUPS are no exception. Setting symptom eradication as a treatment goal will only lead to clinician and patient dissatisfaction. Clinicians intent on cures often feel as though they have nothing to offer patients with MUPS. They may devalue their role with patients with MUPS as “doing nothing” or “hand-holding.” The importance of a supportive, empathic, and person-centered (rather than disease-centered) approach cannot be overemphasized.

Comforting patients with MUPS often entails reassurance. This means more than simply telling them that their symptoms are not serious. It involves elucidating harmful illness beliefs and directing education and advice to those beliefs. The following are common examples of harmful beliefs:

- “My symptoms are a sign of disease.”
- “When I hurt it means I am seriously injuring myself” (e.g., “pinching a nerve”).
- “When I have symptoms I can’t make it without rest and a break from my responsibilities.”

Clinicians can also learn the phrases that people with MUPS find belittling and avoid them. Similarly, they can learn some phrases that “join” the clinician and patient in a collaborative dialogue. For example, most individuals with MUPS describe their distress as secondary to symptoms. Although research is clear that distress increases the risk of subsequent physical symptoms and vice versa, it is best to adopt the patient’s words and views regarding causation, no matter how faulty the clinician may think they are. Patients understandably react negatively to physician statements such as, “There’s nothing physiologically wrong.” Perhaps most physicians suffer from a good deal of overconfidence in their own clinical conclusions and would benefit from allowing their patients to have more input than they currently do.

Comforting involves office-based patient education and often centers on the health effects of adverse life events and toxic exposures, the impacts of anxiety and mood on physiology, symptoms, and functioning, the limits of medical testing, and the impacts of medication side effects on functioning. Self-help materials such as audiotapes and books about physical activation, relaxation tech-

niques, and coping with chronic pain and similar symptom-based disorders are widely available.

Negotiate behavioral goals targeting illness and disability. Reducing disability requires specific changes in patient behavior. It requires patients to take an active, collaborative role in their treatment rather than a more traditional passive role (“fix me doc”). Provider-patient collaboration and negotiation of behavioral goals will usually prove to be more rewarding than striving for an elusive cure. Goals must be specific, incremental, realistic, and achievable, and they should center on observable or reportable behaviors. First and foremost, goals must be negotiated with the patient such that the patient “owns” the goals. If goals are simply clinician imposed, the patient may have no investment in them, view them as impossible, or covertly oppose them. It is often useful to have patients graph their incremental progress toward their goals and review the graphs with them at their follow-up appointments. Examples of good areas for goal formulation are occupational, household, or social task performance, physical activation, sleep hygiene, or medication adherence.

Hold the patient responsible for change, but avoid “the blame game.” In disease-centered care, the patient is a passive participant. The patient is to “comply” with the doctor’s “orders.” The patient visits the doctor in search of answers, and the doctor is responsible for providing them. In person-centered care, the clinician must move out of the “answer man” role and join with the patient as a facilitator of behavioral change. The clinician negotiates the goals of treatment with the patient, helps him or her solve the problems “they” encounter, and carefully addresses the patient’s expectations for quick or magical solutions. Simply acting as an “idea generator” for the obstacles that patients describe helps to facilitate behavioral gains. Clinicians must shift the responsibility for change to the patient, but they must also remain vigilant not to blame the patient for their lack of progress or their illness predicament.

Encourage physical and role reactivation. Regular exercise in tolerable doses helps patients with MUPS discharge distress, increase stamina, and improve functioning. Physical therapy programs of gradually increasing physical activity are sometimes useful for overcoming the deactivation and weight gain that occurs for many patients with MUPS. Usually, a physical therapist is not necessary to initiate reactivation strategies; these can be negotiated in the physician’s office. Similarly, patients need encouragement to remain gainfully employed and active in supportive relationship roles. This reduces dependence and improves morale, self-confidence, and ability to meet expectations. In most occupational settings and especially in the military, reactivation strategies require careful coordination with employers or supervisors. The best reactivation plan will go awry if workplace supervisors are unaware of it or do not support it.

Involve social supports. Social supports may include family or close friends. Clinicians should encourage participation of support systems in nearly all aspects of care, provided that the patient approves of this. Family or friends can help clarify concerns, illness beliefs, symptoms, and deficits in functioning. Often, the patient's most important concerns are related to those closest to the patient, and their involvement in care can make or break the clinician's ability to successfully engage the patient in a constructive dialogue about the patient's health concerns. In occupational settings, the extent of involvement of the supervisor or employer must be similarly considered. "Collaboration" with the employer should seldom occur without the expressed (and usually written) permission and direct involvement of the patient. If organizational conditions, rules, or regulations pertain to employer or supervisor involvement, these should be clear and available to the patient from the time of the initial clinical contact or whenever it becomes apparent to either the patient or the clinician that employer involvement may occur.

Coordinate care with one designated clinician. Proper management of the delivery of care is both cost-effective and in the best interest of the patient. This is especially important for patients with many MUPS and those with chronic symptoms. In the absence of well-coordinated and centralized care, patients with multiple MUPS are likely to bounce from specialist to specialist, receive many unnecessary diagnostic procedures, and end up on multiple unnecessary medications. The key elements of coordinated care include (1) establishment of a relationship with a single primary care provider, (2) appointments at regular, time-contingent intervals of about every 4 to 6 weeks, (3) a brief physical examination at each visit to address new physical concerns, and (4) limits on patient-initiated visits for an exacerbation of otherwise chronic symptoms. Whenever possible negotiate an advance plan as to how symptom-contingent visits will be handled. If it is anticipated that this may become a problem, it is often sensible for clinician and patient to negotiate a written plan that both can refer to if limits become necessary. Some patients may fear that these limits mean that the doctor is angry with them or going to reject them. If the plan was previously negotiated and drafted in writing, these patient concerns may be tactfully addressed when they arise with minimal damage to the doctor-patient relationship. Consultants to the primary care physician must understand that they are to recommend care rather than assume it. Similarly, primary care clinicians should present consultants with a focused question. Consultants must understand their role and the key aspects of caring for patients with MUPS.

Anticipated and judicious mental health care referral. Psychiatric referral is frequently appropriate for those with MUPS, especially for patients who request it, have suffered a recent stressor, have a treatment-refractory psychiatric disorder, or describe suicidal or other clinically worrisome issues. However, most patients with MUPS do not require psychiatric treatment or psychological testing. Evidence suggests that a surprisingly large proportion of patients with

MUPS receive mental health referrals without an adequate explanation as to why they are needed.⁸⁶ In some cases, there is little doubt that a clinician desires primarily to “turf” (i.e., reject) a difficult patient. Not surprisingly, this message is seldom lost on the patient. Clinicians should not wait until the entire biomedical evaluation is complete and then obtain a referral because “potential medical causes are ‘ruled out’ and therefore the patient needs a psychiatrist.” To prevent patients from experiencing mental health referral as rejection, it is usually best for clinicians to anticipate the potential need and introduce it early in a non-threatening way. Patients are best told that a frequent consequence of MUPS is disabling distress and that appropriate care can mitigate the impacts of their symptoms on their quality of life. It is important that primary care clinicians see patients after completion of the mental health referral to reduce the patient concerns that the doctor is rejecting or abandoning them. Primary care clinicians should ask patients how they experienced the consultation and contact the consultant directly for recommendations if possible.

Unfortunately, most mental health professionals have only infrequent exposure to patients with MUPS, are not skilled in their management, and do not readily appreciate the need to collaborate closely with primary care. Even when done under ideal conditions, less than half of referred patients ever obtain mental health evaluation. Patient defensiveness, excessive rejection fears, and social stigma associated with having a psychiatric disorder are among the significant obstacles to effective mental health consultation for patients with MUPS.¹¹³

Clinicians often obtain psychological tests such as the Minnesota Multiphasic Personality Inventory with the expectation that it will provide them with hard-and-fast evidence that MUPS are psychological rather than physical in origin. These tests can offer information regarding the relative style, quality, and success of patient coping and distress. However, they are not effective for diagnosing a psychological etiology for physical symptoms. Extensive psychological testing is not a panacea and may be quite threatening to patients when administered under any clinical circumstance, especially when the assessment may have occupational or military ramifications.

Teaching MUPS Management to Primary Care Physicians One reason that physicians minimize the importance of MUPS is their lack of awareness of and comfort with appropriate management strategies. Naturally, they focus on things they know how to treat, and most think there is nothing they can do about MUPS. It is important to enable them through proper educational experiences that focus on the basic primary care strategies described earlier.

MUPS-related clinical training experiences may add to the overall quality of patient care by improving the routine primary care management of associated, frequently unrecognized, and treatable psychiatric disorders. Research suggests that an excessively biomedical approach to MUPS or coexisting chronic medical illness markedly diminishes physician attention to psychosocial aspects of care such as recognition of treatable anxiety and depressive disorders. Kirmayer and Robbins⁸⁴ studied 685 patients presenting to a primary care clinic and found that

approximately three-fourths of those with major depression or anxiety disorders complained exclusively of physical symptoms. Studies have shown that mentally ill patients with emotional complaints are usually detected, whereas those with only physical complaints are generally missed.⁵¹

Providers in medical settings may sometimes collude with patients in ways that undermine effective health care. For example, the provider may detect mental illness in a patient but fail to offer treatment because he or she senses that the patient might be unreceptive. Some clinicians are better than others at identifying treatable psychiatric disorders in their patients.¹⁰¹ Conversely, distressed patients will more readily share their emotional concerns with those clinicians who are best at addressing them.⁵¹ Appropriate medical education emphasizing communication skills, MUPS, and the recognition and treatment of anxiety and depressive disorders by primary care providers may improve clinical outcomes and provider confidence in addressing patients' psychosocial issues.

Efforts to improve physicians' communication skills are critical to improving the routine primary care management of MUPS. Too often clinicians fail to acknowledge to themselves and to their patients the high degree of uncertainty inherent in all clinical practice, perhaps especially for those patients in whom no explanation is found for physical symptoms. Clinicians must learn and relearn that the "absence of an explanation" is not synonymous with a "psychological explanation." A fundamental tenet in the art of caring for MUPS is to acknowledge the centrality of aversive symptoms to the patient's life before asking the patient to take responsibility for overcoming those symptoms. Often physicians admonish their patients to actively seek a state of health, and some even equip their patients with tools for seeking that health. However, unless they first validate, empathize, and even immerse themselves in the patient's physical symptoms and their sense of personal damage, sacrifice, and suffering, most patients will feel misunderstood. Some will feel that the physician is blaming them for their illness. A few patients will experience an unspoken challenge, the challenge to prove the reality of their suffering. In short, physicians must make it their routine clinical mission to develop an appreciation for the extent that each patient constructs his or her life around symptoms, suffering, and limitations, whether or not medical explanations are available. For example, Marple and colleagues¹⁰² found that when physicians addressed patients' health worries and fears and understood the rationale behind their fears, their physical symptoms and functioning improved faster and the patient was more satisfied with care.

Physicians must develop strategies and experience explaining the limits of diagnostic testing and clinical treatments to their patients. Gallagher and co-workers⁴⁹ illustrated this in a recent study. Those investigators explored 39 internists' responses to a patient request for an expensive, unindicated diagnostic test. An actor was used to play out a standardized and blinded clinical scenario. Participating internists practiced in a health maintenance organization, and each encountered a young woman presenting with only chronic fatigue and no neurological symptoms. The patient desired magnetic resonance imaging (MRI) to rule out multiple sclerosis because of a friend's recent experience with the dis-

ease. Only 10 percent of internists asked about the friend's illness, but 8 percent ordered the MRI and 22 percent said they might in the future. Fifty-three percent referred the patient for a neurology consultation on the day of the visit, and all but 13 percent of internists said they might refer the patient in the future.⁴⁹ This study is but one of many that illustrate the need for greater clinician education regarding strategies for addressing patients who press for unnecessary diagnostic testing or treatments.

Step Four: Collaborative Interventions in Primary Care

To benefit patients, specialists and primary care providers need to learn and respect each others' ideas, share resources, and learn ways of successfully working together to develop consensus around common goals like the population-based care of patients with MUPS. Particularly important is the need to develop collaborative on-site programs of behavioral health care for primary care providers. Such programs can enhance patient adherence to behavioral approaches initiated in primary care. In addition, on-site consultation reduces stigma by presenting it as a routine part of the primary care experience rather than something mysterious and remote. On-site collaboration also provides primary care providers with satisfying opportunities to interface with and learn from specialists from the other disciplines rather than the more traditional approach of referring complex primary care patients to specialists "right when they get interesting."

Several groups have looked at primary care-based psychosocial interventions for persons with MUPS, distress, or both. Strategies have most commonly involved screening,¹¹⁴⁻¹¹⁶ physician and patient education,⁵ primary care-based mental health consultation,⁷² interdisciplinary treatment teams,¹⁰⁸ and psychotherapy techniques adapted for primary care use.²⁰ Smith and colleagues^{71,124,137} have found replicable reductions in the cost of care and even small improvements in health-related quality of life for patients with the most severe forms of MUPS (i.e., patients with somatization disorder) simply by sending a set of short, codified recommendations to patients' primary care providers advising them on how to manage them.

Katon and colleagues⁷⁹ completed a randomized trial of psychiatric consultation for "distressed high utilizers of primary care" at Group Health Cooperative of Puget Sound, a health maintenance organization serving over 350,000 enrollees in Washington State. Distressed high utilizers were defined as the top 10 percent of ambulatory care utilizers over the year prior to study who were identified as distressed either by their primary care physician or by high scores on a validated paper-and-pencil measure. This 10 percent of patients utilized approximately one-third of all outpatient visits, 26 percent of all prescriptions, and one-half of all inpatient hospital days. The intervention consisted of a structured psychiatric research interview followed by a 30-minute collaborative patient interview and treatment planning session involving the generalist, psychiatrist, and patient. Patients in the control group received usual primary care.

Improvements in mental status or service utilization of intervention patients over that of controls could not be demonstrated. In retrospect, the intensity of the intervention was low, perhaps serving notice that MUPS involve many complex factors that are not responsive to a brief, one-time intervention that targets mainly psychiatric disorders. Prescription practices were marginally better for the intervention group, but subsequent antidepressant regimen adherence was generally poor for patients in both groups. There was no formalized mechanism for interdisciplinary collaboration after the initial consultation and no way of subsequently enhancing primary care clinicians' effectiveness or their adherence to the original collaborative care plan.⁷⁷

More recently, primary care approaches to physically symptomatic and distressed primary care patients have focused on "multimodal" or "multifaceted" interventions. These are best administered in steps, so that the most intensive, expensive, or burdensome treatments are held in reserve for those who are otherwise treatment refractory. Components have included screening; on-site mental health consultation; cognitive-behavioral and problem-solving therapies aimed at medication adherence, depression, MUPS, physical activation, and relapse prevention; videotapes, pamphlets, and other education materials on self-care; structured follow-up strategies; and standardized written primary care instructions. Other efforts to enhance primary care clinicians' ability to tackle the multiple needs of their patients have employed "academic detailing," feedback to clinicians from their patients' automated pharmacy or health care utilization records, and case management.

Katon and colleagues^{75,80} used a multifaceted approach to assist depressed primary care patients, an approach that can serve as a model for similar primary care-based MUPS interventions. Elements of their intervention targeted the patient, the physician, and the process of health care delivery. Elements that targeted patients were reading materials on depression, antidepressants, simple self-administered cognitive-behavioral techniques for managing depression, and a videotape on similar topics for viewing with spouses. Elements that targeted primary care physicians were didactics on antidepressants and behavioral treatment of depression, case-based consultation for each depressed patient, and ongoing interaction and feedback between the psychologist and primary care physicians. Elements that targeted the process of care were extensive and manualized. These included behavioral therapy done in the primary care setting. Behavioral therapy aimed at teaching patients depression self-management skills, improving medication regimen adherence, and preventing future relapses. Psychologist contacts were scheduled and occurred in the primary care setting. These contacts involved skills training, education, and homework. Relaxation training, assertiveness training, problem-solving training, and collaborative psychologist-patient development of a relapse prevention plan were done. Additional telephone contacts with the psychologist occurred after completion of primary care-setting contacts. Symptom monitoring occurred by a standardized measure and a checklist. The psychologist screened and documented antidepressant side effects, dosing, and adherence. During weekly interdisciplinary team

meetings, a psychiatrist reviewed antidepressant-related information and overall treatment progress. The psychiatrist would advise medication alterations as indicated, and the psychologist communicated these recommendations to the primary care physician, who would carry them out. This integrated process of care was carefully monitored for integrity by using a numeric rating system. These integrity ratings were monitored and used to provide regular clinician feedback.

Katon and coworkers⁷⁵ compared this collaborative interdisciplinary intervention to usual care for depressed primary care patients using a randomized controlled design. As long as 4 months after completion of the intervention, intervention patients with major depression reported greater satisfaction with care, adherence to the medication regimen, and improvement in depressive symptoms than major depression patients receiving usual care. The results of the intervention were less clearly favorable among patients with minor depression (significantly improved antidepressant regimen adherence and perceived antidepressant helpfulness, but there were no significant differences between the groups regarding depression symptoms or satisfaction with depression care).⁷⁵ Other analyses of these data have found evidence of improvements in physical symptoms. Analyses of cost-effectiveness found that the intervention was more costly than usual care for patients with both major and minor depression. However, for the major depression patients, the multifaceted intervention offered significantly greater cost-effectiveness than usual primary care.¹⁴⁹

Given the added expense associated with collaborative models, we suggest that they be held in reserve for patients for whom routine primary care management strategies for MUPS fail. Symptom duration is a key step four indicator to monitor using IS-generated reports. When a patient's symptoms reach some threshold of extended duration, more intensive collaborative efforts may be proactively introduced.

Step Five: Specialized Intensive Multimodal Care

There are several excellent examples on which to model tertiary prevention programs for patients with MUPS who fail to improve in response to collaborative primary care approaches. These programs are multimodal and multidisciplinary, occur in specialized (i.e., non-primary care) settings, and involve either a 3- to 4-week inpatient or intensive outpatient program or a 10- to 15-week program of weekly or biweekly individual or group visits. These programs emphasize carefully planned psychosocial elements that address the chronic nature of reduced functioning and the factors that reinforce it.

Usually, psychosocial and medical care is combined with a highly structured and generally supervised physical activation or exercise plan. These programs view disability as a behavior amenable to modification regardless of its biomedical etiology. Engel and colleagues³⁶ have described such a program for veterans with MUPS after service in the Gulf War. The intervention, called the Specialized Care Program (SCP), is a 3-week intensive outpatient program

modeled directly after the University of Washington's Multidisciplinary Pain Center.⁹⁹ Their preliminary data suggest that treated patients make mild to moderate gains in multiple domains including functional status and health-related quality of life, psychosocial distress, physical symptoms, and physical health concerns.³⁵

Bonica at the University of Washington was among the first to apply a multidisciplinary approach to the treatment of chronic pain patients in the late 1950s.⁹⁹ Since then, the approach has gained relatively wide acceptance for work-impaired chronic pain patients, especially those with back pain and fibromyalgia. A recent meta-analysis of 65 controlled studies of multidisciplinary interventions for chronic pain patients noted improvements in return to work rates, pain, mood, and health care utilization.⁴³ The authors were cautious in their conclusions, noting that the level of methodological rigor for most studies was low.

IS-generated reports may monitor the patient population for individuals who develop chronic MUPS-related disability. If patients are recognized early and enrolled in specialized intensive multimodal care for MUPS, the chances of satisfactorily returning them to work may be maximized.

Components of Specialized Services The following sections review the common components of most intensive programs and the research that supports their efficacy.

Cognitive-behavioral therapy. Until recently, most approaches to patients with treatment-refractory chronic pain or other persistent disabling MUPS have involved an intensive burst of multimodal care delivered over several weeks, usually in an inpatient setting. Perhaps not surprisingly, given the general shift in emphasis from inpatient care to less expensive outpatient approaches, recent studies have evaluated less intensive but more longitudinal treatment strategies. The best studied of these involve combined cognitive-behavioral therapy (CBT) and physical reactivation. CBT used in this context aims to help patients test and appropriately adjust harmful beliefs that they may have regarding the cause of their symptoms and the ways of treating their symptoms. Empirical trials have shown the benefits of CBT for a range of MUPS including chronic fatigue,¹³¹ irritable bowel syndrome,^{117,146} temporomandibular disorders,³² burning mouth syndrome,¹⁰ hypochondriasis,¹⁵⁷ and multiple MUPS.^{64,96,138}

Wessely's group³⁰ in London found that 63 percent of patients with chronic fatigue syndrome (CFS) showed significant improvement in their physical functioning after random assignment to CBT and physical activation, whereas only 19 percent assigned to relaxation training showed significant improvement. Improvements were enhanced over the 6 months following treatment. Significant improvements among CBT-physical activation recipients over those among the relaxation group were also noted in work and social adjustment, symptoms of fatigue, fatigue-related problems, and progress toward individualized long-term goals. Of note, improvements in dis-

tress and depression were only slightly better in the CBT-physical activation group, and the differences were not statistically significant.

Sharpe and colleagues¹³¹ completed a randomized trial of CBT for patients with CFS by comparing it with usual medical care. They found that 73 percent of patients assigned to CBT rated their outcome as satisfactory or better, whereas only 27 percent of the usual care group gave such a rating, a difference that was highly statistically significant. Sixty-three percent of the CBT group improved in their work functioning, whereas only 20 percent of the usual care group improved in their work functioning. Functioning, fatigue, and depression but not anxiety were also significantly improved. As one would hypothesize under a model of treatment with CBT, illness beliefs and coping were more positively altered for those assigned to CBT than for those assigned to usual care. As was observed in the previously described CBT-physical activation trial, outcomes continued to improve for months after the completion of the intervention.¹³¹

Physical activation and exercise. Exercise is known to have important physical and psychological impacts upon health and well-being.^{104,160} Using a randomized design, Fulcher and White⁴⁸ examined the impact of a gradually increasing program of supervised aerobic exercise for patients with CFS, comparing this approach to stretching and relaxation. After 12 weekly sessions, 51 percent of those assigned to exercise rated themselves globally as “much better” or “very much better,” whereas 27 percent of the stretching and relaxation group gave such a rating, a statistically significant difference, and improvements were stable over the subsequent several months. Fatigue, physical functioning, and fitness were also significantly better in the exercise group.⁴⁸ Similar findings after exercise programs have been noted for other chronic or symptom-based disorders such as post-polio syndrome,⁴¹ chronic low back pain,^{46,47} depressive disorders,¹⁶⁰ fibromyalgia,¹⁰³ and “effort syndrome.”¹¹⁰

Return-to-work strategies. Challenges exist around when and how to return workers with MUPS to work. There is general agreement that an early return to work is important to maintain role functioning and reduce chronic disability. There is evidence in the low back pain literature that a return to modified work can be successful.⁴⁵ Currently, the Army employs a profiling system of temporary or permanent work restrictions for those with diminished occupational functioning because of illness. Unfortunately, this approach may actually reinforce disability unless it is used in combination with a carefully supervised and graduated but relatively rapid return-to-work plan that is introduced to the worker very early in the rehabilitation process. For example, a 1-year follow-up of the use of work restrictions for nonspecific low back pain indicated they actually diminished the likelihood of return to work and did not reduce subsequent work absence or recurrences of back pain.¹⁹ A supervised and graduated return-to-work approach may be especially important in the military when aerobic physical conditioning such as long-distance running is required. A “profile” brands the worker as a problem to supervisors and coworkers. The loss of physical con-

ditioning and endurance that occurs in response to persistent physical symptoms and resulting deactivation requires time and a graduated program to reverse. Abrupt and haphazard return of personnel to full physical duties and the expectation that they will immediately perform at the same levels as others in their unit will commonly produce failure and an increased sense of defeat for the worker. In contrast, a rapid return of workers to their full levels of supervisory and other nonphysical roles is indicated to reinforce organizational expectations that a rapid return to productivity is expected. Likewise, worker productivity helps bolster self-esteem and a sense of accomplishment.

Obstacles to Specialized Services The greatest obstacle to the development of specialized care for patients with MUPS is the perception on the part of administrators, policy makers, and clinicians that MUPS are neither disabling nor important. Although explanations of “stress” or “somatization” for unexplained physical symptoms serve an important clinical purpose for many MUPS patients, they are often used to minimize the needs of affected patients. Another barrier at present is the lack of an institutionalized niche for specialized care for MUPS, especially after combat and deployments. Both primary and tertiary care of MUPS is, as we have shown, interdisciplinary and requires the collaboration of many clinicians such as generalists, psychiatrists, psychologists, physiatrists, anesthesiologists, nurses, social workers, physical therapists, occupational therapists, and dietitians. In the current health care environment, each of these clinicians is responsible to a department head, and departments are demarcated along specialty lines. Interdisciplinary care of MUPS is a lesser priority for each of these departments than illnesses that fall more clearly within their specialty purview. When competing clinical demands are high, the argument that patients with MUPS suffer more from “nothing” than “something” seems compelling organizationally.

Another important obstacle to intensive models of MUPS care is the conventional sense that such care is too costly. Currently, it is not known whether the extra costs associated with appropriate intervention are offset by longer-term decreases in health care use and improvements in occupational functioning. Most patients referred to intensive MUPS care, however, are using unusually large amounts of health care and are functioning poorly, so the potential for gains appears to be great. Left untreated, patients with MUPS remain costly to society. For the military, MUPS seem certain to occur after future wars, and excellence in this aspect of patient care may pay public relations dividends as well as improve the care of affected veterans. Further research on the cost-effectiveness of specialized services for patients with treatment-refractory MUPS is needed to rigorously examine these issues.

CONCLUSIONS

Hadler⁵⁸ has described four major areas in which occupational physicians might contribute to the care of workers: clinical, educational, research, and pol-

icy making. We adhere to his comprehensive outline and offer our own thoughts and a few of his in concluding this review of MUPS and their relevance for the military and perhaps other employers.

First, physicians caring for workers with MUPS must foster improved worker adaptation to illness *as the worker experiences it*. Hadler has urged physicians to try to understand the “sociopolitical arena” in which illness occurs. We urge clinicians to go several steps further and design a system of care that is responsive to people and their subjective health concerns rather than diseases per se.

Second, physicians caring for workers with MUPS must develop appropriate educational experiences for other providers and for affected workers and their significant others. Clinician education should emphasize the psychosocial and behavioral contexts of illness and disability rather than only simplistic biomedical perspectives. Providers must become more sophisticated regarding the ways that environmental factors may shape behavioral responses to symptoms and to ill health.

Third, physicians caring for workers with MUPS must develop short-, intermediate-, and long-term clinical research and policy research agendas with explicit goals and objectives. These research agendas must address important military health practice and policy questions. Research into biological mechanisms, although important for understanding one basis of unexplained symptoms, is costly. History suggests that mechanistic research is slow to yield immediate answers of importance to workers, patients, and organizations. Rather, epidemiological research is necessary to aid policy makers’ attempts to comprehend the societal and military burdens of MUPS and the historical relevance of MUPS to diverse deployments.¹²² Hadler has recommended research on the impact of job demands on physical and emotional health and workers’ health perceptions, and this remains an area of need. Where, how, and why veterans with postdeployment health concerns seek their care and their satisfaction with that care is currently completely unknown within the military and is of great importance to prevention, treatment, and risk communication efforts.

Fourth, we suggest that physicians and policy makers move as rapidly as possible toward population-based models of health care and create system incentives for local-level development of novel interdisciplinary approaches to MUPS, interventions that span the spectrum of precare, primary care, collaborative primary care, and intensive specialty care. Physicians and policy makers must consider human factors whenever they are engaged in workplace structure and task design, since in the end, new technologies are effective only if the people who operate them are functioning well. Physicians and policy makers should carefully consider the impact of the prevailing military and U.S. Department of Veterans Affairs disability compensation system on incentives for workers to improve their health.

Given the necessary breadth of efforts to prevent MUPS in the military, we suggest the development of a “center of excellence” to lead clinical, research, and educational efforts related to MUPS in the military. A center of excellence could initiate and monitor efforts to implement clinical, educational, and re-

search agendas pertaining to MUPS. When appropriate, the center could provide input to military policy makers interested in ensuring that they consider the impact of MUPS as they design, monitor, and adjust military health policy. A center of excellence would centralize U.S. Department of Defense responsibility in this arena and enhance organizational accountability. Eventually, military medicine's innovations may provide an important model for civilian health care organizations seeking solutions to the difficult challenge of medically unexplained physical symptoms.

REFERENCES

1. Abbey SE, Garfinkel PE. Neurasthenia and chronic fatigue syndrome: The role of culture in the making of a diagnosis. *American Journal of Psychiatry* 1991; 148(12):1638–46.
2. Adler RH, Zlot S, Hurny C, Minder C. Engel's "Psychogenic Pain and the Pain-Prone Patient:" A retrospective, controlled clinical study. *Psychosomatic Medicine* 1989;51(1):87–101.
3. Alexander RW, Bradley LA, Alarcon GS, et al. Sexual and physical abuse in women with fibromyalgia: Association with outpatient health care utilization and pain medication usage. *Arthritis Care Research* 1998;11(2):102–15.
4. Almy TP. Experimental studies on the irritable colon. *American Journal of Medicine* 1951;10:60–7.
5. Andersen SM, Harthorn BH. Changing the psychiatric knowledge of primary care physicians. The effects of a brief intervention on clinical diagnosis and treatment. *General Hospital Psychiatry* 1990;12(3):177–90.
6. Anderson JS, Ferrans CE. The quality of life of persons with chronic fatigue syndrome. *Journal of Nervous and Mental Disease* 1997;185(6):359–67.
7. Angell M. Breast implants—protection or paternalism? *New England Journal of Medicine* 1992;326(25):1695–6.
8. Barsky AJ, Delamater BA, Clancy SA, Antman EM, Ahern DK. Somatized psychiatric disorder presenting as palpitations. *Archives of Internal Medicine* 1996; 156(10):1102–8.
9. Beitman BD, Kushner MG, Basha I, Lamberti J, Mukerji V, Bartels K. Follow-up status of patients with angiographically normal coronary arteries and panic disorder. *JAMA* 1991;265(12):1545–9.
10. Bergdahl J, Anneroth G, Perris H. Cognitive therapy in the treatment of patients with resistant burning mouth syndrome: A controlled study. *Journal of Oral Pathology and Medicine* 1995;24(5):213–5.
11. Bigos SJ, Battie MC, Spengler DM, et al. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. *Spine* 1991;16(1):1–6.
12. Blumer D, Montouris G, Hermann B. Psychiatric morbidity in seizure patients on a neurodiagnostic monitoring unit. *Journal of Neuropsychiatry and Clinical Neurosciences* 1995;7(4):445–56.
13. Bombardier CH, Buchwald D. Outcome and prognosis of patients with chronic fatigue vs. chronic fatigue syndrome. *Archives of Internal Medicine* 1995;155(19): 2105–10.
14. Boxer PA. Indoor air quality: A psychosocial perspective. *Journal of Occupational Medicine* 1990;32(5):425–8.

15. Bridges KW, Goldberg DP. Somatic presentation of DSM III psychiatric disorders in primary care. *Journal of Psychosomatic Research* 1985;29(6):563–9.
16. Brown J, Chapman S, Lupton D. Infinitesimal risk as public health crisis: News media coverage of a doctor–patient HIV contact tracing investigation. *Social Science and Medicine* 1996;43(12):1685–95.
17. Buchwald D, Garrity D. Comparison of patients with chronic fatigue syndrome, fibromyalgia, and multiple chemical sensitivities. *Archives of Internal Medicine* 1994;154(18):2049–53.
18. Buchwald D, Umali P, Umali J, Kith P, Pearlman T, Komaroff AL. Chronic fatigue and the chronic fatigue syndrome: Prevalence in a Pacific Northwest health care system. *Annals of Internal Medicine* 1995;123(2):81–8.
19. Burton AK, Erg E. Back injury and work loss. Biomechanical and psychosocial influences. *Spine* 1997;22(21):2575–80.
20. Catalan J, Gath DH, Anastasiades P, Bond SA, Day A, Hall L. Evaluation of a brief psychological treatment for emotional disorders in primary care. *Psychological Medicine* 1991;21(4):1013–8.
21. Chester AC, Levine PH. The natural history of concurrent sick building syndrome and chronic fatigue syndrome. *Journal of Psychiatric Research* 1997;31(1):51–7.
22. Clark MR, Katon W, Russo J, Kith P, Sintay M, Buchwald D. Chronic fatigue: Risk factors for symptom persistence in a 2 1/2-year follow-up study. *American Journal of Medicine* 1995;98(2):187–95.
23. Clauw DJ, Schmidt M, Radulovic D, Singer A, Katz P, Bresette J. The relationship between fibromyalgia and interstitial cystitis. *Journal of Psychiatric Research* 1997;31(1):125–31.
24. Colligan M, Pennebaker J, Murphy. *Mass Psychogenic Illness: A Social Psychological Analysis*. Hillsdale, N.J.: Lawrence Erlbaum Associates; 1982.
25. Coryell W, Norten SG. Briquet’s syndrome (somatization disorder) and primary depression: Comparison of background and outcome. *Comprehensive Psychiatry* 1981;22(3):249–56.
26. Craig TK, Boardman AP, Mills K, Daly-Jones O, Drake H. The South London Somatisation Study. I: Longitudinal course and the influence of early life experiences. *British Journal of Psychiatry* 1993;163:579–88.
27. Craig TK, Brown GW. Goal frustration and life events in the aetiology of painful gastrointestinal disorder. *Journal of Psychosomatic Research* 1984;28(5):411–21.
28. Craufurd DI, Creed F, Jayson MI. Life events and psychological disturbance in patients with low-back pain. *Spine* 1990;15(6):490–4.
29. Creed F. Life events and appendectomy. *Lancet* 1981;1(8235):1381–5.
30. Deale A, Chalder T, Marks I, Wessely S. Cognitive behavior therapy for chronic fatigue syndrome: A randomized controlled trial. *American Journal of Psychiatry* 1997;154(3):408–14.
31. Drake MEJ, Padamadan H, Pakalnis A. EEG frequency analysis in conversion and somatoform disorder. *Clinical Electroencephalography* 1988;19(3):123–8.
32. Dworkin SF, Turner JA, Wilson L, et al. Brief group cognitive-behavioral intervention for temporomandibular disorders. *Pain* 1994;59(2):175–87.
33. Eisenberg L. Treating depression and anxiety in primary care. Closing the gap between knowledge and practice. *New England Journal of Medicine* 1992;326(16):1080–4.
34. Engel CC Jr. A collaborative primary care-mental health program in the military. *Federal Practitioner* 1994;11(11):18–29.

35. Engel CC Jr., Liu X, Miller R, et al. A 3-week intensive outpatient behavioral medicine intervention for veterans with persistent, unexplained post-war symptoms (slide presentation). The Society of Behavioral Medicine, 19th Annual Scientific Sessions, New Orleans, 1998.
36. Engel CC Jr., Roy M, Kayanan D, Ursano R. Multidisciplinary treatment of persistent symptoms after Gulf War service. *Military Medicine* 1998;163(4):202-8.
37. Engel CC Jr., von Korff M, Katon WJ. Back pain in primary care: Predictors of high health-care costs. *Pain* 1996;65(2-3):197-204.
38. Escobar JI, Golding JM, Hough RL, Karno M, Burnam MA, Wells KB. Somatization in the community: Relationship to disability and use of services. *American Journal of Public Health* 1987;77(7):837-40.
39. Escobar JI, Rubio-Stipec M, Canino GJ, Karno M. Somatic symptom index (SSI): A new and abridged somatization construct. *Journal of Nervous and Mental Diseases* 1989;177(3):140-6.
40. Feinstein AR. Nosologic Challenges of Diagnostic Criteria for a "New Illness." Conference on Federally Sponsored Gulf War Veterans' Illnesses Research: June, 1998; Pentagon City, Va.
41. Feldman RM, Soskolne CL. The use of nonfatiguing strengthening exercises in post-polio syndrome. *Birth Defects Original Article Series* 1987;23(4):335-41.
42. Fisher ES, Welch HG. Avoiding the unintended consequences of growth in medical care: How might more be worse? *JAMA* 1999;281(5):446-53.
43. Flor-H, Fydrich T, Turk DC. Efficacy of multidisciplinary pain treatment centers: A meta-analytic review. *Pain* 1992;49:221-30.
44. Flor-Henry P, Fromm-Auch D, Tapper M, Schopflocher D. A neuropsychological study of the stable syndrome of hysteria. *Biological Psychiatry* 1981;16(7):601-26.
45. Frank JW, Brooker AS, DeMaio SE, et al. Disability resulting from occupational low back pain. Part II: What do we know about secondary prevention? A review of the scientific evidence on prevention after disability begins. *Spine* 1996;21(24):2918-29.
46. Frost H, Klaber Moffett JA, Moser JS, Fairbank JC. Randomised controlled trial for evaluation of fitness programme for patients with chronic low back pain. *BMJ* 1995;310(6973):151-4.
47. Frost H, Lamb SE, Klaber Moffett JA, Fairbank JC, Moser JS. A fitness programme for patients with chronic low back pain: 2-year follow-up of a randomised controlled trial. *Pain* 1998;75(2-3):273-9.
48. Fulcher KY, White PD. Randomised controlled trial of graded exercise in patients with the chronic fatigue syndrome. *BMJ* 1997;314(7095):1647-52.
49. Gallagher TH, Lo B, Chesney M, Christensen K. How do physicians respond to patient's requests for costly, unindicated services? *Journal of General Internal Medicine* 1997;12(11):663-8.
50. Gatchel RJ, Polatin PB, Kinney RK. Predicting outcome of chronic back pain using clinical predictors of psychopathology: A prospective analysis. *Health Psychology* 1995;14(5):415-20.
51. Goldberg D. Reasons for misdiagnosis. Sartorius N, Goldberg D, de Girolamo G, Costa e Silva J-G, Lecrubier Y, Wittchen U., Editors. *Psychological Disorders in General Medical Settings*. Lewiston, N.Y.: Hogrefe and Huber; 1990:139-45.
52. Goldenberg DL. Fibromyalgia syndrome. An emerging but controversial condition. [Review]. *JAMA* 1987;257(20):2782-7.

53. Goldman SL, Kraemer DT, Salovey P. Beliefs about mood moderate the relationship of stress to illness and symptom reporting. *Journal of Psychosomatic Research* 1996;41(2):115–28.
54. Gomborone JE, Gorard DA, Dewsnap PA, Libby GW, Farthing MJ. Prevalence of irritable bowel syndrome in chronic fatigue. *Journal of the Royal College of Physicians of London* 1996;30(6):512–3.
55. Gordon E, Kraiuhin C, Kelly P, Meares R, Howson A. A neurophysiological study of somatization disorder. *Comprehensive Psychiatry* 1986;27(4):295–301.
56. Gureje O, Von Korff M, Simon GE, Gater R. Persistent pain and well-being: A World Health Organization study in primary care. *JAMA* 1998;280(2):147–51.
57. Gwee KA, Graham JC, McKendrick MW, et al. Psychometric scores and persistence of irritable bowel after infectious diarrhoea. *Lancet* 1996; 347(8995):150–3.
58. Hadler NM. Occupational illness. The issue of causality. *Journal of Occupational Medicine* 1984;26(8):587–93.
59. Hadler NM. If you have to prove you are ill, you can't get well. *Spine* 1996;21(20): 2397–2400.
60. Hadler NM. Fibromyalgia, chronic fatigue, and other iatrogenic diagnostic algorithms. Do some labels escalate illness in vulnerable patients? [see comments]. [Review] [46 refs]. *Postgraduate Medicine* 1997;102(2):161–2, 165–6, 171–2 passim.
61. Hahn SR, Thompson KS, Wills TA, Stern V, Budner NS. The difficult doctor–patient relationship: Somatization, personality and psychopathology. *Journal of Clinical Epidemiology* 1994;47(6):647–57.
62. Hall EM, Johnson JV. A case study of stress and mass psychogenic illness in industrial workers. *Journal of Occupational Medicine* 1989;31(3):243–50.
63. Haynes RB, Sackett DL, Taylor DW, Gibson ES, Johnson AL. Increased absenteeism from work after detection and labeling of hypertensive patients. *New England Journal of Medicine* 1978;299(14):741–4.
64. Hellman CJ, Budd M, Borysenko J, McClelland DC, Benson H. A study of the effectiveness of two group behavioral medicine interventions for patients with psychosomatic complaints. *Behavioral Medicine* 1990;16(4):165–73.
65. Hotopf M, Mayou R, Wadsworth M, Wessely S. Temporal relationships between physical symptoms and psychiatric disorder. Results from a national birth cohort. *British Journal of Psychiatry* 1998;173:255–61.
66. Hotopf M, Mayou R, Wessely S. Childhood risk factors for adult medically unexplained symptoms: Results from a prospective cohort study. 44th Annual Meeting of the Academy of Psychosomatic Medicine 1998.
67. Hyams KC. Developing case definitions for symptom-based conditions: The problem of specificity. *Epidemiological Reviews* 1998;20(2):148–56.
68. Irwin C, Falsetti SA, Lydiard RB, Ballenger JC, Brock CD, Brenner W. Comorbidity of posttraumatic stress disorder and irritable bowel syndrome. *Journal of Clinical Psychiatry* 1996;57(12):576–8.
69. Johanning E, Auger PL, Reijula K. Building-related illnesses. *New England Journal of Medicine* 1998;338(15):1070; discussion 1071.
70. Kaplan DS, Masand PS, Gupta S. The relationship of irritable bowel syndrome (IBS) and panic disorder. *Annals of Clinical Psychiatry* 1996;8(2):81–8.
71. Kashner TM, Rost K, Smith GR, Lewis S. An analysis of panel data. The impact of a psychiatric consultation letter on the expenditures and outcomes of care for patients with somatization disorder. *Medical Care* 1992;30(9):811–21.
72. Kates N. Psychiatric consultation in the family physician's office. Advantages and hidden benefits. *General Hospital Psychiatry* 1988;10(6):431–7.

73. Katon W, Hall ML, Russo J, et al. Chest pain: Relationship of psychiatric illness to coronary arteriographic results. *American Journal of Medicine* 1988;84(1):1–9.
74. Katon W, Lin E, Von Korff M, Russo J, Lipscomb P, Bush T. Somatization: A spectrum of severity. *American Journal of Psychiatry* 1991;148(1):34–40.
75. Katon W, Robinson P, Von Korff M, et al. A multifaceted intervention to improve treatment of depression in primary care. *Archives of General Psychiatry* 1996; 53(10):924–32.
76. Katon W, Russo J. Chronic fatigue syndrome criteria. A critique of the requirement for multiple physical complaints. *Archives of Internal Medicine* 1992;152(8):1604–9.
77. Katon W, Von Korff M, Lin E, Bush T, Ormel J. Adequacy and duration of antidepressant treatment in primary care. *Medical Care* 1992;30(1):67–76.
78. Katon W, Von Korff M, Lin E, et al. Distressed high utilizers of medical care. DSM-III-R diagnoses and treatment needs. *General Hospital Psychiatry* 1990; 12(6):355–62.
79. Katon W, Von Korff M, Lin E, et al. A randomized trial of psychiatric consultation with distressed high utilizers. *General Hospital Psychiatry* 1992;14(2):86–98.
80. Katon W, Von Korff M, Lin E, et al. Collaborative management to achieve treatment guidelines. Impact on depression in primary care. *JAMA* 1995;273(13):1026–31.
81. Katon W, Von Korff M, Lin E, et al. Population-based care of depression: Effective disease management strategies to decrease prevalence. *General Hospital Psychiatry* 1997;19(3):169–78.
82. Katon WJ, Buchwald DS, Simon GE, Russo JE, Mease PJ. Psychiatric illness in patients with chronic fatigue and those with rheumatoid arthritis. *Journal of General Internal Medicine* 1991;6(4):277–85.
83. Katon WJ, Sullivan M. Antidepressant treatment of functional somatic symptoms. Mayou RA, Bass C, Sharpe M, Editors. *Treatment of Functional Somatic Symptoms*. New York: Oxford University Press; 1995.
84. Kirmayer LJ, Robbins JM. Three forms of somatization in primary care: Prevalence, co-occurrence, and sociodemographic characteristics. *Journal of Nervous and Mental Disease* 1991;179(11):647–55.
85. Koss MP, Koss PG, Woodruff WJ. Deleterious effects of criminal victimization on women's health and medical utilization. *Archives of Internal Medicine* 1991; 151(2):342–7.
86. Kouyanou K, Pither CE, Wessely S. Iatrogenic factors and chronic pain. *Psychosomatic Medicine* 1997;59(6):597–604.
87. Kroenke K. Symptoms in medical patients: An untended field. *American Journal of Medicine* 1992;92(1A):3S–6S.
88. Kroenke K, Arrington ME, Mangelsdorff AD. The prevalence of symptoms in medical outpatients and the adequacy of therapy. *Archives of Internal Medicine* 1990;150(8):1685–9.
89. Kroenke K, Jackson JL, Chamberlin J. Depressive and anxiety disorders in patients presenting with physical complaints: Clinical predictors and outcome. *American Journal of Medicine* 1997;103(5):339–47.
90. Kroenke K, Mangelsdorff AD. Common symptoms in ambulatory care: Incidence, evaluation, therapy, and outcome. *American Journal of Medicine* 1989;86(3):262–6.
91. Kroenke K, Price RK. Symptoms in the community. Prevalence, classification, and psychiatric comorbidity. *Archives of Internal Medicine* 1993;153(21):2474–80.

92. Kroenke K, Spitzer RL, deGruy FV3rd, et al. Multisomatoform disorder. An alternative to undifferentiated somatoform disorder for the somatizing patient in primary care. *Archives of General Psychiatry* 1997;54(4):352–8.
93. Kroenke K, Spitzer RL, Williams JB, et al. Physical symptoms in primary care. Predictors of psychiatric disorders and functional impairment. *Archives of Family Medicine* 1994;3(9):774–9.
94. Kroenke K, Wood DR, Mangelsdorff AD, Meier NJ, Powell JB. Chronic fatigue in primary care. Prevalence, patient characteristics, and outcome. *JAMA* 1988;260(7):929–34.
95. Lembo T, Fullerton S, Diehl D, et al. Symptom duration in patients with irritable bowel syndrome. *American Journal of Gastroenterology* 1996;91(5):898–905.
96. Lidbeck J. Group therapy for somatization disorders in general practice: Effectiveness of a short cognitive-behavioural treatment model. *Acta Psychiatrica Scandinavica* 1997;96(1):14–24.
97. Lin EH, Katon W, Von Korff M, et al. Frustrating patients: Physician and patient perspectives among distressed high users of medical services. *Journal of General Internal Medicine* 1991;6(3):241–6.
98. Linton SJ. A population-based study of the relationship between sexual abuse and back pain: Establishing a link. *Pain* 1997;73(1):47–53.
99. Loeser JD, Egan KJ, Editors. *Managing the Chronic Pain Patient: Theory and Practice at the University of Washington Multidisciplinary Pain Center*. New York: Raven Press; 1989.
100. Loeser JD, Sullivan M. Doctors, diagnosis, and disability: A disastrous diversion. *Clinical Orthopaedics and Related Research* 1997(336):61–6.
101. Marks JN, Goldberg DP, Hillier VF. Determinants of the ability of general practitioners to detect psychiatric illness. *Psychological Medicine* 1979;9(2):337–53.
102. Marple RL, Kroenke K, Lucey CR, Wilder J, Lucas CA. Concerns and expectations in patients presenting with physical complaints. Frequency, physician perceptions and actions, and 2-week outcome. *Archives of Internal Medicine* 1997;157(13):1482–8.
103. McCain GA, Bell DA, Mai FM, Halliday PD. A controlled study of the effects of a supervised cardiovascular fitness training program on the manifestations of primary fibromyalgia. *Arthritis and Rheumatism* 1988;31(9):1135–41.
104. McCully KK, Sisto SA, Natelson BH. Use of exercise for treatment of chronic fatigue syndrome. *Sports Medicine* 1996;21(1):35–48.
105. McDonald AJ, Bauchier PAD. Non-organic gastro-intestinal illness: A medical and psychiatric study. *British Journal of Psychiatry* 1980;136:1276–83.
106. Meador CK. The art and science of non-disease. *New England Journal of Medicine* 1965;272:92.
107. Murray J, Corney R. Not a medical problem? An intensive study of the attitudes and illness behaviour of low attenders with psychosocial difficulties. *Social Psychiatry and Psychiatric Epidemiology* 1990;25(3):159–64.
108. National Institute of Mental Health. Mental disorder and primary medical care: An analytical review of the literature. DHEW Publication No. (ADM) 78-661. Washington, D.C.: Superintendent of Documents, U.S. Government Printing Office; 1979.
109. Neitzert CS, Davis C, Kennedy SH. Personality factors related to the prevalence of somatic symptoms and medical complaints in a healthy student population. *British Journal of Medical Psychology* 1997;70(Pt. 1):93–101.
110. Newham D, Edwards RH. Effort syndromes. *Physiotherapy* 1979;65(2):52–6.

111. Noyes R, Reich J, Clancy J, O'Gorman TW. Reduction in hypochondriasis with treatment of panic disorder. *British Journal of Psychiatry* 1986;149:631–5.
112. Olfson M. Primary care patients who refuse specialized mental health services. *Archives of Internal Medicine* 1991;151(1):129–32.
113. Orleans CT, George LK, Houpt JL, Brodie HK. How primary care physicians treat psychiatric disorders: A national survey of family practitioners. *American Journal of Psychiatry* 1985;142(1):52–7.
114. Ormel J, Giel R. Medical effects of nonrecognition of affective disorders in primary care. Sartorius N, Goldberg D, de Girolamo G, Costa e Silva J-G, Lecrubier Y, Wittchen U., Editors. *Psychological Disorders in General Medical Settings*. Lewiston, N.Y.: Hogrefe and Huber; 1990:146–58.
115. Ormel J, Koeter MW, van den Brink W, van de Willege G. Recognition, management, and course of anxiety and depression in general practice. *Archives of General Psychiatry* 1991;48(8):700–6.
116. Ormel J, van den Brink W, Giel MWJ, van der Meer K, van de Willige G, Wilink FW. Recognition, management and outcome of psychological disorders in primary care: A naturalistic follow-up study. *Psychological Medicine* 1990;20:909–23.
117. Payne A, Blanchard EB. A controlled comparison of cognitive therapy and self-help support groups in the treatment of irritable bowel syndrome. *Journal of Consulting and Clinical Psychology* 1995;63(5):779–86.
118. Plesh O, Wolfe F, Lane N. The relationship between fibromyalgia and temporomandibular disorders: Prevalence and symptom severity. *Journal of Rheumatology* 1996;23(11):1948–52.
119. Potts SG, Bass CM. Psychological morbidity in patients with chest pain and normal or near-normal coronary arteries: A long-term follow-up study. *Psychological Medicine* 1995;25(2):339–47.
120. Quill TE. Somatization disorder. One of medicine's blind spots. *JAMA* 1985;254(21):3075–9.
121. Richard K. The occurrence of maladaptive health-related behaviors and teacher-related conduct problems in children of chronic low back pain patients. *Journal of Behavioral Medicine* 1988;11:107–116.
122. Robins LN. Psychiatric epidemiology. *Archives of General Psychiatry* 1978;35:697–702.
123. Rose G. *The Strategy of Preventive Medicine*. New York: Oxford University Press; 1992.
124. Rost K, Kashner TM, Smith RGJ. Effectiveness of psychiatric intervention with somatization disorder patients: Improved outcomes at reduced costs. *General Hospital Psychiatry* 1994;16(6):381–7.
125. Rothman KJ, Greenland S. Measures of disease frequency. Rothman KJ, Greenland S, editors. *Modern Epidemiology*. 2nd ed. Philadelphia: Lippincott-Raven Publishers; 1998.
126. Russo J, Katon W, Clark M, Kith P, Sintay M, Buchwald D. Longitudinal changes associated with improvement in chronic fatigue patients. *Journal of Psychosomatic Research* 1998;45(1 Spec. No.):67–76.
127. Schappert SM. National Ambulatory Medical Care Survey: 1989 Summary. *Vital Health Statistics* 1992;13(110)1–80.
128. Schrader H, Obelieniene D, Bovim G, et al. Natural evolution of late whiplash syndrome outside the medicolegal context. *Lancet* 1996;347(9010):1207–11.
129. Schweitzer R, Kelly B, Foran A, Terry D, Whiting J. Quality of life in chronic fatigue syndrome. *Social Science and Medicine* 1995;41(10):1367–72.

130. Sharpe M, Hawton K, Seagroatt V, Pasvol G. Follow up of patients presenting with fatigue to an infectious diseases clinic. *BMJ* 1992;305(6846):147–52.
131. Sharpe M, Hawton K, Simkin S, et al. Cognitive behaviour therapy for the chronic fatigue syndrome: A randomized controlled trial. *BMJ* 1996;312(7022):22–6.
132. Sharpe MC. Cognitive-behavioral therapy for patients with chronic fatigue syndrome: How? In: MA Demitrack; SE Abbey (Eds.). *Chronic Fatigue Syndrome: An Integrative Approach to Evaluation and Treatment*. New York: Guilford Press; 1996:240–262.
133. Shuchman M, Wilkes MS. Medical scientists and health news reporting: A case of miscommunication. *Annals of Internal Medicine* 1997;126(12):976–82.
134. Silver FW. Management of conversion disorder. *American Journal of Physical Medicine and Rehabilitation* 1996;75(2):134–40.
135. Simon GE, VonKorff M. Somatization and psychiatric disorder in the NIMH Epidemiologic Catchment Area study. *American Journal of Psychiatry* 1991;148(11):1494–1500.
136. Smith GR Jr. *Somatization Disorder in the Medical Setting*. Washington, D.C.: American Psychiatric Press; 1991.
137. Smith GR Jr., Monson RA, Ray DC. Psychiatric consultation in somatization disorder. A randomized controlled study. *New England Journal of Medicine* 1986;314(22):1407–13.
138. Speckens AE, van Hemert AM, Spinhoven P, Hawton KE, Bolk JH, Rooijmans HG. Cognitive behavioural therapy for medically unexplained physical symptoms: A randomised controlled trial. *BMJ* 1995;311(7016):1328–32.
139. Struwing JP, Gray GC. An epidemic of respiratory complaints exacerbated by mass psychogenic illness in a military recruit population. *American Journal of Epidemiology* 1990;132(6):1120–9.
140. Sullivan MD, Katon W, Dobie R, Sakai C, Russo J, Harrop-Griffiths J. Disabling tinnitus. Association with affective disorder. *General Hospital Psychiatry* 1988;10(4):285–91.
141. Sullivan MD, Loeser JD. The diagnosis of disability. Treating and rating disability in a pain clinic. *Archives of Internal Medicine* 1992;152(9):1829–35.
142. Swartz M, Landerman R, George LK, Blazer DG, Escobar J. Somatization disorder. Robins LN, Regier DA, Editors. *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study*. New York: Free Press; 1991.
143. Symonds TL, Burton AK, Tillotson KM, Main CJ. Absence resulting from low back trouble can be reduced by psychosocial intervention at the work place. *Spine* 1995;20(24):2738–45.
144. Thompson WG. *Gut Reactions: Understanding the Symptoms of the Digestive Tract*. New York: Plenum; 1989.
145. Twemlow SW, Bradshaw SLJ, Coyne L, Lerma BH. Patterns of utilization of medical care and perceptions of the relationship between doctor and patient with chronic illness including chronic fatigue syndrome. *Psychological Reports* 1997;80(2):643–58.
146. van Dulmen AM, Fennis JF, Bleijenbergh G. Cognitive-behavioral group therapy for irritable bowel syndrome: Effects and long-term follow-up. *Psychosomatic Medicine* 1996;58(5):508–14.
147. Vaughan KB, Lanzetta JT. The effect of modification of expressive displays on vicarious emotional arousal. *Journal of Experimental Social Psychology* 1981;17(1):16–30.

148. Vercoulen JH, Swanink CM, Fennis JF, Galama JM, van der Meer JW, Bleijenberg G. Prognosis in chronic fatigue syndrome: A prospective study on the natural course. *Journal of Neurology, Neurosurgery and Psychiatry* 1996;60(5):489–94.
149. Von Korff M, Katon W, Bush T, et al. Treatment costs, cost offset, and cost-effectiveness of collaborative management of depression. *Psychosomatic Medicine* 1998;60(2):143–9.
150. Von Korff M, Le Resche L, Dworkin SF. First onset of common pain symptoms: A prospective study of depression as a risk factor. *Pain* 1993;55(2):251–8.
151. Wagner EH, Austin BT, Von Korff M. Organizing care for patients with chronic illness. *Milbank Quarterly* 1996;74(4):511–44.
152. Walker E, Katon W, Harrop-Griffiths J, Holm L, Russo J, Hickok LR. Relationship of chronic pelvic pain to psychiatric diagnoses and childhood sexual abuse. *American Journal of Psychiatry* 1988;145(1):75–80.
153. Walker EA, Katon WJ, Hansom J, Harrop-Griffiths, Holm L, Jones ML, Hickok L, Jemelka RP. Medical and psychiatric symptoms in women with childhood sexual abuse. *Psychosomatic Medicine* 1992;54(6):658–64.
154. Walker EA, Katon WJ, Keegan D, Gardner G, Sullivan M. Predictors of physician frustration in the care of patients with rheumatological complaints [see comments]. *General Hospital Psychiatry* 1997;19(5):315–23.
155. Walker EA, Keegan D, Gardner G, Sullivan M, Bernstein D, Katon WJ. Psychosocial factors in fibromyalgia compared with rheumatoid arthritis: II. Sexual, physical, and emotional abuse and neglect. *Psychosomatic Medicine* 1997;59(6):572–7.
156. Walker EA, Roy-Byrne PP, Katon WJ, Li L, Amos D, Jiranek G. Psychiatric illness and irritable bowel syndrome: A comparison with inflammatory bowel disease. *American Journal of Psychiatry* 1990;147(12):1656–61.
157. Warwick HM, Clark DM, Cobb AM, Salkovskis PM. A controlled trial of cognitive-behavioural treatment of hypochondriasis. *British Journal of Psychiatry* 1996; 169(2):189–95.
158. Wessely S, Nimnuan C, Sharpe M. Functional somatic syndromes—one or many? *Lancet* In Press.
159. Wessely S, Rose S, Bisson J. Brief psychological interventions (“debriefing”) for treating immediate trauma-related symptoms and preventing post-traumatic stress disorder (Cochrane Review). In: *The Cochrane Library* 1999; Oxford: Update Software.
160. Weyerer S, Kupfer B. Physical exercise and psychological health. *Sports Medicine* 1994;17(2):108–16.
161. White KL, Williams F, Greenberg BG. The ecology of medical care. *New England Journal of Medicine* 1961;265(18):885–92.
162. Woodman CL, Breen K, Noyes RJ, et al. The relationship between irritable bowel syndrome and psychiatric illness. A family study. *Psychosomatics* 1998;39(1):45–54.