The Natural History and Attentional Correlates of Conversion Symptoms in Neurological Patients.

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Abstract

Patients with conversion symptoms continue to present a substantial burden to neurologists and to psychiatrists who work in liaison with them. A survey of British neurologists suggested 36,000 of their consultations per annum were for symptoms presumed psychological in origin (Appendix 1).

A historical analysis of the concept of hysterical conversion shows it to be 200 years old. Freud's rediscovery of the term 100 years ago led to substantial revision by his analytic followers. Reasons for this, and for constant changes in its use within psychiatric diagnosis, are discussed (Chapter 1).

Past studies of the prognosis of conversion symptoms have been crucial for arguments concerning the diagnosis of hysteria and somatisation disorder. A new 10 year follow-up of 79 patients traced 73 of them. Medical reports and interviews indicated the original symptom had not improved in 30 patients, while neurological diagnoses accounting for the presenting symptom had been made in only 11. Symptom persistence was strongly associated with higher levels of consultation through the follow-up period, and with evidence of somatisation disorder at follow-up. Correlations are reported between symptomatic and diagnostic outcome with some initial findings on neurological and psychiatric examination (Chapter 2).

Two experimental studies tested hypotheses implicating attentional deficits in the pathogenesis of conversion symptoms. Groups with pseudoepileptic seizures and lower limb weakness of non-organic origin were compared with controls on tests of perceptual span and verbal-manual interference. Although a group with non-organic weakness performed more poorly than other groups on the latter task, the original hypotheses were not upheld (Chapter 3).
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Note on publication.


An edited version of sections 3.14 and 3.4 has been submitted for publication to the Journal of Nervous and Mental Disease.

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Introduction and thesis.

Patients whose symptoms mimic neurological illness, but for which no neurological disease can be diagnosed, continue to represent a significant part of the work of neurological hospitals. These "pseudoneurological" symptoms have traditionally received a diagnosis of "hysteria", especially where there has been positive evidence to suggest psychological causation. Concepts of hysteria have been greatly influenced by Sigmund Freud, who claimed to have introduced the term "conversion" to refer to a particular psychological mechanism by which symptoms were brought about. Whereas official classifications of mental and neurological disorder have eschewed "hysteria" as an approved term, "conversion" has been retained, at least until recently, to refer to the traditional phenomena of hysteria as in references to "conversion symptoms" and a syndrome of "conversion disorder". These epithets are no less controversial in their own way than "hysteria" has been. A growing body of opinion has suggested diagnosis of "conversion disorder" is unnecessary and dispensable. The work described here was inspired by my disagreement with such views.

To those who work in neurological hospitals, where patients with pseudoneurological symptoms continue to represent a significant and taxing clinical demand, this trend can seem unwarranted. Before any attempt is made to abolish this diagnostic option, it would be sensible to appraise its strengths and weaknesses so that an attempt to defend, consolidate or modify it is based on evidence rather than prejudice. This thesis asks whether and in what way reference to a category of conversion disorder remains justified. It will present a series of related studies intended to assist reconsideration of the diagnostic place of this term. They examine its usefulness, its historical rationale, and its validity with respect to prognosis and psychopathology.

Although "conversion disorder" is placed firmly in the ranks of mental disorder, and its diagnostic criteria drafted by psychiatrists, the clinicians with most exposure to patients with "conversion" symptoms are practising consultant neurologists. Appendix I contains a survey undertaken among 167 members of the British Neurological Association that enquired about the terms they currently used in both "formal" and "informal" contexts to describe symptoms not attributed to neurological disease. It shows that "conversion" ranked well below such favourites as "hysteria", "psychogenic" and "functional" among their preferred diagnostic terms, while confirming that these presentations represent an area of substantial clinical need.

The work described in the rest of the thesis reflects my initial expectation that, given this need, a syndrome based on "conversion hysteria" might be defended despite controversy. However, in order to consolidate it as a diagnosis, new work seemed to be needed into its scope, its validity and its pathogenesis.

While "hysteria" continues to exercise great interest among medical and social historians, the history of "conversion" has been wholly neglected. Chapter I presents a history of the clinical use of hysterical "conversion" which needed to begin not with Freud, but in the
18th century. It demonstrates how modern diagnostic usages of conversion stand against
distinct pre-Freudian and post-Freudian usages and have also been influenced by these.
Understanding of how the current diagnostic use of 'conversion' came about will illustrate its
strengths and weaknesses as a diagnostic concept.

Within recent clinical history, argument about the standing of traditional hysteria and
newly coined syndromes such as somatisation disorder have placed great weight on the
evidence of follow-up studies. These have suffered from a range of methodological
idiosyncracies and, because they have been hostile to the integrity of hysteria as a diagnosis,
or have been concerned to justify alternatives, have failed to distinguish clear criteria by which
diagnoses of a conversion disorder are likely to breed true. In chapter 2, a 10 year follow-up
study is described, conducted on a cohort of 79 patients who had received unusually detailed
initial assessments at a neurological hospital in the course of investigation for symptoms which
were not attributed to neurological disease. Neurological, psychiatric and psychological
assessments were reviewed in the light of the outcome of the original symptom, of whether
neurological disease supervened, and the subsequent treatment history. As well as clarifying
factors at presentation that might predict symptom persistence, diagnostic change and future
use of medical services, this longitudinal study indicated that symptoms attributed to
conversion had a distinct natural history. However, their association with other symptoms
attributable to somatisation suggested that the current range of diagnostic options may not be
optimal.

The case that patients having the disparate variety of presenting symptoms shown by
those with conversion disorder merit the same diagnosis would be greatly helped if a
pathology common to all of them could be demonstrated. "Conversion" has implied a shared
psychogenetic mechanism, and this has also been the rationale of those promoting
"dissociation" as an alternative. Such arguments would be greatly helped by an experimental
demonstration of a specific psychological mechanism or disability, otherwise it is hard to see
why one term should be preferable over another, or to know what its scope should be.
Chapter 3 describes two sets of experiments that attempted to show defects in attention
among conversion patients in controlled comparisons using the paradigms of dual task
performance and perceptual span in newly computerised versions. While neither is
conclusive in identifying a common deficit, they provide some evidence of subgrouping
within this population.

Each of these studies has definite implications for diagnostic reform. The survey of
clinicians demonstrates the practical need for useful and reliable categories that would apply
to many thousands of patients. The longitudinal survey, confirming the clinical reality of
chronic pseudoneurological symptoms, provides evidence that associates these with the
psychopathology of personality disorder rather than affective illness, and suggests they are
associated with longstanding behavioural predispositions in the ways hypothesised by
proponents of "abnormal illness behaviour" and "somatisation disorder". Conversely, the
failure of that study to discriminate absolutely between the syndrome of conversion disorder
and that of somatisation disorder is complemented by the failure of the attempt in chapter 3 to identify a distinct psychological pathology common to patients with conversion symptoms. Neither of the clinical studies can represent a definitive answer to the questions of diagnostic validity that informed them. However, the research summarised here will suggest future avenues of research focusing on the behavioural antecedents of conversion symptoms, and their interrelationships with other forms of somatisation. These are summarised at the conclusion of the thesis.
Chapter 1: A historical study of hysterical conversion.

Aims:

• To survey past usage of hysterical conversion.
• To analyse how its meaning has changed with context.
• To assess contemporary implications of this history for practice and research.

1.1 Introduction.

Although the history of "hysteria" is continuing to be rewritten (Veith, 1965; Trillat, 1986; Micali, 1995) the history of the concept of hysterical conversion or conversion hysteria has attracted neither an independent study, nor significant attention within these larger works. It is generally assumed that "hysterical conversion" has been a recent, Freudian invention. A preliminary survey suggested its history was significantly longer than Freud himself believed, and that it can be divided into four distinct phases. In each of these periods, from the late 18th. century until the present, "conversion" was used to bring an apparently disparate set of clinical features together under a common label. These usages have differed considerably in the extent to which they were generally adopted, and in the clinical syndromes to which they referred. However, in each instance, the role found for hysterical "conversion" results from relatively complex clinical logic. It will be argued that the role of hysterical conversion has always been to link cases with disparate physical features, so that these became assimilated within a way of thinking that found them difficult to accommodate in any other way. For each of the four systems in which attempts were made to introduce and develop the concept of hysterical "conversion" (18th. century nosology; Freud's early writings; post-Freudian psychoanalysis and 20th. century psychiatric classification), an attempt will be made to analyse factors in contemporary clinical thinking likely to have shaped each of these usages of "conversion".
1.2 The Conversion of Disease: Hysteria in the age of Ferriar

It was Richard Hunter and Ida MacAlpine (1963) who drew attention to the use made by the English physician John Ferriar of the term "hysterical conversion". Recent research indicates that prior even to this, the term "conversio" was employed in the Middle Ages to refer to a propensity shown by what was then termed "suffocation of the womb" to develop into other diseases following a "crisis" (Jacquard & Thomasset, 1988, p.175). This use of "conversio", to be found among fifteenth century commentators on Avicenna such as Jacques Despar, anticipated the meaning bestowed upon "hysterical conversion" by Ferriar once "hysteria" had become a substantive disease.

1.21 Ferriar's description of hysterical conversion

In the second volume of his "Medical Histories and Reflections" (1795) Ferriar discussed a general concept of the "conversion of diseases" by which:

"new symptoms arise ...which require a different designation, and which either put a period to the original disorder, or combining with it, alter the physician's views regarding the prognostics, or the method of cure" (p.1).

Conversions could arise in many contexts: when one recognised disease develops into another; when it predisposes to the subsequent development of another; or through the ill-advised therapeutic suppression of a disease. (On the last, Ferriar deplores for instance how hysteria and hypochondriasis would convert, perhaps fatally, into scirrus of liver, dropsy, apoplexy, or palsy through medicinal use of "spirituous liquors" (p.56.).) The disease of hysteria, however, enjoyed an anomalous position alongside other diseases. It had its own characteristic symptoms, and like others could itself arise through conversion from other diseases, e.g.

"Fevers often terminate into hysterical disorders, especially in women; men too, are sometimes hysterically inclined, upon recovering from typhus, for they experience a capricious disposition to laugh or cry, and a degree of the globus hystericus" (p.37).

However, it was in the very nature of hysteria that, once established, conversion could supervene while hysteria was still undeniably present:

"the histories of usual chronic diseases, are absolutely cases of conversion: to be convinced of this we need only refer to SYDENHAM's description of hysteria and gout" (p.69).

This characteristic propensity to repeated conversion was different in either of those two cases:

"There is a strong resemblance between hysteria and gout, in the power of counterfeiting different diseases, but with this material distinction, that the hysterical representations are commonly void of danger, while those produced by gout are often more dangerous, than the simple disorders they imitate"(p.23).

Thus, in contrast to other instances of the conversion of diseases, in
"hysterical conversion ... the body possesses a power of representing the most hazardous disorders, without incurring danger; of counterfeiting the greatest derangement in the circulating system, without materially altering its movements; of producing madness, conscious of its extravagancies, and of increasing the acuteness of sensation, by oppressing the common sensorium" (p. 12).

Ferriar reflects:

"Nature, as if in ridicule of the attempts to unmask her, has in this class of diseases, reconciled contradictions, and realised improbabilities, with a mysterious versatility which inspires the true philosopher with diffidence, and reduces the systematist to despair" (p. 12).

1.22 Ferriar and the limitations of classical taxonomy.

During these remarks, Ferriar appears to be in dialogue with several protagonists. One of these is Sydenham, with whom Ferriar had a special sympathy having devoted his own MD to Sydenham’s greatest single contribution to medicine, the description of the natural history of variola (smallpox). Sydenham’s interest in clear description and precise classification is echoed in Ferriar, as is his interest in the peculiarly taxing nature of what Sydenham still called the "hysteric affection". Sydenham’s own descriptions of hysteria had been at odds with Ferriar’s in several significant respects, however. The clinical description of the "hysteric affection" offered by Sydenham (1848, p.85 et seq) had had three major components: a set of characteristic signs and symptoms (which included the voiding of large quantities of limpid urine, shifting sensations of cold, and odorous eructations); a chameleon like capacity to imitate other conditions; and a tendency to be associated with disturbances of the "passions" (this last being subject to much misinterpretation by later historians). Although Ferriar was not primarily concerned with the clinical description of hysteria, the passages already quoted from his treatise imply he was in agreement concerning its visible signs, while differing somewhat from Sydenham with regard to its consequences for the sufferer. Ferriar’s allusion to the lack of "material" alteration during hysterical imitation contrasts with Sydenham’s belief that considerable post-mortem collections of fluid were compatible with the diagnosis. And while Ferriar points to the dangerlessness of hysteria, Sydenham warns of the grave dangers of failing to diagnose it (Sydenham, 1848 p.111). The clue linking such differences is, of course, Sydenham’s views on the pathogenesis of hysteria, which he put down to "the faulty disposition of the animal spirits" (p.91). The ability of the animal spirits to selectively inhabit regions of the body accounted for the polymorphous nature of the hysteric affection’s symptomatology and course: their quasi-humoral status made it fitting that during their derangement "the mind sickens more than the body" (p.88). Ferriar offers no opinion on animal spirits, or indeed the pathology of hysteria at all.

However, other figures loom between Sydenham and Ferriar, notably William Cullen, Professor of Physick at Edinburgh while Ferriar was a student there in the 1770’s. Cullen is renowned for his contributions to nosology and for his introduction of the concept of the
neuroses as a broad class of conditions, among which he was to place "hysteira". As a nosologist, Cullen had been impressed by the intricate taxonomies of such continental contemporaries as Linnaeus and Sauvages, becoming more ambitious for the formal completeness of his diagnostic schema than any of his predecessors at home. Cullen was also highly critical of any attempt to explain illness in terms of invisible "animal spirits", and sought to explain apparently sympathetic action between remote parts as the result of nervous action. In this, Cullen was anticipated by Whytt (French, 1969 ch.4), but whereas Whytt was willing to attribute the greater part of "nervous disorder" to hysteria (together with hypochondriasis), Cullen was anxious to restrict the scope of hysteria as far as possible, complaining (with respect to hysteria and hypochondriasis) of the "great impropriety that almost every degree of the irregular motions of the nervous system has been referred to one or other of these two diseases" (Cullen, 1791 p.103).

Cullen made no positive contribution concerning the pathology of hysteria. Instead, he refers to its abdominal origins in the 1860s in a manner reminiscent of Sydenham (Cullen, 1797); in his "nosology" he followed Sagar and Sauvages in placing hysteria within the order of "spasmi" where (along with epilepsy, chorea, asthma, tetanus, asthma, dyspnea, pertussis, colic, cholera, diarrhea and diabetes) it is attributed to "irregular motions of the muscles or muscular fibres" (Cullen, 1827 p.74); while in his "first lines on the practice of physick" the "proximate cause" of hysteria became "a turgescence of blood in the uterus, or in other parts of the genital system" (Cullen, 1791 p.106).

However, as Foucault has pointed out, in Cullen's era anatomical considerations were of secondary importance in the identification of a given disease: "what communicates the essential "body" of the disease to the real body of the patient are not, therefore, the points of localization, nor the effects of duration, but, rather, the quality" (Foucault, 1973, p.12). According to Cullen, the essential quality of hysteria was one of "mobility". It was mobility that permitted one symptom to be rapidly substituted by another in the course of the disease, and the greater mobility of hysteria that distinguished it from hypochondriasis. This degree of mobility was itself a reflection of the particular "sanguine" constitution that Cullen believed predisposed patients to hysterical illness before the operation of any proximate causes (Cullen, 1797, p.59). Although Cullen, scrupulously insisting on the separateness of hysteria from other conditions and the folly of overdiagnosis, had been reluctant to confer it powers of imitation, contemporaries such as Gregory did not hesitate to stress an imitative quality common to the cases of hysteria seen at the Edinburgh infirmary (Risse, 1988).

Ferrier's purposes become somewhat clearer when placed in this context. He acknowledged himself that his interest in the way diseases may substitute one for another had not been new - his use of "conversion" here corresponding to classical concepts of "epigenesis" or "metastasis". Making such transformations inherent to the character of hysteria, however, certainly was novel. Yet the development was a natural one once the pursuit of nosology had emphasised the separability of morbid entities rather than
continuities, and had done so on the basis of newly conceived rational relationships rather than original nosographic discoveries (cf. Faber, 1923).

In Ferriar's writing, therefore, one finds little interest in the pathogenesis of hysteria, and hesitation as to how far any individual symptom might be characteristic of it. Instead, the qualities formerly subsumed under the notion of "mobility" are used to describe a unique property of the formal relationship enjoyed by hysteria with respect to other diseases, and this is identified with "hysterical conversion". The propensity for "imitation" that Sydenham had recognised, but Cullen had evaded, became the essential quality of hysteria within a medical discourse that valued taxonomic adequacy before other considerations.

While it dealt neatly with a challenging anomaly, Ferriar's concept of hysterical conversion appears not to have been adopted by others. Contemporaries such as Pinel, who cites Ferriar's volume in introducing his own "Treatise on Insanity" (Pinel, 1806, p.1), were nonetheless aware of it. Its rapid obsolescence is likely to be attributable not only to Ferriar having shared preoccupations peculiar to the Edinburgh school, but to a concurrent shift in interest away from the ordering of disease on the basis of formal resemblance, towards a nosographic renaissance founded more securely on pathological understanding. In most areas of medicine, this trend was to see the gradual replacement of the idiosyncratic labels of eighteenth century nosologists with categories that enjoyed a wider consensus. However, the topic of hysteria would remain the object of contradictory and highly personal formulations throughout the following century.

1.3 The Conversion of Affect: Hysteria at the time of Freud

Freud's re-adoption of "hysterical conversion" in the 1890s followed nearly a century of disuse, and it was taken for a complete innovation. Indeed, the phrase now stood for a concept quite distinct in its referents, its character, and in its legacy.

1.31 Freud's usage of hysterical conversion.

Freud adopted the term hysterical "conversion", (Ger. "Konversion") at the beginning of his career as a psychopathologist. In formulating the psychodynamics of hysteria, he reserved "conversion" for a form of "defense" that was (almost) exclusive to hysterical patients, and which accounted for the pathogenesis of what were in effect the least stereotyped of their physical symptoms. He introduced it:

"In hysteria, the incompatible idea is rendered innocuous by its sum of excitation being transformed into something somatic. For this I would like to propose the name of conversion" (Freud, 1894 p.49).

The process of conversion involved a separation of affect associated with the "incompatible" idea, but not necessarily a disappearance from consciousness of the idea.
itself. Freud quickly came to equate an "incompatible" idea with a traumatic one having an exclusively sexual content. Contrary to views then prevailing on predisposition to hysteria, he was not willing to preclude anybody from exhibiting hysterical symptoms so long as the content of this idea was sufficiently traumatic. Freud stressed that the symptoms that resulted were primarily fixations of physical patterns relating directly to events at the time of the patient's trauma. It was uncommon, therefore, for the relation between the determining cause and the hysterical symptom to be a merely symbolic one, an outcome Freud claimed would be far harder for the psyche to achieve. In fact he explicitly distinguished between symbolisation and conversion as psychological mechanisms:

"the mechanism of symbolization, which has its place, in some sense, midway between autosuggestion and conversion" (Breuer & Freud, 1895, p.180).

According to Freud and Breuer, a wide range of symptoms were attributable to conversion: various pains and neuralgias, anesthesias, contractures, paralyses, vocal and motor tics, paraphasia, haemorrhage, chronic vomiting and anorexia, deafness, visual and olfactory hallucinations are all cited in the preliminary discussion, or are to be found among the case histories, of the Studies in Hysteria (Breuer & Freud 1893, 1895). Further inspection of that work reveals interesting discriminations between symptoms according to the mechanism of production, however; for instance vomiting, paresis, selective aphasia and a tic are quoted as instances of the immediate impact of trauma (p.4) while pain appears to reveal a more symbolic link to its origin with some frequency (p.178 ff.). Moreover, "classical" hysterical symptoms of hemianaesthesia, contraction of the field of vision and epileptiform convulsions were thought impermeable to analysis in terms of past experience (Breuer and Freud, 1893 p.5) and were not attributed to conversion at all.

Freud continued to refine his views on the pathogenesis of hysteria during the 1890s, and to refer to a mediating process of "conversion" that involved somatic displacement of traumatic affect. Although he did not explicitly redefine it, the nature of the provoking "incompatible" idea changed, while "conversion" itself played a progressively less exclusive role in Freud's accounts of the pathogenesis of hysteria after 1900. Indeed, as Freud continued to describe and review the underlying mechanisms of normal as well as pathological psychic life, "conversion", like patients having hysteria, received less and less of his attention.

In 1909, Freud introduced a new use for "conversion", one that has been particularly influential. He used the term "conversion hysteria" to designate a particular hysterical syndrome, in contradistinction to the "anxiety hysteria" he diagnosed in his patient "Little Hans":

"There exist cases of pure conversion-hysteria, without any trace of anxiety, just as there are cases of simple anxiety-hysteria, which exhibit feelings of anxiety and phobias, but have no admixture of conversions" (Freud, 1909b, p.116).
From then on, references in Freud's writing to the entity "conversion hysteria" greatly exceed any to conversion as a mechanism.

1.32 Freud and the scope of the anatomico-clinical paradigm.

The breadth of Freud's personal culture ensures his thinking on many topics does not lack antecedents. As in Ferriar's case, his formulation of hysterical conversion can be represented as a way of resolving tensions peculiar to medical thinking of the period, achieved by a synthesis of much of his immediate predecessors' work on hysteria. By the 1890s, a great deal had been done to further the programme of linking clinical syndromes to underlying tissue changes - the so-called anatomico-clinical method (Lain-Entralgo, 1961). Nosology adjusted accordingly as superficial levels of description were abandoned, new syndromes delineated, and the inevitability of evolution within diagnosis was more readily recognised. However, many disparate clinical phenomena were still subsumed under the rubric of hysteria, and the anatomico-clinical method had spurred several potentially incompatible lines of thought. Essentially, hysteria had been cast as a frankly gynaecological disorder, as a neurological disease, and also as a condition that was peculiarly resistant to anatomico-clinical explanation. If the first two cast shadows across Freud's conception, the third can be presented as its heart.

Until very late in the 19th. century, a large body of medical opinion held that where hysteria was diagnosed, disease of the female reproductive organs would be found (cf. Hollender, 1983). Such attributions took various forms, the uterus, ovaries and accessory organs all being variously implicated, but were founded upon an apparent wealth of evidence e.g. "respecting the origin of hysteria, I have been led to believe, from the symptoms observed in these two hundred cases ... that hysteria originates in the ovaria" (Lee, 1871, p.23). These beliefs were by no means exclusive to gynaecological specialists, however, as Griesinger's strictures to psychiatrists about the importance of genital lesions in the onset of hysteria confirm (Griesinger, 1867). Similar ideas of the reflex effects of genital changes figured in Benedict's (1868) description of "libido", the precursor for Freud's concepts of erotogenesis and his own libido theory.

While gynaecological accounts pursued a very old line of association (Merskey & Potter, 1989), concurrent attempts to link hysteria to physical changes in the nervous system also had a significant pre-history (Merskey, 1983). In the 19th. century, a crucial advocate of hysteria as a neurological disease was Paul Briquet, whose treatise on hysteria (Briquet, 1859) had been strangely neglected by modern historians (Mai & Merskey, 1980). Presenting hysteria as a "neurosis of the brain", Briquet defined it in terms of a set of neurological symptoms that should yield to pathological investigation like those of any other disease. Subsequently, Charcot, like leading neurologists elsewhere, accepted hysteria as a neurological syndrome, while discriminating between "major" and "minor" varieties that resembled epilepsy and chronic paralyses respectively (Charcot & Marie, 1892). He had to
concede defeat in the search for structural pathology, attributing hysteria instead to a "functional" lesion whose site was nevertheless expected to be anatomically specific according to its effects (Charcot, 1889, lecture XXI). The notion was a forerunner of Freud's own model of psychic physiology (Freud, 1895b), which was the framework that gave his mechanistic theory of hysterical conversion its shape (Knight, 1984).

The third line of study that the anatomico-clinical method prompted was the most decisive. It led to an explicit association not only with the absence of structural pathology, but with symptoms quite inconsistent with the effects of any other known pathology. Thus, in order to explain pain that was not commensurate with the recognised effects of local joint disease, Brodie had introduced the concept of "local hysteria" (Brodie, 1837). Within his neurological model, Charcot had recognised a necessary contrast between the pattern of symptoms such as seizures, paralysis, anesthesia or visual failure that arose in hysteria and the pattern characteristic of other conditions. It was in elaborating these insights that Freud began his own study of hysteria. As he extended Salpêtrière teaching in "Some points for a comparative study of organic and hysterical paralyses" (Freud, 1893) he notes

"since there can only be a single cerebral anatomy that is true, and since it finds expression in the clinical characteristics of the cerebral paralyses, it is clearly impossible for that anatomy to be the explanation of the distinctive features of hysterical paralyses",

hence his famous comment that:

"hysteria behaves as though anatomy did not exist" (Freud, 1893 p.168; also 1888 p.49).

After observing the singular challenge hysteria posed to pathological explanation, Freud proceeded firstly to formulate hysteria in terms of its etiology rather than reliable clinical findings, and then implicitly to redefine it to ensure that one etiology would be common to all cases. This remarkable move set him apart from nearly all his predecessors, yet its significance has scarcely been acknowledged. One notable exception has been K. Codell Carter, who felt that these ambitions were prompted by the recent successes of "germ theory" within German medicine (Carter, 1980). However, there is no documentary evidence of a direct influence here, while Freud's personal attempts to define syndromes within neurology do record how his preoccupation with the issue of etiology arose. (In fact Freud's substantial neurological writings (Miller & Katz, 1989) have remained an area of extraordinary neglect, even on the part of writers who have argued for a "biological" reinterpretation of his thinking (e.g. Sulloway, 1979)). At the same time as Freud was compiling his "Studies in Hysteria", he was busy with his equally substantial work, "Infantile Cerebral Paralysis" (Freud, 1897). He took a critical approach to that field, e.g.

"...it is neither equated to a pathologico-anatomical nor to an etiological entity. It is therefore probable that even clinically the term can only claim the value of a temporary entity that may soon be abandoned in favor of certain more coherent and possibly etiologically well-determined disease pictures" (Freud, 1897 p.21).
Despite making descriptive innovations, Freud admitted himself unable to reorganise infantile paralyses on the basis of etiology alone.

From his earliest writings on psychopathology, however, it is apparent that he came to the neuroses with the intent of linking each of the "actual" or "psychoneuroses" that he defined to a specific etiology. As a psychopathologist he could enjoy an extraordinary freedom to realign descriptive boundaries according to unique causes, asserting his faith in 1895 that "when a mixed neurosis is present, it will be possible to discover an intermixture of several specific aetiologies" (Freud 1895a, p.113).

In the particular case of hysteria, a "psychoneurosis", Freud was aware that his clinical predecessors, including Charcot, had attributed its occurrence to multiple causes, constitutional and environmental, physical and psychological. A major exception was Moebius, who wished to define all hysterical symptoms as psychologically induced, either by emotion or by idea, (Moebius, 1894) and from whose ideas Freud was naturally keen to distinguish his own. He did so through a revision of a form of hysteria Charcot had already set apart on account of its characteristic aetiology. In Charcot's concept of "hystero-traumatism", the particular pattern of physical symptoms that the (usually male) patient sustained following an accident were out of all proportion to injuries actually received, but could be understood in terms of the patient's own expectations of mutilation (Charcot 1889, Appendix I). Freud and Breuer's guiding idea was that the clinical findings in most cases of hysteria, lacking a clear history of physical trauma, could be attributed to a past insult that was emotional in nature, and had subsequently been forgotten. Their "Studies in Hysteria" therefore addressed itself from the outset not to the task of defining hysterical illnesses, but to "discovering their precipitating cause" (Breuer and Freud, 1893, p.1).

Freud's opinions underwent a series of refinements, in which he strove to keep sight of an etiology common to all cases. He postulated the specific mechanism of "conversion" when the variety of traumatic experiences that might precipitate hysterical symptoms still appeared to be relatively wide, and to be potential precipitants of other neuroses also. It allowed cases of hysterical neurosis to share a common predisposing cause in the form of a somatic weakness that favoured the occurrence of conversion (Freud, 1894). Subsequently, as Freud held all neuroses to be sexual in origin, yet distinct according to the form this pathology took, hysteria became identified with one specific form of traumatic precipitant, i.e. the occurrence of passive sexual experience before puberty (Freud, 1896). This idea remained consistent with his original description of "conversion" as a process, an "incompatible" precipitating idea effectively becoming one associated with an earlier experience of this type (Freud, 1896).

However, around 1897 two interrelated changes rendered the concept of hysterical conversion virtually redundant. One was Freud's well-known renunciation of the seduction theory in favour of an emphasis on the ubiquity of infantile sexual experience and the role of phantasy (cf. Freud, 1907). The other was a gradual retreat from attempts to link types of
neurosis to exclusive aetiologies, towards a willingness to discuss the aetiology of neurosis in
general, and the concession that any given case was likely to be multiply determined (Freud,
1906). Hence, in Freud's later discussions of hysteria (Freud, 1908; 1909a) several
alternative etiologies are recognised. At first, the mechanism of "conversion" was adapted to
include the response to "incompatible" ideas taking the form of unconscious fantasy (Freud,
1908), while it lost its exclusivity in the pathogenesis of hysteria. It became quite superfluous
to Freud's 1909 account of the provocation of hysterical attacks, when a new understanding
of libidinal economics entailed that attacks could be brought about "organically" entirely by
"internal somatic reasons" (Freud, 1909a p.231). The concept of conversion became
dispensable once there no longer needed to be an "incompatible idea" to be defended
against.

Freud therefore came to favour the usage referring to the syndrome of conversion
hysteria once he had far less use for conversion as a mechanism. The term's implication of a
consistent aetiology or process was already inconsistent with Freud's own thinking on
hysterical symptoms. This contradiction was to be felt very sharply during the present
century, among his psychanalytic successors as well as official psychiatry.

1.4 Divorcing Conversion from Hysteria: the Post-Freudian Aftermath.

"Conversion" was soon to regain a place in the vocabulary of many psychoanalysts,
particularly among those sustaining a rather literal and institutionalised adherence to Freudian
procedure within the United States. However, despite appearances, none of Freud's heirs
have used the term in a way comparable to his own.

1.41 The use of Conversion by post-Freudian psychoanalysts

In subsequent analytic history, no single figure managed to appropriate "conversion"
for themselves, and a number of apparently divergent uses arose. Among Freud's immediate
successors, it was to be ignored by Anna Freud in her catalogue of defence mechanisms
(Freud, 1936), but discussed by Ferenczi. Ferenczi readopted the term to distinguish one
subset of physical symptoms from among those he attributed to a mental origin as a result of
"materialisation". The products of conversion were distinct from other materialisations
because they were displacements of a genital impulse only, and were couched in "a peculiar
symbolic language" that he termed the "hysterical idiom" (Ferenczi, 1919, p.100).

A more radical reworking of the concept was offered by Fairbairn (1954), who explicitly
reformulated the concept of conversion in order to make divergences clear between the
"object relations" approach to psychopathology that he promoted and the drive-led approach
identified with Freud. Accordingly, Fairbairn defined hysterical conversion as "the
substitution of a bodily state for a personal problem" (Fairbairn, 1954, p.117). Rather than
accept that symptom formation should be explained in terms of fixed patterns of libidinal

development as Freud's theory of sexuality had proposed, he suggested that "the data upon which the theory of erotogenic zones is based themselves represent something in the nature of conversion phenomena" (ibid, p.121).

However, these authors' acknowledgment of the association of conversion with hysteria notwithstanding, the most enduring revisions of "conversion" have occurred within the American psychoanalytic movement, and have affirmed its estrangement from "hysteria". Already in 1922, Felix Deutsch, an analyst concerned with the treatment of patients with physical symptoms, suggested that "it must be assumed that a continuous conversion process ... takes place in every normal individual" (1959a, p.65) and that "it is during (all) periods of sickness that the conversion process finds an inconspicuous outlet, which is barred at other times" (p66). In fact, the broadening of the concept to include "reactive patterns" such as blushing or perspiration had largely mirrored the clinical scope of Ferenczi's "materialisation" (cf. Ferenczi, 1919, p.96). The hysteric's conversion then:

"differs in so far as its development is based on a constitutional or predispositional inability to ward off emotional tensions ... which ... lead to an inevitable transformation of great amounts of libido into organic manifestations" (ibid. p.66).

Deutsch was to continue to try and formulate how a process of "conversion" could be recognised even in somatisations that were not wholly psychogenic in origin. He was to achieve a mature, if idiosyncratic, position in which "conversion" was typified as a compound process during which

"the symbolisation of external objects leads to a retrojection of these lost objects via sensory pathways onto and into the body where they remain immanent and dormant" (Deutsch, 1959b, p.95).

The divorce of conversion from hysteria, already apparent in this broadened conception, was absolute in a famous review of Rangell (1959). According to Rangell, conversion should apply to all somatisations that:

"speak symbolically, and via body language express a combination both of the forbidden instinctual impulses as well as the defensive forces which bring about the distortions".

He states his conclusion that, due to the range of phenomena it encompassed:

"the process of conversion must perforce be divorced from the concept of hysteria" (Rangell, 1959, p636).

It was a verdict which others such as Sperling (1973) were to uphold within the mainstream of American psychoanalytic thought.

1.42 De-hystericised Conversion and developments of the analytic model.

While Rangell's assertion that his own formulation of conversion "retains in its essence the central meaning of the term as originally intended by Freud" (1959, p.636) aroused little complaint, it can be demonstrated to be mistaken. Rangell's observations, and
others summarised in the previous section, reveal that "conversion" had not only become associated with different complaints, but was a very different type of concept to that described by Freud. This discontinuity seems rooted in the increasing autonomy of the discourse of psychoanalysis from its medical parent. An expansion of psychoanalytic theory, encompassing normal psychology and culture in addition to a large slice of psychopathology, preceded a return of interest to the psychogenic physical symptom. Conversion hysteria became a focus at which the maturing new discourse would restake its perimeter, and readjust its relationship to medicine. Here, as in general, its ability to develop ordering concepts of its own would be critical if it was to steer away from the tramlines of diagnosis and pathology on which medicine was continuing to run.

Freud, in his pioneering contributions to dynamic psychopathology, had continued to emphasise continuities with the mainstream of medical thinking. He remained mindful of the relationship his own work bore to the descriptive psychopathology that, after Kraepelin's example, was dominating the practice of psychiatry. Indeed, Freud continued to make explicit his recognition that theoretical refinements should reinforce rather than defy nosographic distinctions. This is particularly clear for instance when he indicates the implications of his "structural" model of id, ego and super-ego (Freud, 1923) for the whole field of psychopathology:

"Transference neuroses (i.e. hysteria and obsessional neurosis) correspond to a conflict between the ego and the id; narcissistic neuroses (e.g. melancholia) to a conflict between the ego and the super-ego; and psychoses, to one between the ego and the external world" (Freud, 1924, p152; parentheses added).

While Freud's models of psychodynamic operations were in continual evolution, he outlined his view on their relationship to psychiatric description:

"What is opposed to psycho-analysis is not psychiatry but psychiatrists. Psychoanalysis is related to psychiatry approximately as histology is to anatomy: the one studies the external forms of the organs, the other studies their construction out of tissues and cells ... one is a continuation of the other." (Freud, 1917a, p.255).

It is a telling statement, much of the further development of psychoanalysis demonstrating that the analogy was not to be sustained. In its terms, once the task of psychoanalytic research devolved to an extending network of clinicians, competing strains of "histology" arose that often lacked a clear basis for comparison. In general, these also became increasingly estranged from the corresponding distinctions of "anatomy".

Within the orthodox psychoanalytic succession (whose most prolific early representatives were Jones, Abraham and Ferenczi) one finds much initial effort dedicated to matching syndromes with etiological factors according to stages of libidinal fixation. A template of oral, anal, and genital pathologies was sketched by Freud (1907), highlighted by Abraham (1926), and engraved in the textbook of Fenichel (1946). However, it came under constant pressure for revision in the face of local theoretical innovations (such as recognition
of the contribution of different ego states in symptom formation) as well as external concern that such an emphasis on the determining role of early experience was too exclusive.

The etiological ideas attaching to "conversion" were progressively modified in this light. In the terms of libidinal pathology, it was associated with conflicts that were increasingly, and finally exclusively, "pre-genital". Abraham, for instance, opined that a tic could be "a conversion symptom at the anal-sadistic stage" (Abraham, 1921a); Fenichel's textbook vacillates around "the pregenital expression of predominantly genital wishes" (Fenichel, 1946 p.232); but after further elaboration, Sperling firmly declares that "... all conversion is (therefore) pre-genital by nature" (Sperling, 1973, p.569). The importance accorded such instinctual fixations was challenged by the evident influence of the current environment in some situations, not least the widespread occurrence of hysteria in times of war (Abraham, 1921b). Freud's own remarks on "secondary purposes" had recognised the contribution of current influences (Freud, 1909a), although he had always insisted upon the secondary importance of immediate gains relative to primary unconscious conflict. The introduction of the notion of "dependency needs" to account for the pathogenesis of conversion symptoms (Nemiah, 1967) proved a felicitous innovation here. Being instinctually driven (as a consequence of "oral" fixation), but socially satisfied, the concept of dependency needs allowed the evident use made of others by hysterical patients to continue to be referred back to primarily unconscious motives arising out of adverse infantile experience.

Nevertheless, "conversion" emerges here as a grouping that could not be typified in terms of an exclusive pathogenesis, rather as Freud had had to admit defeat in trying to link "hysteria" to acts of "conversion" alone (cf. section 1.32). It indicates some of the difficulties faced by any attempt to apply psychodynamic insights to explain how a given symptom typically develops. Freud himself had not been insensitive to the problem. His inferences about the "proximate" causes of symptoms had been founded upon the analytic uncovering of meaningful links, by a method similar to that used to trace parapraxies or dreams to their sources. However, it was the most individual and uncommon symptoms that had that opened themselves to interpretation in this way. While these formed the evidence for subsequent generalisations about pathogenesis, it was the most frequently occurring (and uninterpretable) symptoms that gave a diagnostic grouping its stable identity (cf. Freud, 1917b). This had been the reason why, at a descriptive level, Freud's first idiosyncratic cases of "hysteria" had already been unrepresentative of the "hysteria" of Charcot. Its lasting consequence for psychoanalytic explanation was that any correspondence between dynamic psychopathology and a class of symptoms was not only necessarily indirect, but such generalisations themselves were open to re-interpretation. This is the substance of Fairbairn's critique (1954), already quoted on p.20, in which hysterical conversion was selected to demonstrate how even the apparently stable theory of erogenous zones was founded on a questionable transposition of the typical and the particular.
It is not surprising, therefore, to have found progressively less emphasis placed upon universal etiological concepts among analytic attempts to characterise "conversion". Instead, morphologic properties of its symptoms received increasing attention. The symptoms of "conversion" were seen to be not merely meaningful, but to bear their meaning in a characteristic way. Ferenczi's early reference to their "peculiar symbolic language" has been quoted, and is elaborated in the definitions of Fenichel's textbook of conversions as "very specific representations of thoughts which can be retranslated from their "somatic language" into the original word language" (Fenichel, 1946 p.216). This emphasis upon conversion as manifesting peculiar qualities of idiom and grammar prompted qualifications of its clinical scope. Fenichel suggested that: "not all somatic changes of a psychogenic nature should be called conversions because not all are translations of specific fantasies into a 'body language'" (1946, p.236) and introduced the category of "organ neurosis" to take care of cases that, while psychologically induced, were thought impermeable to translation.

In this role as elements in a unique language, conversion symptoms became essentially symbolic. This was a seemingly anodyne extension of Freud's position on conversion symptoms - he had maintained that any symbolic content arose secondarily to an associative connection with a specific precipitant (David-Menard, 1989) - but is quite antithetical to his complex views on symbolism. (The common inference that familiarity with general rules of symbol formation and shared meanings allow a symptom's meaning to be presumed is opposed to Freud's insistence on the primacy of (usually indirect) personal associations in revealing a symbol's content (Forrester, 1980)).

Furthermore, the linguistic analogy presented a paradox. It seemed to consolidate the identity of conversion by presenting it as a natural symptom set, yet it acted to undermine conversion's standing as a grouping in the traditional diagnostic sense. With a redefinition here of the analyst's task as one of translation rather than dissection, the identification of conversion, and determination of its scope, had (as Sperling (1973, p.769) confesses) to depend upon the analyst's interpretative capacity, rather than the objective presence or absence of "pathology".

In this way, psychoanalysis progressed from still seeing the conversion symptom as a "sign" of a circumscribed pathology, as the example of medicine would dictate, towards interpreting it wholly as an intentional "sign", after the model of semiology. This detachment from the essential formulae of medicine resonated with wider trends in psychoanalysis, as a tension surfaced that is apparently inherent between the unfettered pursuit of meaning and attempts to establish categorical and genetic understanding. Rycroft (1966) has argued that psychoanalysis has itself been misunderstood, being a "semantic" discipline that, since inception, has been an exploration into meaning rather than causation: Lacan (1966) has remodelled its theoretical framework to make a "symbolic order" its explicit content, by which signification comes prior to all aspects of subjectivity. The separation of phenomenal and intentional "conversions" from substantive "hysteria" has epitomised this movement.
These developments remain too close to the present for their implications to be clear. They do, however, find a remarkable confluence in the work of one particular analytic writer, Thomas S. Szasz, who was himself sure what they portend. Many readers are surprised to find that his chief polemic, titled "The Myth of Mental Illness" (Szasz, 1972), is devoted exclusively to a critique of conversion hysteria. Yet, given his wish to counter the pathologised thinking he found common to medicine, psychiatry and psychoanalysis, it is fitting he chose this arena, uniquely important to all three.

Szasz' radical scruples forbade him to refer to conversion by name, "the psychoanalytic theory of hysteria (especially the idea of conversion)" having remained "symbols of psychoanalysis as a medical technique and guild". Nevertheless, the alternative he offered was a "semiotical rather than a psychiatric or psychoanalytic analysis of hysteria" (Szasz, 1972, p.29). Echoing other writers that "hysterical conversion is best regarded as a process of translation" (p.102), he elaborated an original theory of the kind of language in which hysterical symptoms were coined. This was a primitive, "iconic" language whose essential feature was that it had a greater power than words to provoke action from others. This language comes into use only if more direct forms of communication are prevented, making the appearance of conversion hysteria as much the responsibility of those around the patient as the patient themselves. A game results between several players "characterised by the goal of domination and interpersonal control" (p.255).

Szasz has provided firm flesh for the semiotic interpretation of conversion, while urging its dissolution as an entity as well as abolition of the term. As he believed any bodily symptom to be potentially an iconic communication, distinctions erected by other analysts between "conversion" symptoms and those that were "psychosomatic" or "organ-neurotic" became quite superfluous. His analysis has done much to encourage a broader shift of focus in psychodynamic thinking, away from historical connections in favour of horizontal ones. However, his faith that his analysis of hysterical conversion could be extended to the entire field of psychiatric "symptoms" seems a curious denial of conversion's unique situation and history.

1.5 "Conversion" rises and falls as a diagnosis

Although the Freudian concept of hysterical conversion had emerged out of the mainstream of 19th century medical thought, his innovative views on the pathogenesis of hysteria were not accepted by the profession at large. The term "conversion" entered general medical discussion of hysteria once "conversion hysteria" was admitted to its diagnostic vocabulary.
1.51 The shifting scope of "conversion hysteria".

Section 1.31 has described how Freud introduced the term "conversion hysteria" to distinguish it as a syndrome from "anxiety hysteria". Although his own interests had then moved away from patients meriting this label, the term was increasingly recommended for general use in descriptive diagnosis. Attempts to standardise diagnostic nomenclature grew throughout Freud's own career, and he had personally made a lasting contribution to 20th-century nosography through his description of "anxiety neurosis" (Freud, 1895a). Whereas that diagnosis was able to enter official taxonomies and see remarkably little change in either its scope or its wording, "conversion hysteria" has been subject to considerable and almost constant amendment. Changes in nomenclature here are summarised in table 1, American usage in particular tending to sanction a separation of "conversion" from "hysteria" promoted among post-Freudian psychoanalysts in the US (cf. section 1.41).

Other trends evident from changes in nomenclature in all these systems include a dropping of the term "neurosis" as well as rejection of the term "hysteria". The term "conversion" itself is preserved in DSM-IV to designate a distinct syndrome as before, while it is subsumed under "dissociative" in the International classification. This significant divergence will be discussed further in section 1.6.

In the past, interpretations of the clinical scope of "conversion" had varied considerably in the absence of clear symptomatic criteria in the early nomenclatures. This corresponded to a good deal of confusion in the clinical literature. Extremely conservative views, restricting conversion hysteria to very few possible symptoms had existed alongside highly liberal ones. The former follow an influential distinction of Alexander (1943) between conversion hysteria proper and other forms of psychosomatic disorder:

"we restrict conversion phenomena to symptoms of the voluntary neuromuscular and the sensory perceptive systems and differentiate them from psychogenic symptoms which occur in vegetative organ systems" (Alexander, 1943 p.206).

As Kendell has pointed out (1983), this could even debar convulsions from the ranks of conversion symptoms if strictly interpreted. Conversely, those who have followed neo-Freudian psychoanalysts in defining conversion syndromes by the presence of symptoms having a certain communicative or symbolic quality can sport vast lists of candidate symptoms. Engel (1970) named over 100 candidate conversion symptoms, referable to all the major body systems.

The criteria provided by diagnostic manuals have been surprisingly wide, despite their tendency to profess increasing stringency with successive editions. The criteria in DSM-III (table 1.2) were remarkable not for the editors' inaccurate assertion that "the definition of this disorder is unique in this classification in that it implies specific mechanisms to account for the disturbance" (APA, 1980 p.244) but because no symptoms were cited as specific to the disorder. This trend was reversed in the the most recent classifications which propose that
distinct syndromes should be delineated around motor, convulsive, sensory and mixed symptoms (table 1.1).

The renewed attention to specific symptoms follows a progressive loss of emphasis on associated features in diagnostic criteria of conversion syndromes. In the criteria of DSM-III (1980; table 1.2), any reference to symptoms' symbolic function had disappeared. This was consistent with the sceptical conclusion of Lewis and colleagues, after an empirical study of the diagnosis of conversion hysteria, that: "for ordinary diagnostic purposes, the criterion of symbolic sense is of little utility" (Lewis et al, 1965 p.281). Criteria B (2) and (3), however, represented a definition of another allegedly characteristic feature of conversion syndromes, "secondary gain". These criteria disappeared in the next revision, DSM-III R (APA, 1987, p.259), not only reflecting further conclusions of Lewis' study that "clearcut manipulation of the environment is by no means an omnipresent feature of cases diagnosed as conversion hysteria" (Lewis et al, 1965, p.280), but Lazare's subsequent dismissal of it in a study evaluating different features for their diagnostic validity (Lazare, 1981).

It is interesting that the criteria that have been retained in DSM-IV (table 1.2) do not appear to have been validated through further study. They leave the demonstration of psychogenesis to apparent precipitation by stress in the presumed absence of voluntary control of the symptoms. Literature is available to challenge these as a secure foundation for diagnosis. For instance, in noting that reports of the discovery of a prior stressor ranged from 52% to 93% of cases, Ford (1985) has recommended that diagnostic weight should only be given to its absence, when he felt organic pathology should be suspected. Moreover, although Ford remains tolerant of the stipulation that voluntary control should be lacking, this too has attracted critical scrutiny. The judgment is based on subjective report, and Miller has pointed out that experimenters' attempts to demonstrate consistent behavioural differences between people who have a disability through a conversion or dissociative disorder, and people who consciously simulate it, have been unsuccessful (Miller, 1986). Jonas and Pope (1985) have tried to argue that, although conversion disorder, factitious disorders and malingering are distinguished according to judgments of consciousness and volition, they occupy an epidemiological continuum and might profitably be studied as a single set of "dissimulating disorders".
**Standard Classified Nomenclature**

5th. revision (A.M.A., 1935) Conversion Hysteria  
6th. revision (A.M.A., 1948) Conversion Hysteria  
7th. revision (A.M.A., 1957) Conversion Hysteria  

**Diagnostic and Statistical Manuals**

DSM-IV (A.P.A., 1994) Conversion Disorder (subtypes:  
with motor symptom or deficit  
with sensory symptom or deficit  
with seizures or convulsions  
with mixed presentation)

**Nomenclature of Disease**


**International Classification of Diseases**

ICD-6 (W.H.O., 1948) 311 Hysterical reaction without mention of anxiety reaction. (includes "hysterical conversion").  
ICD-7 (W.H.O., 1955) 311 Hysterical reaction without mention of anxiety reaction. (includes "hysterical conversion").  
ICD-8 (W.H.O., 1965) 300.1 Hysterical neurosis. (includes "conversion hysteria", "conversion reaction")  
ICD-9 (W.H.O., 1975) 300.1 Hysteria. (includes "conversion hysteria").  
ICD-10 (W.H.O., 1992) F44 Dissociative [conversion] disorders. (These include: dissociative motor disorders; dissociative convulsions; dissociative anesthesia and sensory loss and mixed dissociative [conversion] disorders).

*Table 1.1: Uses of "conversion" in the official classification of hysteria.*  
**(In each case, first and subsequent references are quoted).**
Diagnostic Criteria for Conversion Disorder.

A (*). The predominant disturbance is a loss of or alteration in physical functioning suggesting a physical disorder.

B. Psychological factors are judged to be etiologically involved in the symptom, as evidenced by one of the following:

(*) (1) there is a temporal relationship between an environmental stimulus that is apparently related to a psychological conflict or need and the initiation or exacerbation of the symptom.

(2) the symptom enables the individual to avoid some activity that is noxious to him or her.

(3) the symptom enables the individual to get support from the environment that otherwise might not be forthcoming.

C. (*) It has been determined that the symptom is not under voluntary control.

D. (*) The symptom cannot, after appropriate investigation, be explained by a known physical disorder or pathophysiological mechanism.

E. (*) The symptom is not limited to pain or to a disturbance in sexual functioning.

F. (*) Not due to Somatization disorder or Schizophrenia.

Table 1.2: DSM-III Criteria for Conversion Disorder (American Psychiatric Association, 1980).

(*) Denotes criteria that, with minor modification, remain in DSM-III-R and DSM-IV. DSM-IV adds an additional criterion:

The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation.
Conversion hysteria and nosological need.

The problems encountered by attempts to establish reliable diagnostic criteria for conversion syndromes highlight how they have rarely had a clearly defined set of symptoms or an objective basis for their diagnosis. To understand why "conversion hysteria" and its descendants not only entered diagnostic listings, but have been able to remain there in a way that other categories arising at the same time like neurasthenia or anxiety hysteria did not, attention must also be paid to the diagnostic system of which it was part.

The diagnosis of conversion hysteria had of course been preceded by that of "hysteria". During the 19th. century, hysteria had become progressively more closely identified with the syndromes of neurology and finally exclusively so. The emergence of "conversion hysteria" can be linked closely to contemporary needs of this department of medicine, where the nosological terrain was significantly different to that familiar to us now, and in a state of considerable flux. Although significant local differences are found within European and North American medical culture, both in the degree of integration between neurology and psychiatry and in the forces keeping them apart, it would be fair to state that a gap that had been widening between them was nearing its furthest extent at the time "conversion hysteria" and its variants were being introduced to general diagnostic use (Rogers, 1987).

The organisation of diagnosis reflected this wholesale change, and the development of a corresponding schism within nosology can be traced via the unique record provided by the Royal College of Physicians' Nomenclature of Disease in its successive editions between 1869 and 1948. In the earliest editions, "mental diseases" had been presented as a third subdivision of "diseases of the nervous system", the others being diseases affecting the nerves, and those of unclear pathology (which had included variants of hysteria) (eg. Royal College of Physicians, 1906). However, as the perceived incompatibility of psychological and biological perspectives grew, "mental diseases" were firstly detached from the umbrella of "diseases of the nervous system" in the 1918 edition, while still following immediately behind in their former place (Royal College of Physicians, 1918). In 1948 a further break is evident, "mental disorders" being subsumed under "diseases of the body as a whole" and removed to a distant section of the classification (Royal College of Physicians, 1948).

Although much reorganisation of the diagnostic net occurs on either side of this divide, only two terms actually transfer from "Diseases of the Nervous System" to "Mental diseases". They were "hysteria" and "neurasthenia". These appear under "mental diseases" in 1931, with neurasthenia carrying the warning that "this term is used in various senses and should therefore be avoided" (Royal College of Physicians, 1931). By 1948, with neurasthenia dropped, hysteria has finally become "conversion hysteria" (Royal College of Physicians, 1948 p.84).

That hysteria's transformation into the psychiatric syndrome of conversion hysteria was integral to the distancing of neurology and psychiatry is indicated by concomitant
changes. As the schism deepened, local nosology had had to respect an increasingly firm barrier between "psychiatric" and "neurological" domains, while needing to maintain the integrity of the diagnostic groupings lying on either side of this divide. The availability of an ambiguous category, created to lie just outwith neurology, allowed a more homogenous diagnostic field to be established for those disorders still owned as representative neurological syndromes. As a relatively expendable category, it could be exempt from whatever constraints were being used to level the field of neurological disorder, yet be on hand to accommodate cases that new diagnostic guidelines could no longer place. The choice of a modified concept of hysteria for such a scapegoating role seems inevitable in retrospect. As the paradigmatic "functional" disorder or "neurosis", hysteria had become uniquely identified with those qualities that were being eschewed by neurological nosography, and ripe for such a displacement.

In consolidating its schema, neurological classification increasingly emphasised anatomy as a common point of reference, in preference to inferences of physiological change. The vicissitudes of the important sub-class of "functional disease" in the English literature, and those of "neurosis" on the Continent, reflect this directly. The concept of "functional disorder" had exercised neurologists considerably in the late 19th. century. The first edition of the College of Physicians' classification assigns "functional diseases of the nervous system" a separate section after conditions which could be attributed to diseases of the brain, its membranes, the spinal cord or the nerves. The functional disorders therefore included tetanus, hydrophobia, epilepsy and other convulsive disorders as well as hysteria (Royal College of Physicians, 1869). However, in the second edition of 1884, this section was headed "the names of symptoms and groups of symptoms related with such pathological conditions, or with other conditions not accurately known", a practice continued with slight variations throughout subsequent editions (Royal College of Physicians, 1884). The demise of a subgroup of "functional" diseases was consistent with the criticisms of Gowers, who had sought to minimise the "functional" sector of disorders in his Manual of Diseases of the Nervous System (Gowers, 1886).

Instead, Gowers had discussed a continuum of disease, ranging through the "organic" (where the lesion could be extraneous, as in hemorrhage or tumours); the "structural" (having evident neural pathology, these accounting for most forms of neurological illness); and the "nutritional" (where there is no evident lesion, only "a disturbance of function", whose effects are nevertheless lasting, as in chorea or paralysis agitans). "Functional" disease became a residuum, in which symptoms had to be transient in addition to being lesionless, and to which Gowers wished to admit only hysteria, neuralgia, and some forms of epilepsy (Gowers, 1887). It was hysteria that typified "functional" illness, and as the concept of functional disease became increasingly anomalous within neurological nosology, the simplest solution by which an internal division between structural and functional diseases could be obliterated was to have hysteria, as the purest representative of the latter, removed
from the ranks of neurological disorder. Its transplantation to the growing domain of the mental disorders has sanctioned a largely implicit identification of "functional" with "psychological" rather than "physiological" disorder. The ambiguity by which "functional" may be used in either of these senses, deprecated by commentators such as Rogers (1988) and Trimble (1982), has been an unfortunate but lasting consequence.

In Britain, the concept of "functional" diseases had effectively displaced that of "neurosis" in nosological discussion from mid-century (Lopez-Pinero, 1983). On the Continent, "neurosis" survived to embrace conditions whose symptoms referred to the nervous system, but for which physiological rather than anatomical explanations were sought. Its own fate shows many parallels with that of "functional" neurological disease. Its range was to become increasingly restricted, until by 1890 hysteria was seen as the quintessential neurosis (Charcot, 1889), while many "intermediate" neuroses unrelated to frank pathology became subsumed under a single bridgehead, "neurasthenia" (Lopez-Pinero, 1983). The subclass of neurosis was then largely banished from the ranks of neurological disorder as it became psychologised, via the inception of the "psychoneuroses", with neurotic disorder becoming the province of psychiatry. This transition, requiring the archetypal neurosis of hysteria to become the psychoneurosis of conversion hysteria, was also to permit neurologists to reorganise classification more assertively on the basis of structural pathology or its plainly acknowledged absence.

This analysis, demonstrating that "conversion hysteria" came to label a niche that had been shaped in order to fit wider nosological demands, suggests that the specific features of Freud's theory of conversion may have been quite contingent to the success of conversion hysteria as a diagnosis. They supplied one means by which hysteria could be constructed as an essentially psychogenic condition, that had appeared at a particularly favourable moment. Indeed, some previous formulations of hysteria had been even more exclusively psychological in their explanations (e.g. Carter, 1853), and their surprising neglect by contemporary medicine may have owed much to a greater incompatibility with contemporary nosology. Conversely, it is less of a surprise that Russell Reynolds, when setting out ideas on the psychogenesis of hysteria in 1869 that were ultimately to be highly influential (cf. Merskey, 1983), took pains to differentiate his "paralysis and other disorders of motion and sensation, dependent on idea" from the condition being diagnosed as "hysteria" at that time (Russell Reynolds, 1869 p.484).

As the precedent of using "conversion hysteria" to indicate a syndrome was more widely followed, the psychiatrised concept of hysteria became tied to the theory of conversion. It is interesting that the refinements it has since undergone (cf. section 1.51) showed that psychoanalytic ideas concerning the interpretability of symptoms have enjoyed progressively less influence on the criteria by which it has been diagnosed, in keeping with the argument that they were largely irrelevant to its entry to the diagnostic arena in the first place. Nevertheless, conversion hysteria's psychoanalytic heritage survives in the general
supposition that symptom formation is a reflection of unconscious processes. It can be argued that this residual emphasis upon the etiological implications of "conversion" has distracted attention from those nosological features that have made it a very singular diagnosis.

1.53 Attempts to establish alternatives to conversion hysteria

However much conversion hysteria or its successors have been needed to indicate a syndrome in which neurological symptoms do not represent neurological disease, disquiet with its diagnostic use has been expressed through a search for alternatives during this phase. These have taken two principal and contrasting forms: the redefinition of hysterical syndromes in term of physical findings, and attempts to abandon diagnosis in favour of behavioural concepts drawn from the social sciences. They are briefly reviewed here as they continue to inform debate about diagnosis, and need to be considered by any research hoping to influence clinical thinking in this area.

1.53(i) The resomatization of hysteria. One way in which the issue of psychogenesis can been minimised is to centre the diagnosis of hysterical illness exclusively around somatic changes. Charcot had insisted that hysteria, being a discrete disease, could be diagnosed on the basis of characteristic physical signs such as tunnel vision, hemianesthesia or ovarian hyperaesthesia. Earlier this century, the neurologist Henry Head had popularised the Charcotian concept of "positive" physical signs, such as particular constellations of weakness or anaesthesia, that were believed to be specific to hysterical complaints (Head, 1922). However, there is recent evidence that, when searched for, such anomalous signs can be found also among patients deemed to have bona fide neurological illnesses (Gould et al, 1986; Chabrol et al, 1995), making any individual sign's usefulness as a discriminator appear increasingly doubtful. Similarly, Walters' recommendation that the "regional" character of symptoms, notably pain (Walters, 1961) but also motor and sensory changes (Walters, 1969) was a specific indicator of their psychogenesis has since been countered by Merskey's argument that regional symptomatology can also result from independent physical pathology (Merskey, 1988).

Distrusting the diagnostic significance of any symptom in isolation, a group at St. Louis had instead proposed an influential equation between hysteria and somatic polysymptomatology. A patient having so-called "Briquet's syndrome" (previously "hysteria" (Perley & Guze, 1962) and, more latterly, "somatisation disorder" (APA, 1980 et seq.)) has accumulated symptoms that relate to nearly all the major systems of the body, yet is highly unlikely to proceed to the development of organic pathology sufficient to justify them (Guze, 1970). This search for "objective criteria" for a valid syndrome was consistent with Briquet's own bid a century earlier to normalise "hysteria" as a recognised neurological disease.
History of Hysterical Conversion

(Briquet, 1859; Mai & Merskey, 1991). The eponym was inexact, however, in attempting to found a diagnosis on complaints alone, rather than material findings as in Briquet's own practice.

Its essential claim was of predictive validity, Perley & Guze (1962) having claimed that cases meeting strict criteria had a 90% probability of remaining unchanged, with no medical disorder emerging to account for them. Guze has also supported his proposals with evidence that the syndrome carries a genetic loading (Woerner & Guze, 1968; Guze et al, 1986). However, only a small, if significant, minority of cases of hysterical illness appear to meet its criteria. Although its prevalence has been researched in North American community samples, studies of patients newly presenting to neurology wards with pseudoneurological symptoms have failed to quote the incidence of SD among patients with conversion symptoms (Metcalfe et al, 1988; Fink, 1992).

Despite progressive simplifications, the research criteria for identifying SD have been both demanding and internally inconsistent (Swartz et al, 1987). This process was potentially helped by the refinement of a subset of symptoms that could act as a screening test (Othmer and DeSouza, 1985) which was recognised in the listing of criteria of somatisation disorder in DSM-III-R (APA, 1987). This is described more fully in the following chapter which details its use in a study whose objectives included a consideration of the overlap between SD and conversion disorders.

While these and other attempts to simplify diagnostic criteria for a polysymptomatic hysteria have justified themselves by apparent concordance with Perley and Guze's criteria, none have been rigorously checked for predictive validity in the same way.

1.55(ii) The behaviourisation of hysteria

A later strategy to avoid the need for diagnoses such as "conversion hysteria" has taken the "illness behaviour" of the patient as its focus. This had been defined by Mechanic (1962) as:

"the ways in which given symptoms may be differentially perceived, evaluated and acted (or not acted) upon by different kinds of persons".

Accordingly, conversion hysteria and related syndromes, cast as deviant variants of illness behaviour, became so many subspecies of "abnormal illness behaviour". Pilowsky proposed that in situations where:

"the doctor does not believe that the patient's objective pathology entitles him to be placed in the type of sick role he expects"

and in which

"if the patient is uninfluenced by the doctor's explanation of the problem and the way in which it should be managed ... the doctor may then reasonably conclude that the patient is manifesting abnormal illness behaviour" (Pilowsky 1969, p.349).
Pilowsky's stated aim was to promote "a full and more meaningful understanding" (1969, p.350) as well as a defusion of conflict between doctor and patient. Although his concept of abnormal illness behaviour deliberately encourages a change of focus from signs and symptoms to the patient's behaviour, it distracts from many instances where a parody is presented not only of the behaviour appropriate to illness (in his terms, inappropriate adoption of a "sick role"), but of the bodily manifestations of somatic disease. A blind spot, apparent from a misapplication of Pilowsky's term that occurs in clinicians' discussions, can result. "Abnormal illness behaviour" is frequently adopted as a shorthand for abnormalities perceived in the pattern of a patient's symptoms, rather than in the patient's reaction to explanation as Pilowsky had proposed.

Pilowsky has attempted to operationalise his concept of abnormal illness behaviour by two instruments, a self-report questionnaire (the "Illness Behaviour Questionnaire" or IBQ (Pilowsky & Spence, 1983)) and a structured interview (the "Illness Behaviour Assessment Schedule" (Pilowsky et al, 1983)). The IBQ is described more fully in the following chapter (section 2.32(iii)), where some of its limitations are also discussed in the light of an empirical study the chapter describes.

Pilowsky had intended that his concept would offer a "flexible framework" that could substitute for a range of labels including "hysteria" and "conversion reaction" which he located in a "medical no-man's land" (Pilowsky, 1969, p.347). While professing that his interest was not in how "a patient may be placed in a psychiatric or any other category", (p.347) he has rather inconsistently continued to offer guidelines on the "diagnosis" of AIB instead (Pilowsky, 1971; 1978).

Proponents of abnormal illness behaviour appear to be pulling in the opposite direction to enthusiasts for somatisation disorder in their mutual wish to avoid reference to conversion hysteria (and other apparently ill-defined syndromes such as hypochondriasis). While the former overtly seek to eschew diagnosis, the latter have sought reform through a more rigorous approach to diagnosis within the existing framework. However both traditions share an important characteristic. Neither grants any special priority to symptoms suggestive of neurological disease over symptoms that resemble those of disease in other body systems. (This is especially true of the earliest definitions of Briquet's hysteria where neurological symptoms were only one of ten symptom categories (eg. Gatfield & Guze, 1962): subsequent simplification has left pseudoneurological symptoms as one of only four specified types of candidate symptom for somatisation disorder in DSM-IV (APA, 1994)). Even if these formulations fail to provide direct substitutes for the traditional category of (conversion) hysteria, the challenge they represent to the special status enjoyed by pseudoneurological symptoms is something that diagnostic reformers shall have to contend with for some time to come.
1.6 Conclusion: The future of conversion.

The history of hysterical conversion suggests that no one interpretation of the term, including Freud's, deserves an absolute priority. The way in which first Freud and then post-Freudian psychoanalysts had to compromise on the clarity with which 'conversion' was defined as a pathogenic mechanism is mirrored in the extraordinary flexibility of diagnosticians' descriptive criteria. The argument that much recent usage has been a response to wider demands within the diagnostic system highlights a parallel between recent practice and Ferriar's eighteenth century formulation, in which 'hysterical conversion' was required to account for otherwise uncomfortable discontinuities in diagnostic practice.

As "hysteira" has fallen into diagnostic disuse, "conversion disorder" has been retained as a categorical diagnosis in spite of any intrinsic weaknesses because of its undoubted usefulness in accommodating findings and cases that were otherwise hard to place. Nevertheless, it has been a focus of controversy and, despite its retention in recent classifications, it remains under threat. This is very clear in the WHO classification for ICD-10 (table 1.1) where the former "conversion hysteria" and "dissociation hysteira" were merged to produce a list of seven distinct syndromes identified by the prominence of one symptom, viz.: amnesia; fugue; stupor; trance or possession; motor symptoms; convulsions; and anesthesia or sensory loss, with three other categories for mixed and residual cases. The whole sequence is collectively described as "dissociative [conversion] disorders", and it is easy to imagine the "[conversion]" being omitted altogether in a future edition while the phenomena it subsumed stay within an extended menu of dissociative syndromes.

The latest APA classification differs in retaining the rubric "conversion disorder" amid a growing swathe of "somatoform disorders" (APA, 1994). Among these, considerable attention is given to "somatisation disorder" in recognition of its specific criteria and the wealth of associated findings. These criteria are much more specific, and restrictive, than those in ICD-10. However, significant attention is also given to an additional category that had appeared in DSM-III-R (APA, 1987) namely "undifferentiated somatoform disorder". The criteria for this diagnosis are simply that one or more physical complaints which have been present for six months or longer, and which either fail to correspond to physical pathology after investigation or represent impairments grossly in excess of what would be expected from the physical findings. The original description remains unchanged in DSM-IV (APA, 1994).

The introduction of "undifferentiated somatoform disorder" (USD) in this way appears to represent a different threat to the integrity of the "conversion disorder". It has been argued already (section 1.51) that, with the gradual attrition of specific psychological criteria, conversion disorder has come to have a residual function in official classifications. The criteria that remain are weak, leaving it as a clearing house for certain classes of unexplained symptoms. This is not disguised by the recent attempts to be explicit about what its symptoms are (cf. table 1.2). Its differentiation from undifferentiated somatoform disorder is
still weak. Apart from the fact that onset of conversion disorder should be related to a precipitating event, and undifferentiated somatisation disorder needs to be present for six months before diagnosis, the only distinguishing feature between them is the nature of the presenting symptom(s). USD, like conversion disorder hitherto, is described only in terms of "typical" symptoms, such as "fatigue, loss of appetite, gastrointestinal or urinary complaints" (APA, 1994, p.451). No reason is given why pseudoneurological symptoms that have been present for more than six months should be diagnosed as "conversion disorder" rather than USD, although its exclusion criteria ensure, as a matter of convention, that conversion disorder takes precedence. The implicit trend here, therefore, is that conversion disorder could ultimately be absorbed within a family of pan-symptomatic disorders distinguished according to chronicity.

These incipient threats to retention of a distinct diagnosis for pseudoneurological complaints might lead them to be subsumed within an extended family of dissociative disorders or a family of somatisation disorders. As in the diagnostic shifts reviewed in section 1.52 (and indeed the much earlier ones of section 1.12) recent nosological proposals in this area owe much to convention and a desire to make the diagnostic system appear internally coherent. This is at the expense of direct study of conversion syndromes and their clinical and epidemiological similarities to and differences from other disorders with which they have been grouped.

Despite the intent of the WHO and APA classificatory systems to harmonise their schemata with one another, the contradictions between their approaches towards the former conversion hysteria are probably as great as ever. Further understanding seems essential on several fronts if diagnostic reform is to be useful, enduring, and able to satisfy the needs of both classificatory systems. The relationship of somatisation disorder to undifferentiated somatoform disorder needs to be established. It is by no means clear that the latter has a greater resemblance to the former with respect to prognosis than any of the other syndromes listed as somatoform disorders, including conversion disorder. This disquiet is being increasingly felt, eg. by proponents of a three-symptom 'multisomatoform disorder' (Kroenke et al, 1997) which, by virtue of its more reliable criteria, is more open to objective study of its validity.

Another concerns the natural history of pseudoneurological symptoms and whether this is understood sufficiently well to enable comparisons with the history of other physical symptoms occurring in the absence of systemic disease to be made. Yet another is whether presumptive reference to "dissociation" in patients with pseudoneurological symptoms is borne out. The historical emphasis on diagnosing hysterical syndromes as being either of conversion type or dissociation type has led to a deficiency of data on how commonly the features of each actually occur together or even substitute for one another (Mace, 1993). Moreover, the move to substitute references to the pathogenic mechanism of "dissociation" in place of one of "conversion" requires some demonstration of an underlying mechanism.
that is "dissociative" yet widespread among patients with "conversion" symptoms for it to be justified (Krüger & Mace, under submission).

If future diagnostic reform is to reflect knowledge rather than prejudice, more investigation of these relationships is required. This is the context of the studies to be described in the remainder of this thesis. Clearly, the question of the relationship of conversion syndromes to somatisation disorder can only be resolved by further work on the long term prognosis of either of these, prognostic validity having been the raison d'être of the latter diagnosis (Gatfield & Guze, 1962). The work in chapter 2 represents an attempt to clarify some outstanding questions concerning the prognosis of chronic "conversion" symptoms.
Aims of the study:

- To assess the prognosis over 10 years for patients undergoing investigation at a neurological hospital with somatic symptoms attributed to "hysteria".
- To investigate which factors from initial physical, psychiatric and psychological examination were prognostically significant with respect to symptomatic and diagnostic outcome on follow-up.
- To assess how these outcomes correlated with actual use of medical services, and whether different uses of services reflected initial differences with respect to a measure of "abnormal illness behaviour".

2.1 Background to the study

2.11 Previous longitudinal studies of hysteria.

There have been at least 11 significant follow-up studies of patients diagnosed as having hysterical illnesses in the last 50 years. These are summarised in Table 2.1. Despite this apparent wealth of evidence, it is not simple to draw general conclusions concerning the prognosis of conversion symptoms. Studies that have aimed to be naturalistic, perhaps with hysteria as one of several conditions being observed over a long time-scale (eg. Ljungberg, 1957) differ in key respects (wide inclusion criteria; longer follow-up; a broader range of assessed outcome) from an intervention study in which follow-up was intended to assess the impact of a planned intervention (eg. Hafeiz, 1980). Other studies have been conducted in order to assist arguments about the diagnosis of hysteria, their design reflecting these interests. Thus Gatfield & Guze (1962), keen to argue for an alternative syndrome, paid particular attention to the profile of symptoms in each member of their cohort. whereas Slater and Glithero (1965), out to pass judgment on all diagnoses of "hysteria", paid little attention to heterogeneity within their group of neurological patients at several crucial points of their study - within the initial sample; the quality of follow-up; and in the results obtained.

In fact, no two follow-up studies have been truly comparable in their subjects, methods and measures. These inconsistencies also compromise attempts to generalise about the prognosis of conversion symptoms. For instance, a trend for reports to be more optimistic about the prognosis overall is counterbalanced by a failure of more recent studies to match the lengthier follow-up intervals of the previous generation of studies. Furthermore, as a result of Slater's contentions about undiagnosed neurological disease, interest in diagnostic outcome has competed with statements about symptom persistence, with some studies
2: A 10 year follow-up study reporting exclusively on one of these clinical aspects (eg. Watson & Buranen (1979) on diagnosis; Chandrasekaran (1994) on symptoms ). At the same time, little attention has been paid to an increasingly important outcome variable, patients' subsequent use of other services. This is surprising not only because of the increasing influence of health economics on research, but the fact that either of the conceptual initiatives seeking to replace conversion hysteria ('somatisation' and 'behaviourisation' as discussed in section 1.55) have highlighted a presumed tendency for patients with pseudoneurological symptoms to make higher use of services than others. No study has looked at this specifically, although Fink (1992) includes patients with chronic pseudoneurological symptoms among a cohort of 'chronic somatisers' whose use of hospitalisation was reported in detail.

Apparent disparities in outcome may derive from the population from which patients were drawn. Cases from a psychiatric setting have been reported to retain their diagnosis more reliably than those from neurological hospitals (Lewis, 1975; Chandrasekaran et al 1994), and the rapid recovery of most virgin referrals contrasts with the relatively intractable symptoms of patients seen at tertiary referral centres (Ford, 1985; Hafeiz, 1980). Indeed, the propensity for chronic symptomatology to persist in a significant sub-group of cases underpins the diagnostic proposals made by Guze and colleagues, by which past treatment and hospitalisations is one feature of the apparently stable diagnosis of "Briquet's syndrome" and its successor, "somatisation disorder" (Guze, 1975; APA, 1980).
Table 2.1: Follow-up studies of patients with "conversion hysteria/disorder".

<table>
<thead>
<tr>
<th>Investigator(s)</th>
<th>Year</th>
<th>Period</th>
<th>Initial n =</th>
<th>Final n =</th>
<th>Ages</th>
<th>% female</th>
<th>% disabled</th>
<th>History</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carter</td>
<td>1949</td>
<td>4-6yrs.</td>
<td>100</td>
<td>90</td>
<td>16-40</td>
<td>60</td>
<td>21</td>
<td>(brief)</td>
</tr>
<tr>
<td>Ziegler &amp; Paul</td>
<td>1954</td>
<td>20-25 yrs.</td>
<td>66</td>
<td>62</td>
<td>mean 29</td>
<td>94</td>
<td>57</td>
<td>?</td>
</tr>
<tr>
<td>Ljungberg</td>
<td>1957</td>
<td>7-23yrs.</td>
<td>401</td>
<td>381</td>
<td>mean 43</td>
<td>61</td>
<td>20</td>
<td>?</td>
</tr>
<tr>
<td>Gatfield &amp; Guze</td>
<td>1962</td>
<td>3-10yrs</td>
<td>37</td>
<td>24</td>
<td>14-67</td>
<td>?85</td>
<td>85</td>
<td>?</td>
</tr>
<tr>
<td>Slater &amp; Glithero</td>
<td>1965</td>
<td>7-11yrs</td>
<td>99</td>
<td>73</td>
<td>mean 39</td>
<td>67</td>
<td>67</td>
<td>?</td>
</tr>
<tr>
<td>Lewis</td>
<td>1975</td>
<td>7-12yrs</td>
<td>?</td>
<td>91</td>
<td>mean 32</td>
<td>73</td>
<td>41</td>
<td>?</td>
</tr>
<tr>
<td>Watson &amp; Buranen</td>
<td>1979</td>
<td>7-10yrs</td>
<td>?</td>
<td>40</td>
<td>mean 40</td>
<td>0</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>Hafeiz</td>
<td>1980</td>
<td>1yr.</td>
<td>61</td>
<td>61</td>
<td>10-35</td>
<td>92</td>
<td>20</td>
<td>(weeks)</td>
</tr>
<tr>
<td>Chandrasekaran et al</td>
<td>1994</td>
<td>5yrs</td>
<td>51</td>
<td>38</td>
<td>16-30</td>
<td>100</td>
<td>37</td>
<td>up to 1yr</td>
</tr>
<tr>
<td>Couprie et al</td>
<td>1995</td>
<td>1-9yrs</td>
<td>60</td>
<td>56</td>
<td>15-73</td>
<td>64</td>
<td>30</td>
<td>most a few days</td>
</tr>
<tr>
<td>Kent et al</td>
<td>1995</td>
<td>4yrs</td>
<td>?50</td>
<td>32</td>
<td>mean 41</td>
<td>75</td>
<td>?</td>
<td>?</td>
</tr>
</tbody>
</table>
Disparities in prognosis do not necessarily reflect sampling factors alone, however. Contradictions between the interpretations of Slater (1965), who argued that much allegedly hysterical illness reflected inadequate diagnosis of underlying neurological disease, and Lewis (1975), who believed the bulk of such patients did not require any subsequent change of diagnosis, indicated that the future course of events might depend upon whether or not concurrent neurological disease accompanied a hysterical disorder. They also indicated that diagnostic standards may vary according to a clinician's training. In Slater's series, only 30% of the sample had had their discharge diagnosis of hysteria confirmed by a psychiatrist (Slater & Glithero, 1965): all Lewis's patients had seen a psychiatrist, if not a neurologist. Moreover, Coryell & House (1984) have commented favourably on prognosis when discharge diagnoses are confirmed by psychiatrists. In a longer-term retrospective study that paid particular attention to mortality among patients with Briquet's and non-Briquet's (i.e. conversion) hysteria, they found that no natural death occurred within 11 years of admission among the 51 patients in the latter group. (These patients' mean age had been 10 years less than Slater's group). Coryell and House also recorded no deaths from neurological illness over 45 years of follow-up, although only a minority of their patients appear to have been referred initially from specialist neurological facilities.

Generalisations about prognosis among patients with hysterical illnesses may be constrained in still other ways by the study from which they derive, rather than necessarily holding true of all patients with a given diagnosis or set of presenting symptoms. The setting (district hospital or specialist referral centre) may influence diagnostic standards as well as the likely degree of intractibility of new patients’ problems at the point of admission. If the length of the follow-up period has differed substantially among patients in a study (as it did in all of the retrospective studies above) this risks providing an unreliable profile of outcome if a significant change (e.g. remission) can occur between the shorter and longer intervals. Other study factors influencing the reported outcome include the manner in which this is assessed. A clinician's opinion may be different from either a patient's self-report, or the findings of a lay interviewer, especially where questions of rediagnosis are concerned. The possibility that a patient or their lay interviewer might be more willing to accept that subsequent medical opinion was in favour of an organic diagnosis is of considerable significance. The study of Slater and Glithero (1965) reported a high incidence of apparently emergent neurological disease (especially “epilepsy”) after Slater had entrusted the follow-up assessments to a non-medical colleague rather than conducting these himself.

The disparate conclusions that previous studies have reached might therefore reduce to differences in their target populations, methods and objectives. This leaves a considerable degree of uncertainty concerning the prognosis for patients receiving a diagnosis of hysteria in a neurological setting with modern standards of neurological and psychiatric investigation. Three factors should assist any future study in its generalisability.
2: A 10 year follow-up study

One is differentiation of symptomatic and diagnostic prognosis when assessing clinical outcome, with clear procedures for their assessment. The second is specificity about initial variables, allowing discrimination between factors associated with good or bad prognoses within the sample. The third is rigorous attention to the interval of follow-up, which has usually been too varied, too short, or both.

2.12 Patient factors affecting prognosis

One of the present study's primary aims was to evaluate the prognostic significance of a range of initial variables. While these have rarely been examined systematically, it is possible to summarise previous literature concerning patient factors that influencing clinical prognosis.

2.12(i) Length of previous history.

The chronicity of patients' complaints at the time of presentation has not always been reported (cf. table 2.1). The persistence of already longstanding symptoms has been catalogued by Ljungberg (1957). Perley and Guze (1962) report their patients having had symptoms for an average of over 20 years at the time of follow-up, indicating patients with the syndrome of chronic hysteria they describe had unusually long prior histories. Conversely, the relatively brief histories of Hafeiz's (1980) rapidly recovering patients suggests a direct positive relationship between length of history and subsequent symptom persistence.

2.12(ii) Type of presenting symptom.

Findings from studies that relate differences in outcome to the type of index symptom are summarised in table 2.2. To provide a useful comparison, the entries for each row were restricted to groups of 5 or more patients, and ordered with those having the most favourable prognosis within the follow-up period at the top of each list. (Comments on smaller groups are below each column in italics). Comparisons between the different columns of the table suggests that the subjects of these studies came from clinical populations that overlap largely, but not entirely. For instance, some symptoms were cited as conversion symptoms in only one of the studies, viz. twilight states (Ljungberg, 1957) dyspnea (Hafeiz, 1980) and pains (Gatfield & Guze, 1962).
## BETTER OUTCOME

<table>
<thead>
<tr>
<th>Study</th>
<th>Aphonia (%)</th>
<th>Twilight states (%)</th>
<th>Amnesia (%)</th>
<th>Astasia-basia (%)</th>
<th>Pain (%)</th>
<th>&quot;Spells&quot; (%)</th>
<th>Dyspnea (%)</th>
<th>Aphonia (%)</th>
<th>Paralysis (%)</th>
<th>Weakness (%)</th>
<th>&quot;Motor&quot; (%)</th>
<th>Vomiting (%)</th>
<th>Fits (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carter (1949)</td>
<td>31</td>
<td>6</td>
<td>22</td>
<td>47</td>
<td>21</td>
<td>21</td>
<td>20</td>
<td>20</td>
<td>10</td>
<td>25</td>
<td>13</td>
<td>21</td>
<td>20</td>
</tr>
<tr>
<td>Ljungberg (1957)</td>
<td>381</td>
<td>6</td>
<td>22</td>
<td>47</td>
<td>21</td>
<td>21</td>
<td>20</td>
<td>20</td>
<td>10</td>
<td>25</td>
<td>13</td>
<td>21</td>
<td>20</td>
</tr>
<tr>
<td>Gatfield &amp; Guze (1962)</td>
<td>n=24</td>
<td>6</td>
<td>22</td>
<td>47</td>
<td>21</td>
<td>21</td>
<td>20</td>
<td>20</td>
<td>10</td>
<td>25</td>
<td>13</td>
<td>21</td>
<td>20</td>
</tr>
</tbody>
</table>

*Seizures poor; Aphonia good* (Aphonia good)

## POORER OUTCOME

<table>
<thead>
<tr>
<th>Study</th>
<th>Sensation</th>
<th>Blindness</th>
<th>Retention</th>
<th>Poor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensation poor; blindness, retention poor.</td>
<td>(Sensation good; blindness, retention poor.)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2.2. Previous studies providing outcome data by symptom.

(Percentages refer to distribution of symptoms within each study)
A consensus emerges here by which aphonia and amnesia appear to carry a relatively good prognosis; paralyses and vomiting less so; and fits and tremor a relatively poor one. It is consistent with the statement included in DSM-IV (A.P.A., 1994, p.456) that prognosis for seizures and tremor is poor, and for aphonia and anesthesia relatively good.

2.12(iii). Number of symptoms.

Perhaps the clearest association made between hysterical symptoms and outcome, however, has concerned the number of reported symptoms. The polysymptomatic concept of hysteria originally propounded by Purcell et al (1951) and developed subsequently as "Briquet's syndrome" (Perley & Guze, 1962; Guze, 1970) and "somatisation disorder" (APA, 1980) links multiple symptoms across many organ systems with poor outcome. Classical conversion (or "pseudoneurological") symptoms formed only one of 10 different symptom categories, a patient needing to have sought medical attention at some time for symptoms in 9 of these to qualify for the diagnosis. Once made, the diagnosis would remain applicable (in the absence of medical rediagnosis for these symptoms) in over 90% of cases over a 10 year period. During this time, conversion symptoms, like other candidate symptoms, were allowed to give way to others, while the polysymptomatic pattern endured. The over-riding concern of Guze's group has been to provide a phenomenological standard for the clinical identification of hysteria, and they appear never to have analysed their very substantial prospective data to see how far other factors apart from polysymptomatology influenced outcome in comparison with controls.

Guze's group has been the only one to consider how the number of symptoms might bear on outcome among patients presenting with pseudo-neurological symptoms. Their outcome data here is indirect, patients having more than one conversion symptom being found more likely to qualify for the diagnosis of Briquet's syndrome than those with single conversion symptoms (Woodruff, Clayton & Guze, 1971) with all that implies for poor long-term prognosis. The outcome for "sub-Briquet" levels of multiple symptomatology of any type has not been addressed by this group in any depth.

Indeed, questionable assumptions about prognosis have been made subsequently even for the full-blown syndrome. As the criteria for Briquet's syndrome and somatisation disorder became progressively less stringent (both in the number of symptoms required, and the minimum number of systems these should affect), it has been a matter of faith that, if modifications to the original symptom checklist maintain a reasonable face validity in diagnostic comparisons, they would also retain the predictive validity of the original category. Although Guze expressed concern lest the stringency of the original 59-symptom check list (Perley & Guze, 1962) be lessened (Guze et al, 1986), the 37-symptom check list for diagnosis of "somatisation disorder" proposed in DSM-III (APA, 1980) has given way to the DSM-IV requirement that only 8 symptoms be present, representing four broad symptom categories.
2. A 10 year follow-up study

(APA, 1994). The attempt to devise a "simple, practical and valid screening tool" that was also adopted by DSM-III-R (APA, 1987) to identify cases having a "high likelihood" of the diagnosis followed the work of Othmer and DeSouza (1985) to devise a list of 7 screening symptoms (cf. section 2.33 below). Research concentrated on its specificity and sensitivity in relation to the full diagnostic criteria so that its predictive validity too has never been put to the test. Indeed, it can be fairly argued that the original Perley/Guze criteria stand alone in having their predictive validity established (Mace, 1997).

2.12 Neuropathology

It is well-recognised that patients with conversion symptoms are also likely to have central nervous system pathology, some estimates claiming this is so in over 60% of patients (Whitlock, 1967; Merskey & Buhrich, 1975). Slater's work is well-known for claiming that independent neurological disorders will supervene in a majority of cases of hysteria if they are followed up (Slater & Glithero, 1965), and implied a change in diagnosis is to be expected whenever symptoms persist. On this view, any evidence of neuropathy on clinical examination should carry a poor prognosis because it meant a definite disorder was already waiting to be diagnosed. However, the co-presence of neurological pathology could influence prognosis without requiring a change of diagnosis if there were other ways by which it made some symptoms more likely to persist. A potentiating effect of this kind might be expected if emergence of conversion symptoms was associated with loss of integrity of the CNS through a variety of means, rather than a specific focal lesion. (The tradition that all "hysterical" symptoms reflect a subtle and hitherto unidentified neurological lesion is discussed in section 1.53(i) of the previous chapter: chapter 3 presents studies consistent with this tradition).

The opinion has been expressed (Toone, 1990) that Slater's findings belong to a past age in which neurological diagnosis was less precise than now, making it unlikely the same results would be obtained even if a new study of patients at a neurological hospital with conversion symptoms copied Slater's practice of relying largely on neurologists' own diagnoses of hysteria. More efficient initial neurological diagnosis should eliminate cases where focal neuropathy would promote symptom persistence and rediagnosis, with an improvement in overall prognosis. While there is some evidence this has occurred, it is not as great as might be expected. There has been a small but progressive tendency for the proportion of "false positives" among studies of hysteria in neurological hospitals to have declined over time. Studies contemporaneous with Slater and Glithero's (Gatfield & Guze, 1962; Raskin et al, 1966) report very high levels of frank neuropathy (c.20%) emerging over significantly shorter follow-up periods, while Watson and Buranen's (1979) small retrospective cohort of more recently diagnosed medical patients had a 'false positive' rate of 25% over 10 years, a small improvement consistent with more careful initial diagnosis by the 1960s.
One confounding factor in tracing this trend is use of modified selection criteria in recent studies in an active attempt to reduce the likelihood of rediagnosis. Couprie et al (1995) actively excluded patients having any history of neurological illness from a cohort of patients meeting diagnostic criteria for conversion disorder (Couprie et al, 1995). On one hand, their clinical assessments still did not prevent 2 patients out of 32 developing neurological disease to account for their symptoms during a 4 year follow-up. This implies further improvements in diagnostic accuracy remain possible. On the other, symptomatic prognosis for the remainder was relatively good with a clear majority of patients recovering quickly. This finding, taken in comparison with studies where patients with previous neurological illnesses were not excluded, provides at least weak evidence for a non-specific link by which prognosis may remain worse in the presence of neurological disease even when this is no longer active or is apparently unrelated to the target symptom.

2.12 (v) Personality.

The impact of personality traits on prognosis among patients with hysterical symptoms has been discussed over a considerable period. Possession of hysterical symptoms had often been confounded with a personality disorder, especially before hysterical personality disorder and conversion disorders were recognised as distinct diagnostic categories (Chodoff & Lyons, 1958). Their clearer delineation permitted recognition that marked hysterical personality traits were only likely to be found among 20% of patients with conversion symptoms (Merskey & Trimble, 1978, following Kretschmer, 1926). It is curious that in Chandrasekaran et al's (1994) all female sample, "hysterical personality" was deemed to have been present in 57% of those who were subsequently symptomatic at 5 years, and in only 18% of those whose symptoms remitted - being the only significant predictor of later symptomatic status in that study. However, their target cohort of 51 patients had been abstracted from 75 originally given clinical diagnoses of "hysterical neurosis", and one of the selection criteria applied in this selection, contrary to usual practice, was presence of "hysterical personality".

Personality deviance, without special reference to hysterical personality traits, was quoted as a poor prognostic indicator by Kraepelin (1913) and this was apparently confirmed by Ljungberg who divided his sample between groups having "abnormal personalities" (44% of his sample, being three times the number he felt suffered from true "psychopathy") and "non-deviating personalities". He observed a statistically significant contrast, symptoms persisting in 30.8% among the "abnormal" personalities but only 17.2% among the "non-deviating" majority (Ljungberg, 1957, p.42).

Little use has been made of psychometric scales assessing dimensions of personality in examining the contribution of personality to prognosis of conversion symptoms. An exception is the study of Watson & Buranen (1979) in which the Minnesota Multiphasic
Personality Inventory was given to 36 men with conversion symptoms and 4 with dissociative symptoms but failed, contrary to expectation, to predict diagnostic outcome 10 years later. Overall, the association of deviant personality with persistence of conversion symptoms is accompanied by a lack of specificity concerning personality features that are most relevant.

2.12 (vi) Psychopathology.

Psychological factors are usually subsumed under two headings, those of psychopathology and personality. Although studies of the outcome of "hysteria" or "conversion hysteria" take these to be independent diagnoses, significant affective morbidity has been noted by several writers despite the classical association of hysteria with the relative absence of affect that comprised "belle indifférence" (Charcot & Marie, 1892). While the latter has been discredited as a reliable accompaniment of conversion symptoms (Raskin et al. 1966; Bishop & Torch, 1979), the literature on "abnormal illness behaviour" (cf. section 1.53(ii)) has suggested that a pattern of affective distress coupled with "affective inhibition" (ie chronic difficulties in expressing emotion) is common among patients whose atypical somatic symptoms persist over many years (Pilowsky & Spence, 1975).

More recently, reductionist claims have been made that depressive symptomatology should be considered primary, particularly among patients manifesting pseudoseizures (Roy, 1980). The relative significance of affective distress as a predictive factor in conversion patients remains under-explored, despite Ziegler's observations that affective distress was more common among older patients with conversion symptoms (who had perhaps had them for a longer time?), and that conversion symptoms were more common among the older members of a depressive population (Ziegler et al, 1960). The prognostic implications of depressive symptomatology are not clear. They might either carry a relatively good prognosis, on the expectation that it represented psychopathology amenable to treatment, or a poor one in view of the associations with features carrying some risk of chronicity (age; pseudoseizures).

2.12(vii) Attitudes to illness.

The polysymptomatic patients who Guze (1970) described as suffering from "Briquet's syndrome" had a particularly poor prognosis in terms of the chronicity of their symptoms. People diagnosed in this way would typically see themselves as having been sick for most of their lives, having a correspondingly long prior history of consultations (and surgical treatments). The term "somatisation" was used for a personal adaptive style as well as a synonym for the syndrome. It is indirect evidence that a longstanding tendency to express distress in physical form underlies a tendency for somatic symptoms to be persistent. Psychological and behavioural characteristics are not among the classical inclusion criteria of "somatisation disorder" in DSM-III or DSM-IV (APA, 1980; 1994), being recognised only as "associated features", although an attempt is made to admit "persistent refusal to accept the
advice or reassurance of several doctors that there is no physical explanation for the symptoms" as a criterion of SD in ICD-10 (WHO, 1992, p.163).

Deviant attitudes to illness have been a primary focus in discussions of hypochondriasis (Kenyon, 1964) and in the concept of "abnormal illness behaviour" (Pilowsky, 1969) which arose out of an attempt to operationalise the concept of hypochondriasis. Pilowsky's attempts to measure abnormal illness behaviour identified sets of attitudes typified as "disease conviction", "disease phobia" and "somatic focussing" that were commoner among patients whose pain had no definable physical cause (Pilowsky & Spence, 1975). These patients' complaints were also typically chronic. Much of the interest of this approach has been descriptive and diagnostic rather than prognostic. However, the Illness Behaviour Questionnaire (Pilowsky & Spence, 1983; cf. section 2.33 (i)) was used in a prospective study of general practice attenders with diagnosed and undiagnosed physical complaints in an attempt to predict future consultation behaviour (Pilowsky et al, 1987). Although they report that high scores for "disease conviction" among men seemed to predict higher consultation rates, this applied to men whose symptoms actually had a physical basis rather than those who didn't. Follow-up was confined to a 12 month period, indicating considerable scope for further follow-up studies. As the use made of medical practitioners was central to the original definition of "abnormal illness behaviour" (cf. section 1.55(ii)), it can be expected to be associated with higher consultation rates.

2.12 (viii) Interventions

The impact of treatment might be assumed to be a well-researched influence on prognosis. In fact most reports on outcome have been retrospective and pay little attention to this issue. An exception is Hafeiz (1980), whose patients had a uniformly positive response to early behavioural interventions. This suggests that, in accounting for outcome, variation in provision of treatment could be a more important factor than differences in response to it. However, this is extremely difficult to assess unless, as in Hafeiz's case, the investigator had first hand knowledge of the interventions that were attempted. This difficulty is well illustrated by the recent report of Couprie et al (1995), who comment that, over the index admission, "a profound improvement at discharge is such a strong predictive factor that no other characteristic carried additional prognostic information". Yet close reading of their report shows no reference is made to treatment that might have contributed to any improvement during these admissions. In any case, it is evident that inferences about "improvement" were based entirely on a single (retrospective, note-based) assessment of disability at discharge and were therefore of limited validity. Because greatest improvement was equated with least disability on discharge, the latter could simply have reflected the relative severity of disability at admission. Research on this question therefore remains particularly welcome.
2.13 Summary of patient factors known to affect outcome.

The failure of previous surveys to pay equal attention to symptomatic and diagnostic outcome was noted in section 2.11. They are not always the same, and need to be considered separately when evaluating patient factors linked to them. They will be considered in turn here, although they can be mutually reinforcing. Slater and Glithero's (1965) study in particular carried a presumption that symptomatic prognosis would always be poor among patients in whom a subsequent diagnosis of neurological disease was made and that diagnostic revision was most likely when evidence of neurological disease was already present. While presence of overt signs of neurological illness is one factor predisposing to a worse symptomatic outcome, as well as rediagnosis, it is not the only one. Others, including previous history, a conversion symptom's type and associated psychopathology, were identified in the literature reviewed here.

2.13 (i) Symptomatic outcome

The review of past work suggests two broad sets of patient factors affect symptomatic outcome independent of its relation to diagnostic outcome. One is the chronicity of the problem. While this can be expected to show a positive relationship with a patient's age, other indicators of chronicity need to be recognised. The length of the history of the presenting symptom appears to be greater in cases with a poor prognosis (section 2.12(ii)). However, if the complaint were to represent one facet of a more insidious behavioural problem in some patients, other forms of chronicity are relevant. A prior history of medical-seeking behaviour in the absence of neurological disease would be expected to be associated with continuing presentations, as the literature on Briquet's syndrome and SD suggests (section 2.12(vii)).

The remaining set of factors reflects evidence from clinical examination. A reasonable consensus links several clinical findings (somatic and psychological) with poorer symptomatic prognosis. Although the worse prognosis of some symptoms may be linked to a greater risk of diagnostic revision, pseudoseizures seem more likely to persist than motor symptoms (section 2.12 (i)). With the same caveat, presence of other neurological symptoms appears to predispose to a poorer symptomatic prognosis (section 2.12 (iv)). The presence of multiple somatic symptoms seems likely to be associated with poorer prognosis from the work reviewed in Section 2.12(iii). Presence of a personality disorder and absence of affective distress appear to predispose to symptom persistence from work reviewed in section 2.12(vi) although little attempt has been made to evaluate these with standardised measures of personality and mood.
2.13(ii) Diagnostic outcome.

In assessing factors likely to predispose to a subsequent diagnosis of neurological disease that accounted for a 'conversion' symptom, the follow-up literature is less helpful and its evidence less direct. However, individual findings, such as the high proportion of Slater's patients whose symptoms were rediagnosed after having provisional neurological diagnoses at presentation, do appear to implicate shortcomings in the original diagnosis and suggest that 'provisional' diagnoses are ominous in this respect. This association depended upon many patients with pseudoseizures in Slater's sample. This is an area of pathology in which the boundary between organic and psychogenic disorder has been relatively fluid, not only in the diagnostic vicissitudes surrounding individual patients, but the reclassification of some diagnostic features of pseudoseizures as evidence of complex partial epilepsy (Kanner et al, 1990). Some symptoms, notably seizures, may therefore prove inherently more likely to be associated with a future change of diagnosis as well as poorer symptomatic prognosis. It is also possible to collate the reports from neurological centres of a high association of other CNS pathology and conversion symptoms (section 2.12(iv)) and the relatively worse prognosis facing such patients to infer that other evidence of compromised neurological function thought unrelated to the index symptom would be more likely to be found in cases that were subsequently rediagnosed.
2.2 Hypotheses of the investigation.

The present investigation was planned to look at outcome over a more tightly circumscribed period (10 years) than other longer term studies. By taking patients admitted to a specialist neurological hospital for investigation as its target population, it demands comparison with Slater and Glithero's (1965) study, conducted over 25 years earlier at the same hospital. Previous work, including Slater's study, suggests that varied outcomes can be expected. Symptomatically, some patients would remit completely, some improve, some stay much the same, and others worsen. Some patients would have their symptoms re-diagnosed because undiagnosed disease to which these could retrospectively be attributed was recognised. In others, whether symptoms remitted or persisted, there would be no clinical grounds to doubt the original clinical formulation. It might also be expected that some patients would make much greater use of medical services subsequently, in ways that could be disproportionate to perceived medical need. Each of these outcomes (symptom persistence, diagnostic constancy and consultation behaviour) was the subject of distinct hypotheses, tested in the course of the present study.

2.21 Hypotheses concerning symptomatic prognosis.

Patient factors with a known association with symptom persistence concern historical, somatic and psychiatric findings (section 2.13 (i)). Given the inevitable inconsistencies in procedure and subjects between past studies in this field (section 2.11), and the opportunity of more rigorous testing afforded by data from initial psychometric assessments and a controlled follow-up interval, these were re-evaluated with the present sample.

Historical factors took account of the length of the presenting symptom's history, as well as the patient's history of consulting in the apparent absence of diagnosed disease. Clinical factors took account of the symptom itself, evidence of neuropathology (in the form of diagnostic inferences and examination findings), the number of symptoms, and psychopathology in the form of personality disorder or affective disorder.

A conversion symptom was more likely to persist at follow-up when:

(i) Previous history indicated the symptom's appearance was not a new event:
   (I) The symptom was already chronic at the time of presentation.
   (II) The patient had a prior history of "abnormal illness behaviour" (and abnormal attitudes to illness were detected by the Illness Behaviour Questionnaire at presentation.)
(ii) There were relatively stronger grounds for suspecting neurological disease:
   (I) The primary symptom was seizures rather than weakness or loss of sensation.
   (II) There was evidence suggestive of CNS pathology either on initial physical examination, or in the diagnoses made on presentation.

(iii) Psychiatric examination was consistent with a tendency to have chronic hysterical symptoms:
   (I) The patient complained of more than one medically unexplained symptom.
   (II) There was independent evidence of personality disorder.
   (III) There was little evidence of affective distress at presentation.

2.22 Hypotheses concerning diagnostic prognosis.

Past work offers fewer conclusions concerning factors predisposing to subsequent diagnosis of neurological disease that accounts for a pseudoneurological symptom and evidence has been more indirect in kind (section 2.13(ii)). The question is one of great clinical importance, and it was important not to neglect opportunities to test plausible associations even if these had not been examined previously. In particular, it would be important not to concentrate exclusively on a relatively small group of patients whose diagnoses were indeed revised, but to consider which factors, if any, might provide some guarantee that rediagnosis was extremely unlikely. Given that the patient cohort might represent a spectrum ranging from members who had much in common with neurological patients (and ended up as neurological patients) to others who typified a set of true conversion patients (and remained so during the study), hypotheses attempted to characterise either type on the presumption that their diagnostic prognoses would indeed differ.

As many cases of rediagnosis involve confirmation of suspicions formed during earlier assessments, leading to provisional diagnoses rather than no diagnosis, these tentative diagnoses can be expected to be linked to a greater risk of future rediagnosis. However, provisional diagnoses may not be the only indicator of as yet undiagnosed disease. Other features elicited through ordinary clinical assessment, and possibly associated subsequently with diagnosable pathology accounting for the presenting symptom, include past and concurrent neurological illnesses and the presence of signs on examination of the nervous system (irrespective of whether these were attributed to neurological disease at the time of detection).
The expectation that patients whose presentations were most typical of conversion patients as a group would be most immune from subsequent diagnostic revision is the contrary source of hypotheses here. They divide into factors evident on physical and psychiatric examination. In either case, examination of the literature for features of conversion syndromes that are typical or not can assist this search. A number of findings elicited during standard clinical examination have been repeatedly associated with conversion symptoms. One is historical, that conversion patients are likely to have had previous conversion symptoms (Gatfield & Guze, 1962; Raskin et al, 1966). Others involve demography, including their association with female sex (cf table 2.1) and onset in early adulthood (APA, 1994). A further clinical finding concerns a remarkable observation that has been repeated in many studies since Briquet (1859) that, irrespective of the symptom involved, when it is unilateral it will favour the left side of the body over the right (Stern, 1977).

Much of the literature on psychiatric features typical of cases of conversion is negative, particularly that which has investigated the claims of associated psychological features such as "belle indifference", the symbolic function of the symptom or secondary gain as characteristic. These have failed to prove themselves (Lewis et al, 1965; Lazare, 1981). Others, such as the association of onset with significant life events would require methods unavailable to the present study. However, the association of conversion symptoms with affective symptomatology (Roy, 1980) and, at least in the case of unexplained pain, with patterns of response on the Illness Behaviour Questionnaire (Pilowsky, 1982; cf. section 2.33(i)) provide indices of psychopathology 'typical' of conversion.

A subsequent diagnosis of disease accounting for the presenting symptom would therefore be more likely when:

(i) There was evidence of impaired neurological function, in the form of:
   I) An independent neurological illness present at presentation;
   II) A previous history of neurological illness;
   III) A provisional neurological diagnosis being made on the index admission.
   IV) The presenting symptom was more susceptible to misdiagnosis, ie. seizures or tremor.
   V) Signs were recorded on neurological examination.

(ii) The clinical presentation was untypical of patients with conversion disorder, in particular:
   I) Onset of the symptom was late in life;
   II) No history was elicited of past unusual illness behaviour;
   III) The patient was male;
   IV) There was no tendency for unilateral symptoms to favour the left side;
2: A 10 year follow-up study

V) Initial scores on the IBQ subscales did not follow a pattern

Pilowsky described in a “discriminant function” for conversion patients.

(iii) Psychiatric and psychological findings were not consistent with a diagnosis of a neurotic disorder, with:

I) No previous history of psychiatric treatment;

II) A lower incidence of diagnosed psychopathology;

III) Lower scores on questionnaire rating of affective symptoms.

2.22 (iii) Hypotheses concerning future consultations.

Finally, when considering factors likely to influence subsequent consultation behaviour, the lack of attention to this outcome in previous studies means original hypotheses are required. It can be expected to reflect differences in morbidity and disability within the sample, but also those behavioural dispositions to treatment seeking unrelated to objective pathology elaborated in the literature on “illness behaviour” (cf. section 1.55(ii)).

While the illness behaviour literature has studied attitudinal variations in the absence of objective measurement of consultation rates, the follow-up studies that led to the description of Briquet’s hysteria had clearly linked previous high levels of consultation in the absence of organic pathology with a continuing tendency to consult frequently (Gatfield & Guze, 1962).

In the present study, higher levels of treatment seeking behaviour during the follow-up period could be expected to reflect “abnormal illness behaviour” as defined by Pilowsky (1979) as well as the presence of diagnosed illness. Consultation rates raised as a result of organic morbidity would be seen in cases where illnesses had already been evident at presentation, and where readagnosis of the original symptom was necessary. Continuing abnormal illness behaviour accounting for an inflated consultation rate was expected when factors associated with this among patients with somatisation disorder were present, i.e. evidence of a prior pattern of abnormal illness behaviour. These hypotheses have had to be stated in terms of data available in the study, rather than that which would be ideally to hand.

Future consultations were expected to be more frequent when:

(i) The initial symptom had persisted over the follow-up period, with consultation rates highest in cases where the initial diagnosis was changed.

(ii) Initial assessment led to diagnosis of an additional psychiatric or neurological disorder, or a provisional neurological diagnosis for the presenting symptom.
(iii) There had been evidence of abnormal illness behaviour at assessment, either through a history of previous episodes of unexplained illness, or an unjustified degree of conviction about personal ill health had been evident via the Illness Behaviour Questionnaire.

(iv) Among patients who rated their health and disability least favourably on follow-up, or who appeared to be candidates for Briquet's syndrome on a short screening questionnaire given at follow-up.

2.3 Method of the present investigation

2.31. The sample.

The present sample comprised 73 of a cohort of 79 patients that had received extensive in-patient assessments at the National Hospitals between 1978 and 1980 (Wilson-Barnett & Trimble, 1985). That cohort was a series of consecutive patients having well-defined neurological symptoms for which no definite somatic cause was apparent after investigation. They had all been referred to the liaison psychiatry service within the hospital and complied with the psychiatric and psychological assessments described in sections 2.32 and 2.33. All these patients were sought for the follow-up study, and subsequently excluded only if it proved impossible to obtain even a minimal amount of information concerning their medical progress up until the time of the study.

Exclusions were therefore made in cases when it either proved impossible to trace the individual concerned, or, after they had been traced, it was clear they had become lost from medical contact, with any information concerning their clinical status needing to be based upon medical reports over 2 years out of date. Two patients were also excluded from further investigation after their GPs reported they had died during the follow-up period. The cause of death was ascertained in each case, and confirmed to have no relationship to their physical complaints at the time of the original assessment, or to psychiatric illness. The reasons for the exclusion of all six individuals from further follow-up are summarised in table 2.3.

The remaining 73 patients comprised 16 men and 57 women, with a median age of 33 years (range 18 to 63 years). Data on handedness was available for 57 of the sample, with 53 (94%) being right handed. They had presented with a primary complaint in one of six categories (in descending order of size): motor; seizures; special senses; other sensory; amnesia and other. Their relative frequencies are summarised in table 2.4. This groups patients according to the primary symptom for which no clear neurological diagnosis could be made. Where more than one symptom fell into this category, the symptom that had been most prominent in the request for psychiatric assessment was cited. Most of the cases with apparent peripheral sensory impairment (complaints recorded of "numbness" or "paresthesia") also reported motor weakness and are classified under that heading, leaving
only 4 cases with peripheral sensory loss without motor weakness. The 31 patients with a primary complaint of weakness were principally in the lower limbs (14) cases; the upper limbs (1 case, unilaterally); upper and lower limbs (10 cases); vocal impairment through aphonia or dysarthria (4 cases) and 2 presentations of dysphagia.

Nearly half of the 27 patients with pseudoepilepsy had repeated seizures with loss of consciousness (12 cases); 2 further patients had episodes in which impairment of consciousness was associated with complex automatisms; 3 patients appeared to suffer drop attacks in which falling was not accompanied by loss of consciousness; 9 patients had repeated episodes of loss of consciousness, usually associated with simple falls or faints and one patient had had a single episode of loss of consciousness.

The other significant group comprised patients reporting changes in the special senses. This involved visual impairments in 5 cases, and hearing (associated with dizziness) in the remaining one.

2.32. Review of initial assessment

Initial assessments comprised clinical examinations by the admitting neurological team and a research psychiatrist, and a battery of questionnaires assessing mood, personality and abnormal illness behaviour.
<table>
<thead>
<tr>
<th>Subjects [study nos.]</th>
<th>Reason for removal.</th>
</tr>
</thead>
<tbody>
<tr>
<td>[15]; [18].</td>
<td>Died of Carcinoma (each unrelated to original symptom.)</td>
</tr>
<tr>
<td>[38]; [78].</td>
<td>Completely untraceable.</td>
</tr>
<tr>
<td>[45]; [63].</td>
<td>Lost from medical contact during FU period.</td>
</tr>
</tbody>
</table>

*Table 2.3 Subjects not included in follow-up.*
2.32(i) Clinical assessments.

Each patient had had a full neurological and psychiatric assessment. Neurological examinations were almost always conducted by neurologists in training working under supervision at the hospital; the psychiatric assessments had all been performed by the same psychiatrist, Dr. Michael Trimble. Neurological assessments were recorded systematically on a structured 12 page proforma that required details to be entered at each stage of history taking and through detailed neurological and systemic examinations. Neurological diagnoses were clearly stated on the discharge summary for the index admission. Psychiatric examinations were recorded in the form of summary historical data (including any previous episodes of medically unexplained illness behaviour), clear diagnostic conclusions and advice to the referrer. The records of these examinations were all inspected to obtain a data set relevant to the hypotheses of the investigation, viz. the nature of the presenting symptom(s); evidence of previous episodes of neurological illness; recorded psychiatric history; the nature of any pastdiagnoses; concurrent neurological and psychiatric pathology; the nature and number of signs noted during initial neurological examination. Recording of mental state features varied in completeness and was not subject to additional analysis. The use of medication at admission, for psychotropic and non-psychotropic drugs, was also recorded.

Table 2.4 summarises a number of the initial clinical findings, confirmed by detailed re-examination of the contemporary case notes. The median number of presenting complaints that had been recorded was 3. No neurological signs were recorded for 35% of the sample, while the median number for the sample as a whole was 2. The notes were checked for the lateralisation of symptoms and signs when these favoured one side. There was clear evidence of lateralisation in 35 cases (48%), favouring the left side in 22 cases (30%), and the right in 13 (18%).

Neurological opinion on the index admission led to 11 patients (15%) being given an independent neurological diagnosis on the index admission; where patients were given a provisional neurological diagnosis this was usually a sole neurological diagnosis in 13 cases and an alternative diagnosis in one. In a majority of cases (64%), the psychiatrist’s notes record a diagnosis of an additional psychiatric disorder in addition to one of hysteria or functional disorder. These are summarised in table 2.5.

2.32(ii) Additional historical information.

28 cases (38%) had at least one previous recorded episode of neurological illness. These are listed in table 2.6 in order of frequency. Nearly all patients had had a single previous diagnosis: in the few cases with more than one, the most disabling and specific diagnosis is favoured (e.g. epilepsy in place of migraine). These are of considerable interest, in some cases remaining relevant to the symptom under investigation (e.g. headaches with a
<table>
<thead>
<tr>
<th>Symptom</th>
<th>No. with more than one complaint</th>
<th>No. having independent neurological diagnosis</th>
<th>No. with provisional neurological diagnosis</th>
<th>No. with independent Psychiatric diagnosis</th>
<th>Mean no. of neurological signs</th>
<th>Mean length of history (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor (n=31)</td>
<td>25</td>
<td>4</td>
<td>6</td>
<td>16</td>
<td>2.9</td>
<td>78</td>
</tr>
<tr>
<td>Seizures (n=27)</td>
<td>21</td>
<td>4</td>
<td>7</td>
<td>20</td>
<td>1.4</td>
<td>62</td>
</tr>
<tr>
<td>Amnesia (n=3)</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>26</td>
</tr>
<tr>
<td>Special senses (n=6)</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>4</td>
<td>2.3</td>
<td>145</td>
</tr>
<tr>
<td>Other sensory (n=4)</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>1.0</td>
<td>42</td>
</tr>
<tr>
<td>Other (n=2)</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1.5</td>
<td>12</td>
</tr>
</tbody>
</table>

Table 2.4: Clinical findings for the follow-up sample classified according to index symptom.
### Psychiatric diagnoses

<table>
<thead>
<tr>
<th>Psychiatric diagnosis</th>
<th>No.  (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>20 (27%)</td>
</tr>
<tr>
<td>Anxiety neurosis</td>
<td>10 (14%)</td>
</tr>
<tr>
<td>Personality disorder</td>
<td>14 (19%)</td>
</tr>
<tr>
<td>Briquet's syndrome</td>
<td>3 (4%)</td>
</tr>
</tbody>
</table>

**Total:** 47 (64%)

*Table 2.5 Independent psychiatric diagnoses made at initial assessment.*

### Previous diagnosed illness

<table>
<thead>
<tr>
<th>Previous diagnosed illness</th>
<th>Frequency (no. with related current symptom)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epilepsy</td>
<td>5 (3)</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td>3 (2)</td>
</tr>
<tr>
<td>Head injury/RTA</td>
<td>3 (2)</td>
</tr>
<tr>
<td>Meningitis</td>
<td>3 (3)</td>
</tr>
<tr>
<td>Migraine</td>
<td>3 (2)</td>
</tr>
<tr>
<td>Retinal blindness</td>
<td>2 (0)</td>
</tr>
<tr>
<td>Extrapyramidal gait disorder</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Ataxia of unknown etiology</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Transverse myelitis</td>
<td>1 (1)</td>
</tr>
<tr>
<td>S.L.E.</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Multiple Sclerosis</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Vestibular damage</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Thyrotoxic neuropathy</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Nerve deafness</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Cataracts</td>
<td>1 (0)</td>
</tr>
</tbody>
</table>

*Table 2.6 Summary of recorded episodes of previous neurological illness (n=28) (no. with possibly related pseudoneurological symptom in parenthesis)*
history of meningitis; seizures with a history of epilepsy). The number of cases in which there was symptomatic overlap between features of the diagnosed condition, and the principal symptom under investigation, are given in parentheses.

35 cases (48%) had a recorded episode of psychiatric illness. 34 patients (47%) had been noted at the original psychiatric assessment to have had at least one previous episode of atypical illness, in which no physical cause had been found for physical complaints.

The median duration of the patients' hysterical symptom(s) had been 36 months (range 0-400 months). 38 cases (52%) were receiving psychotropic medication (most commonly hypnotics). In addition, other medications (including analgesics and anticonvulsants) were being prescribed for 21 (29%) of the sample at the initial assessment.

2.33 Instruments used in the study.

Psychometric instruments were used both in the initial assessments of each patient, and during the follow-up evaluations. For convenience, they will all be described here.

2.33 (i) Psychological assessments during initial assessment.

Three principal types of structured assessment had been incorporated into the original study, being administered at the time of psychiatric assessment. They measured personality, mood and abnormal illness behaviour.

Personality.

The Hysteroid-Obsessoid Questionnaire (HOQ) of Caine and Hope locates patients on a continuum from hysteroid to obsessoid traits (Caine and Hope, 1967). Forty eight items on a self-report questionnaire are checked as either true or false. High scores represent a marked hysteroid trait; low scores obsessionality. Developed at a time when hystronic and anankastic personality traits were held to be incompatible (eg. Shapiro, 1965), items had been selected for their apparent capacity to discriminate between representative patient groups. It was based on a subsscale of the Symptom and Sign Inventory (Foulds & Hope, 1968), a criterion keyed inventory for a range of neurotic disorders that had hoped to emulate the Minnesota Multiphasic Personality Inventory. Caine and Hope attempted to improve the validity of the hysteroid subscale by extending it. The resulting scale had a poor correlation with conversion and obsessional symptoms despite an apparent association with diagnosed personality traits. Scores showed a high (0.7) correlation with the extraversion scale of the EPI (Caine, 1970). Although the scale has been used in previous studies of hysteria in neurological settings (Merskey & Trimble, 1978; Wilson-Barnett and Trimble, 1985) it has been very rarely used beyond these studies since its introduction. No doubt its closeness to
Eysenck's construct has contributed to its obsolescence. It had failed to discriminate between patients with pseudoneurological symptoms, neurological illness or psychiatric illness in an earlier comparative study (Wilson Barnett and Trimble, 1985).

The Eysenck Personality Inventory (EPI) (Eysenck and Eysenck, 1964) is a 57 item questionnaire also eliciting true-false responses. Three dimensions are scored, a 24 point extraversion scale; a 24 point neuroticism scale and a 9 point 'lie' scale. The instrument was a refinement of its predecessor, the Maudsley Personality Inventory (which assessed the first two dimensions but lacked the 'lie' scale). Factor analyses on data from hundreds of subjects confirmed Eysenck in his view that these two ordinates were independent of each other and represented fundamental second order dimensions of personality underlying more intricate and descriptive formulations. He believed they were associated with basic differences in response to operant conditioning, 'extraversion' being a measure of stimulus dependence, and 'neuroticism' a tendency to rapid habituation. Correlation between the two dimensions was minimal in the EPI, and test/retest reliability is reported in excess of 0.80 for one year. While scores vary on each dimension in 'normals' according to age, sex and occupation, typical scores are around the mid-point (12) for extraversion and slightly less for neuroticism. This is raised in psychopathological groups and 'lie' scores have been found to vary much more than the other dimensions between 'normal' groups. Although Eysenck replaced the EPI with the Eysenck Personality Questionnaire or EPQ, which incorporated a 'psychoticism' scale, it remains a simple and reliable method for assessment of the construct of extraversion in particular.

Mood

The Beck Depression Inventory (BDI) (Beck et al, 1961) provides a global rating of depressive symptomatology on a 63 point scale. It comprises 21 multiple choice questions assessing subjective mood and recent cognitive and biological concomitants of mood change. Although the scale is often used in a shortened 13 question format, the full scale was used here for maximum sensitivity. Initially validated on a clinical population, the questionnaire has been very extensively used in its intended role, as a state measure of the severity of depression. Typical values on this extended scale have been quoted as 'not depressed' 11; 'mildly depressed' 19; 'moderately depressed' 25; 'severely depressed' 28 (Beck et al, 1988). It has been very widely used and high retest reliability (range 0.73 to 0.95). It has been criticised for a relative lack of emphasis on biological features of depression, sensitivity to anxiety, and also for apparent insensitivity to changes in depressed mood when it has appeared to measure a 'trait' rather than affective 'state' (Serra & Pollitt, 1975).
The Mood Adjective Check List (MACL) of McNair and Lorr (1964) comprises 24 adjectives rated on a 4 point ordinal scale. Five dimensions are scored: depression, anxiety, fatigue, vigour and hostility. Eight items contribute to the depression scale, and only four to each of the others. Only the depression and anxiety scales were used in the current study, having maximum scores of 24 and 12 respectively. The method of scoring reflects the scale's development from analysis of the clustering of scores within a 60 item checklist based on the Nowlis mood scales (Nowlis & Nowlis, 1956). McNair and Lorr (1964) refined their choice of moods and their constituent scale items by serial studies on large psychiatric populations intended to isolate the mood variables that were particularly sensitive to psychotherapy and chemotherapy. The concurrent validity of each subscale was independently established in relation to existing questionnaire measures of each mood. Groups of psychiatric outpatients have significantly higher scores for tension, anxiety, depression and fatigue than 'normals', and lower scores for 'vigour'. Although recommending itself through ease of use, and its original validation as a change sensitive measure among large neurotic samples, it has fallen from use in favour of more specific scales designed to assess individual moods in depth.

Illness behaviour.

The Illness Behaviour Questionnaire (IBQ) (Pilowsky, 1982) is a self-report questionnaire examining attitudes to illness and affective status. It was developed from an earlier questionnaire that had identified three distinct factors among the illness beliefs of patients whose behaviour was hypochondriacal (Pilowsky, 1967). This was expanded into a 62 item inventory (of which 52 are scored) that has been used among patients with chronic unexplained pain and other medical conditions. Seven subscales were standardised as a result of factor analyses with pain patients (Pilowsky & Spence, 1975), namely disease phobia (DP), disease conviction (DC), somatic versus psychological attribution (P/S), affective distress (AD), affective inhibition (AI), denial (D) and irritability (I). Scores have always been highly skewed in favour of the lower end of each scale, requiring summated scores to be recorded as the median. Pilowsky has reiterated the validity of this division, despite intervening studies suggesting different factorial structures pertain among other patient populations (Pilowsky, 1993). Two compound "Second order" factors (Disease affirmation (DA) and affective state (AS)) were also described by him (Pilowsky & Spence, 1983), and he has promoted use of a discriminant function (DF) equation said to have 85% to 90% discriminative validity in distinguishing chronic pain patients from general practice patients. A high discriminant score reflects high ratings on "disease conviction" and "denial" with a tendency to somatic over psychological attribution in the formula

\[DF = 53.8 + 5.7(DC) - 10.2(P/S) - 0.6(AI) + 2.4(D)\]

Pilowsky claims DF scores of more than 50 characterise 85% of pain patients and cardiac patients, in distinction from patients consulting in general practice and psychiatry. He has consistently described this as a discriminant function for 'likelihood of a conversion
reaction although he has not personally used it with other pseudoneurological symptoms and it has failed to discriminate between 'organic' and 'functional' patients on a neurology ward (Creed et al, 1990). Strictly speaking, none of first or second order IBQ scales measures abnormal illness behaviour according to Pilowsky's own definition (cf. section 1.55(ii)). A second, later instrument, the Illness Behaviour Assessment Schedule (Pilowsky et al, 1983) attempted to rectify this by asking patients to recount the explanations they had received of their original illness, and then contrasting these with their current beliefs concerning their condition. The latter instrument has received little subsequent attention, and has not been validated on other clinical populations, while the IBQ continues to be used in psychosomatic research.

2.33 (ii) Additional standardised assessments used at follow-up.

Two further published instruments were used in the course of follow-up alongside repeated adminstrations of instruments used above.

The 'Screening Test for Somatisation Disorder' of Othmer and DeSouza (1985) is a simple checklist of 7 symptoms selected for their diagnostic significance from the DSM - III criteria for somatisation disorder. Subjects are asked if they have ever suffered incapacitation from, sought medical advice about or had treatment for each of 7 symptoms in turn (shortness of breath, dysmenorrhea, burning in sex organs, lump in throat, amnesia, vomiting, painful extremities), their replies being scored totalled on a simple 7 point scale. It was developed by extracting a subset of female psychiatric outpatients who had a history of multiple unexplained physical symptoms beginning before the age of 30 years. A cohort of 85 (12% of those screened) divided almost equally into those meeting DSM-III criteria of SD (14 symptoms plus) and those who did not. The scale's 7 symptoms were selected on the basis of their discriminant index. The resulting list was validated against DSM-III diagnostic criteria for SD, the authors reporting a score of 2 or more made the diagnosis of SD "highly probable" and that, at least in a population where around half the patients should qualify for the diagnosis, sensitivity is 93% and specificity 59% with two symptoms as the criterion; sensitivity is 73% and specificity 94% with three symptoms. After this report, the screening test was highlighted (literally) in the diagnostic recommendations of DSM-III-R, the manual current at the time of the follow-up study. No explanation was offered in DSM-IV of its complete removal.

The Nottingham Health Profile (NHP) (Hunt, McKewen & McKenna, 1986) was also used in the study, during the follow-up phase. This is a self-report measure of disability that has been developed and extensively tested in community studies. A first part comprises 38 questions requests 'yes' or 'no' to whether a problem is currently present. Replies lead to scores on 6 subscales (energy, pain, emotional reactions, sleep, social isolation and physical
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Items are weighted according to their gravity: each scale has a maximum score of 100 when all its candidate problems are checked. Part 2 assesses social functioning related to health, comprising 7 yes/no questions: "Is your present state of health causing problems with your ... job of work/looking after the home/ social life/ home life/ sex life/ interests and hobbies/ holidays". The subscale structure permitted reference norms to be established for age and social class (scores in both parts increase with age and with decrease in social class) and analysis of the differential impact on scores of specific medical conditions. Scores on all subscales have been shown to correlate with general practice attendance irrespective of medical diagnosis by detailed comparison between over 200 "non-consulters" (no visits in previous year) and "consulters" (3 or more visits in previous year). This validation study also illustrates the skewed distribution of NHP scores, with more than 50% of scores being zero on each section (op.cit, ch. 5).

2.33 (iii) Baseline findings among the cohort.

Findings on the questionnaire measures of mood, personality, and illness behaviour for the 73 patients are summarised in table 2.7 In a previous study with these patients, only depression scores had been seen to differ from controls with neurological and psychiatric illnesses (Wilson Barnett and Trimble, 1985). Table 2.8 tabulates the distribution of these scores according to the psychiatric diagnoses described in table 2.5. Among the mood scales, the depression scores from the BDI were as raised in patients given diagnoses of personality disorder as those recognised to have clinically significant anxiety or depression. The Eysenck scales did not discriminate between those judged to have abnormal personality and those who were clinically anxious.
Affective measures:

I) Mood Adjective Check List:
- Anxiety Median 4.0 (Range 0-11)
- Depression Median 5.0 (Range 0-23)
- Vigour Median 1.0 (Range 0-12)
- Fatigue Median 4.0 (Range 0-12)
- Hostility Median 0.0 (Range 0-12)

II) Beck Depression Inventory:
- Depression Median 14 (Range 0-50)

Personality measures:

I) Hysteroid-obsessoid score: Mean 21.0 (SD 6.8)

II) EPI: Extraversion: Mean 3.67 (SD 1.9)
- Neuroticism: Mean 11.45 (SD 5.9)
- Lie: Mean 3.67 (SD 1.9)

Illness Behaviour Questionnaire:
(factor scores:)
1. General hypochondriasis. Median 2 (Range 0-7)
2. Disease conviction. Median 3 (Range 0-5)
3. Psychological/somatic focus Median 2 (Range 0-5)
4. Affective inhibition Median 2 (Range 0-5)
5. Affective disturbance Median 3 (Range 0-5)
6. Denial Median 4 (Range 0-6)
7. Irritability Median 2 (Range 0-5)
(2nd order factors:)
I) Affective state (1+5 +7 above) Median: 6.0 (Range 0-16)
II) Disease affirmation (2 +(5-3)) Median: 4.5 (Range 0-18)

Table 2.7: Psychological questionnaire scores at initial assessment (n=73).
<table>
<thead>
<tr>
<th></th>
<th>Anxiety Neurosis (n=10)</th>
<th>Depression (n=20)</th>
<th>No other diagnosis (n=26)</th>
<th>Personality disorder (n=14)</th>
<th>Briquet's syndrome (n=3)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BDI:</strong></td>
<td>14.5 (15.0)</td>
<td>20.15 (17.5)</td>
<td>8.5 (7.0)</td>
<td>18.9 (19.0)</td>
<td>25.7 (25.0)</td>
</tr>
<tr>
<td><strong>MACL:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>4.0 (4.0)</td>
<td>5.2 (4.5)</td>
<td>3.3 (3.0)</td>
<td>5.1 (5.5)</td>
<td>7.7 (7.0)</td>
</tr>
<tr>
<td>Depression</td>
<td>6.8 (3.5)</td>
<td>8.8 (7.0)</td>
<td>2.9 (1.0)</td>
<td>6.9 (5.0)</td>
<td>11.7 (9.0)</td>
</tr>
<tr>
<td><strong>IBQ:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affective state</td>
<td>9.3 (10.5)</td>
<td>8.05 (8.5)</td>
<td>4.0 (2.0)</td>
<td>6.1 (6.0)</td>
<td>12.3 (12.0)</td>
</tr>
<tr>
<td>Affective disturbance</td>
<td>6.1 (6.5)</td>
<td>5.75 (6.0)</td>
<td>6.1 (6.0)</td>
<td>6.4 (6.0)</td>
<td>5.0 (5.0)</td>
</tr>
<tr>
<td><strong>EPI:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neuroticism</td>
<td>12.1 (12.0)</td>
<td>13.0 (14.5)</td>
<td>9.0 (8.0)</td>
<td>12.9 (12.5)</td>
<td>13.0 (14.0)</td>
</tr>
<tr>
<td>Extraversion</td>
<td>11.2 (11.0)</td>
<td>8.25 (7.5)</td>
<td>10.3 (11.0)</td>
<td>11.4 (12.0)</td>
<td>11.3 (13.0)</td>
</tr>
<tr>
<td>Lie scale</td>
<td>3.0 (3.0)</td>
<td>3.95 (4.0)</td>
<td>3.8 (4.0)</td>
<td>3.6 (3.0)</td>
<td>3.0 (3.0)</td>
</tr>
</tbody>
</table>

Table 2.8  Comparison of diagnostic and questionnaire assessments of affective state and personality. Mean scores (median).
2.34. Procedure for the follow-up study.

Ethical approval was received from the ethics committee of the hospital, and permission obtained to re-contact the patients from the consultants who had originally treated them. The patients' notes were then examined, revealing that only 5 remained in attendance at the out-patient department at the time of the study. In 74 other cases, their GP was contacted by a letter that explained the nature of the study and my wish to recontact the patients, together with a short questionnaire that requested some preliminary information. This comprised: the patient's current whereabouts; their current clinical status (diagnoses and treatment); the nature of any additional investigation and treatment at other hospitals they had received since the time of the earlier study.

In cases where it was evident that the patient had either had the original complaint rediagnosed, or where there had been further investigation of the original complaint, hospital departments were contacted to ascertain the nature of the discharge diagnosis and the tests on which it was based.

An attempt was then made to contact all living patients directly by letter, with the permission either of the general practitioner or consultant who was currently attending them. They received a short questionnaire requesting the following information: whether they had been satisfied with the medical treatment received at the time of the previous assessment; whether the complaint they presented then still persisted; whether they had had further investigations for the complaint; whether they currently suffered from illness of any other kind; whether there were everyday activities with which they could no longer cope; the frequency with which they had visited their GP in the intervening period; and whether they would be prepared to participate in the next stage of this investigation.

The questionnaire concluded by asking respondents to supply a current telephone number, and advised them that they would receive a short telephone interview and one further set of questionnaires to complete if they were willing to continue to assist in the study.

The telephone interview that respondents then received followed the same format in each case. The interviewee was told that questions would cover four areas in turn, namely, their original problem; their present health; the use they had made of medical and related services over the study period; and their current ability to carry out everyday activities. Questioning on their original problem asked for confirmation of the symptoms that had prompted the previous assessment; the subsequent course the symptom had taken (noting how quickly it had remitted if it had disappeared); what explanation, if any, they recalled being given during the original admission of their condition; and the outcome of any subsequent reinvestigation they had received for it. The questions concerning their present health requested an overall rating of their health on a 1 to 10 scale and a list of the current symptoms, treatment and diagnoses that were reported by them on request. They were also asked whether or not they had ever considered requesting medical attention for each of a checklist
of seven symptoms that duplicated Othmer and DeSouza's (1985) 'Seven symptom screening test for somatisation disorder' (cf. Section 2.3(iii)).

Information concerning their use of medical services over the follow-up period was elicited by asking independently about use of their general practitioner; hospital out-patient departments; hospital in-patient units; other NHS agencies, both hospital and domiciliary; private medical consultations; and use of alternative practitioners.

Current functioning was assessed by requesting a self-assessment of their overall disability on a 1 to 10 scale; by eliciting the nature of any activities they were no longer able to perform, and the degree of assistance that was required from partners, relatives, friends and professionals; and specific difficulties with transport and mobility. (This information was used to produce an overall estimate on the part of the interviewer of the patient's disability on a 1 to 10 scale). All respondents were also asked whether they were in employment, and whether they were living alone. At the conclusion of the interview, confirmation was sought that they were still willing to receive and return a set of 3 further short questionnaires. In a few instances, respondents were willing to supply further information, but not by telephone, either because they lacked access or the ability to use one, or because they wished to restrict any contact to correspondence. In these cases, the respondent was either visited at home (3 instances), or asked to complete a longer questionnaire covering the factual material of the semi-structured interview (4 cases) as appropriate.

The questionnaires that were sent to the interviewees on completion of the telephone interview comprised: The Illness Behaviour Questionnaire (Pilowsky & Spence, 1983); The Beck Depression Inventory (Beck et al, 1961); The Nottingham Health Profile (Hunt, McEwen & McKenna, 1986). The questionnaires are described in Section 2.33.

Data was collated and analysed using the Statistical Package for the Social Sciences (Norusis, 1995). Hypothesis testing used the chi-square statistic (discontinuous variables) and the Mann-Whitney or Kruskal Wallis tests (continuous variables).
2.4 Results.

2.4.1 Summary of data collection.

The quantity of information available for each patient varied according to their compliance at each of the three stages of collection of follow-up data. These are summarised in table 2.9, where a small progressive attrition of numbers is evident through each stage of the study.

In only one case did a GP fail to respond after reminders. The patient accepted the offer of a consultation for the purposes of the study so that checks could be made on his history of subsequent treatment. (There had been no major interventions, and he remained well).

Of the three outcome measures: symptomatic outcome; diagnostic outcome and consultation behaviour, assessment of the third depended upon co-operation with the interview, and was therefore only possible for the 56 patients complying with this. By assessing the first two on the basis of medical records, it was possible to check whether clinical outcome was affecting co-operation with the study, with the attendant risk of bias in its results. As table 2.10 shows, the overall proportion of patients having a particular clinical outcome was comparable irrespective of whether they had co-operated with the study.
<table>
<thead>
<tr>
<th>Form of contact:</th>
<th>Information requested:</th>
<th>Complete responses:</th>
</tr>
</thead>
<tbody>
<tr>
<td>GP questionnaire/current hospital</td>
<td>Clinical condition, diagnoses, consultation rate and hospital referrals over fu period.</td>
<td>73 (100%)</td>
</tr>
<tr>
<td>hospital records.</td>
<td>Permission to contact patient.</td>
<td></td>
</tr>
<tr>
<td>(&quot;Level 1&quot;)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Letter to patient.</td>
<td>Current condition. Permission to proceed.</td>
<td>58 (79%)</td>
</tr>
<tr>
<td>Semi-structured interview with</td>
<td>Sections: current condition; interim history; ratings.</td>
<td>56 (76%)</td>
</tr>
<tr>
<td>patient.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(&quot;Level 2&quot;)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rating scales from patient.</td>
<td>IBQ; BDI; Nottingham inventory.</td>
<td>52 (71%)</td>
</tr>
<tr>
<td>(&quot;Level 3&quot;)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Table 2.9: Design of study and compliance with each stage.*
## 2: A 10 year follow-up study

### Symptomatic outcome:

<table>
<thead>
<tr>
<th>Status</th>
<th>Level (1)</th>
<th>Level (2)</th>
<th>Level (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>22 (30%)</td>
<td>15 (27%)</td>
<td>14 (29%)</td>
</tr>
<tr>
<td>Improved</td>
<td>21 (29%)</td>
<td>19 (34%)</td>
<td>16 (31%)</td>
</tr>
<tr>
<td>Unchanged</td>
<td>17 (23%)</td>
<td>13 (23%)</td>
<td>13 (25%)</td>
</tr>
<tr>
<td>Worse</td>
<td>13 (18%)</td>
<td>9 (16%)</td>
<td>9 (17%)</td>
</tr>
</tbody>
</table>

### Diagnostic outcome:

<table>
<thead>
<tr>
<th>Status</th>
<th>Level (1)</th>
<th>Level (2)</th>
<th>Level (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No change</td>
<td>62 (85%)</td>
<td>48 (86%)</td>
<td>44 (85%)</td>
</tr>
<tr>
<td>Revised</td>
<td>11 (15%)</td>
<td>8 (14%)</td>
<td>8 (15%)</td>
</tr>
</tbody>
</table>

*Table 2.10 Status on follow-up and compliance with the study.*
2.42. Symptomatic outcome.

In the vast majority of cases, patient and GP reports on this were both available. Categorisation of the index symptom as absent, improved, unchanged or worse was on the basis of medical reports. Discrepancy with patient reports when these were available was extremely rare, with no disagreements as to whether the symptom improved ("absent" or "improved" at follow-up) or had not improved ("same" or "worse"). As table 2.11 shows, the original symptom was unchanged or worse in over 40% of the sample. This proportion did not differ according to the nature of the presenting symptom.

The 15 interviewed patients whose index symptom had completely remitted had been asked about the extent of its persistence. Table 2.12 summarises their responses. Although most remissions had occurred by the end of the first year after admission, three had not occurred until the second half of the follow-up period.

Data concerning the factors hypothesised to be associated with differences in symptomatic outcome will be presented according to the order of the hypotheses in section 2.21.

The index symptom was much more likely to persist at follow-up if it had been present for a number of years, and if the patient was relatively elderly (table 2.13). The age of onset of the symptom, obtained by subtracting the duration from the patient's age at presentation, did not itself show an association to symptomatic outcome.

Symptomatic outcome showed no evident relationship to the other historical variable of interest, past illness behaviour evidenced by previous medical attention for unexplained symptoms. There was also no relationship to the study's other measure of illness behaviour, IBQ subscale scores (table 2.14).

Table 2.15 shows that no association was evident between symptomatic outcome and diagnosis of another neurological illness at presentation, a past history of neurological illness, or a provisional diagnosis being recorded of a neurological condition to account for the index symptom. Although the median number of signs recorded on systematic physical examination appeared to increase progressively as prognosis worsened, this did not satisfy tests of statistical significance.
A 10 year follow-up study

<table>
<thead>
<tr>
<th>Symptom Type</th>
<th>Absent</th>
<th>Improved</th>
<th>Unchanged</th>
<th>Worse</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor</td>
<td>8</td>
<td>10</td>
<td>6</td>
<td>7</td>
<td>31 (42%)</td>
</tr>
<tr>
<td>Seizures</td>
<td>11</td>
<td>6</td>
<td>7</td>
<td>3</td>
<td>27 (37%)</td>
</tr>
<tr>
<td>Amnesia</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>3 (4%)</td>
</tr>
<tr>
<td>Special senses</td>
<td>0</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>6 (8%)</td>
</tr>
<tr>
<td>Other sensory</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>4 (5%)</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2 (2%)</td>
</tr>
</tbody>
</table>

**Total:** 22 (30%) 21 (29%) 17 (23%) 13 (18%)

*Table 2.11: Original symptom at follow-up grouped by symptom type*

<table>
<thead>
<tr>
<th>Months to remission</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>12</td>
<td>2</td>
</tr>
<tr>
<td>23</td>
<td>1</td>
</tr>
<tr>
<td>24</td>
<td>1</td>
</tr>
<tr>
<td>48</td>
<td>1</td>
</tr>
<tr>
<td>60</td>
<td>1</td>
</tr>
<tr>
<td>80</td>
<td>1</td>
</tr>
<tr>
<td>90</td>
<td>1</td>
</tr>
</tbody>
</table>

*Table 2.12: Time to remission of 15 patients recovering during the follow-up period.*
Table 2.13: Mean (median) duration of symptoms and symptomatic outcome
(Kruskall Wallace 1-way ANOVA.)

<table>
<thead>
<tr>
<th></th>
<th>Absent</th>
<th>Improved</th>
<th>Unchanged</th>
<th>Worse</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Duration of symptom</strong></td>
<td>43 (10)</td>
<td>30 (43)</td>
<td>97 (60)</td>
<td>138 (60)</td>
<td>Chi-sq 13.275; p=0.004</td>
</tr>
<tr>
<td><strong>(months).</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Age of patient.</strong></td>
<td>31.4(28)</td>
<td>32.8(30)</td>
<td>33.0(34)</td>
<td>48.4(51)</td>
<td>Chi-sq 13.7 p=0.003</td>
</tr>
<tr>
<td><strong>Age at onset.</strong></td>
<td>27.8 (26)</td>
<td>29.2 (27)</td>
<td>24.9 (26)</td>
<td>36.9(31)</td>
<td>Chi-sq 5.47 p=0.14</td>
</tr>
</tbody>
</table>
A 10 year follow-up study

<table>
<thead>
<tr>
<th></th>
<th>Absent</th>
<th>Improved</th>
<th>Unchanged</th>
<th>Worse</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>History of unexplained illness.</strong></td>
<td>10/22</td>
<td>9/21</td>
<td>11/17</td>
<td>4/13</td>
</tr>
</tbody>
</table>

**IBQ:**

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>General hypochondriasis</td>
<td>1.95 (2.0)</td>
<td>1.90 (1.0)</td>
<td>2.82 (3.0)</td>
<td>1.77 (1.0)</td>
</tr>
<tr>
<td>Disease conviction</td>
<td>2.59 (3.0)</td>
<td>2.67 (2.0)</td>
<td>2.47 (2.0)</td>
<td>3.15 (4.0)</td>
</tr>
<tr>
<td>Psychological vs. somatic</td>
<td>2.27 (2.0)</td>
<td>1.43 (1.0)</td>
<td>1.47 (1.0)</td>
<td>1.31 (1.0)</td>
</tr>
<tr>
<td>Affective inhibition</td>
<td>2.81 (3.5)</td>
<td>2.62 (3.0)</td>
<td>3.0 (3.0)</td>
<td>2.77 (3.0)</td>
</tr>
<tr>
<td>Affective distress</td>
<td>3.36 (4.0)</td>
<td>2.19 (2.0)</td>
<td>2.53 (3.0)</td>
<td>2.38 (2.0)</td>
</tr>
<tr>
<td>Denial</td>
<td>2.68 (2.0)</td>
<td>3.81 (4.0)</td>
<td>2.82 (3.0)</td>
<td>3.61 (5.0)</td>
</tr>
<tr>
<td>Irritability</td>
<td>2.38 (2.0)</td>
<td>1.52 (1.00</td>
<td>1.76 (2.0)</td>
<td>1.46 (1.0)</td>
</tr>
<tr>
<td>Affective state</td>
<td>7.64 (9.0)</td>
<td>5.62 (5.0)</td>
<td>7.12 (6.0)</td>
<td>5.61 (3.0)</td>
</tr>
<tr>
<td>Disease affirmation</td>
<td>5.32 (5.0)</td>
<td>6.24 (6.0)</td>
<td>6.0 (6.0)</td>
<td>6.85 (6.0)</td>
</tr>
<tr>
<td>Discriminant function</td>
<td>50.1 (50.2)</td>
<td>62.0 (60.7)</td>
<td>57.86 (56.5)</td>
<td>65.45 (64.0)</td>
</tr>
</tbody>
</table>

Table 2.14 Measures of illness behaviour and symptomatic outcome.
2: A 10 year follow-up study

Table 2.15 Symptomatic outcome and indicators of neuropathology.

<table>
<thead>
<tr>
<th></th>
<th>Absent</th>
<th>Improved</th>
<th>Unchanged</th>
<th>Worse</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of signs</td>
<td>1.54(0.5)</td>
<td>1.76(1.0)</td>
<td>2.75(2.0)</td>
<td>2.38(3.0)</td>
<td>Chi-sq. 4.16 (p=0.24)</td>
</tr>
<tr>
<td>Provisional diagnosis</td>
<td>3</td>
<td>3</td>
<td>5</td>
<td>3</td>
<td>n.s.</td>
</tr>
<tr>
<td>Independent diagnosis</td>
<td>0</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>n.s.</td>
</tr>
<tr>
<td>Previous neurological illness</td>
<td>7</td>
<td>5</td>
<td>11</td>
<td>5</td>
<td>n.s.</td>
</tr>
</tbody>
</table>
The number of complaints recorded at presentation, and the proportion of patients presenting a single complaint was similar for each type of symptomatic outcome (table 2.16).

The psychiatric assessments of psychopathology had led to diagnoses in two thirds of the sample. These were of anxiety neurosis, depression, personality disorder or Briquet's syndrome. As table 2.17 indicates, the first two appeared to be associated with relatively good prognosis, and the latter two with a worse one. They were amalgamated for the purpose of statistical comparison with those patients who had not received an additional psychiatric diagnosis (table 2.18) from which it appeared that patients with affective symptoms did considerably better than those with no diagnosis, while those with personality disorder (including Briquet's syndrome) did worst of all.
### Table 2.16 Number of initial complaints and symptomatic outcome.

<table>
<thead>
<tr>
<th>No. of complaints</th>
<th>Absent</th>
<th>Improved</th>
<th>Unchanged</th>
<th>Worse</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.82 (3.0)</td>
<td>3.05 (3.0)</td>
<td>3.47 (3.0)</td>
<td>3.08 (2.0)</td>
<td>Chi-sq.1.59; p=0.66</td>
<td></td>
</tr>
<tr>
<td>Single complaint.</td>
<td>5/22</td>
<td>4/21</td>
<td>4/17</td>
<td>6/13</td>
<td>Chi-sq.3.45  p=0.33</td>
</tr>
</tbody>
</table>

### Table 2.17: Symptomatic outcome according to initial psychiatric diagnoses.

<table>
<thead>
<tr>
<th>No other diagnosis</th>
<th>Anxiety</th>
<th>Depression</th>
<th>Personality Disorder</th>
<th>Briquet's syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>7</td>
<td>5</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>Improved</td>
<td>7</td>
<td>4</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Unchanged</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Worse</td>
<td>9</td>
<td>0</td>
<td>4</td>
<td>0</td>
</tr>
</tbody>
</table>

Totals: 26(36%) 10(14%) 20(27%) 14(19%) 3(4%)
Table 2.18 Symptomatic outcome across the 3 chief psychiatric subgroups.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Anxiety or Depression</th>
<th>Diagnosis: No other diagnosis</th>
<th>Personality disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>14</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>Improved</td>
<td>10</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Unchanged</td>
<td>2</td>
<td>3</td>
<td>12</td>
</tr>
<tr>
<td>Worse</td>
<td>4</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Totals:</td>
<td>30 (41%)</td>
<td>26 (36%)</td>
<td>17 (23%)</td>
</tr>
</tbody>
</table>

ChiSq (6df) 35.46 (p=0.000);
Mantel Haenszel test for linearity ChiSq (1df) 6.66 (p=0.009)
Symptomatic outcome is compared against clinical diagnoses of personality disorder and EPI and HOQ scores in table 2.19. It does not show any evident relationship to the EPI and HOQ assessments. However, when prognosis is categorised as good (symptom improved or absent) or poor (symptom unchanged or worse), presence of diagnosed personality disorder correlated strongly with poor symptomatic outcome (table 2.20).

Of the two affective diagnoses, anxiety disorders were associated with a particularly good prognosis (tables 2.21 and 2.22). However, ratings of mood on the BDI and MACL, and the composite "affective state" score from the IBQ, failed to show any trend linking them to symptomatic outcome.
Table 2.19: Clinical and psychometric measures of abnormal personality with symptomatic outcome. [Mean (median) values for continuous measures].

<table>
<thead>
<tr>
<th>Diagnosis of PD</th>
<th>Absent</th>
<th>Improved</th>
<th>Unchanged</th>
<th>Worse</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPI (N)</td>
<td>12 (14)</td>
<td>11 (10)</td>
<td>12 (13)</td>
<td>10.6 (10)</td>
</tr>
<tr>
<td>EPI (E)</td>
<td>8.8 (8.5)</td>
<td>10.9 (8.0)</td>
<td>10.9 (13)</td>
<td>10.1 (11)</td>
</tr>
<tr>
<td>EPI (L)</td>
<td>3.4 (3.0)</td>
<td>3.6 (3.0)</td>
<td>4.0 (4.0)</td>
<td>3.8 (4.0)</td>
</tr>
<tr>
<td>HOQ</td>
<td>21.4 (20.5)</td>
<td>22.1 (21)</td>
<td>19.5 (22)</td>
<td>20.1 (21)</td>
</tr>
</tbody>
</table>

Table 2.20: Final symptomatic status and diagnoses of personality disorder.

<table>
<thead>
<tr>
<th>No personality disorder</th>
<th>38</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td>Personality disorder</td>
<td>5</td>
<td>12</td>
</tr>
</tbody>
</table>

Chi-Square (1 df) 7.96,  p=0.005
2: A 10 year follow-up study

<table>
<thead>
<tr>
<th>Diagnoses:</th>
<th>Absent</th>
<th>Improved</th>
<th>Unchanged</th>
<th>Worse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety disorder</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Depression</td>
<td>9</td>
<td>6</td>
<td>1</td>
<td>4</td>
</tr>
</tbody>
</table>

**Questionnaire ratings - mean (median) values:**

<table>
<thead>
<tr>
<th></th>
<th>Absent</th>
<th>Improved</th>
<th>Unchanged</th>
<th>Worse</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI score</td>
<td>16.7 (16)</td>
<td>13.4 (9)</td>
<td>17.8 (19)</td>
<td>12.3 (14)</td>
</tr>
<tr>
<td>MACL: anxiety</td>
<td>5.5 (5.5)</td>
<td>3.9 (3)</td>
<td>4.8 (5)</td>
<td>3.0 (3)</td>
</tr>
<tr>
<td>MACL: depression</td>
<td>5.6 (6)</td>
<td>6.1 (5)</td>
<td>7.8 (5)</td>
<td>3.5 (4)</td>
</tr>
<tr>
<td>IBQ: Affective state.</td>
<td>6.58(6)</td>
<td>5.62 (5)</td>
<td>7.12 (6)</td>
<td>5.61 (3)</td>
</tr>
</tbody>
</table>

Table 2.21: Symptomatic outcome and initial assessments of mood.

<table>
<thead>
<tr>
<th>Symptomatic improvement</th>
<th>No symptomatic improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>No affective diagnosis</td>
<td>19</td>
</tr>
<tr>
<td>Anxiety neurosis</td>
<td>9</td>
</tr>
<tr>
<td>Depression</td>
<td>15</td>
</tr>
</tbody>
</table>

Chi-square (2 df) 9.98  p=0.007

Table 2.22 Final symptomatic status vs. initial affective diagnoses.
The combined power of the simple variables assessed here to predict symptomatic outcome was tested using logistic regression analysis. For this purpose, outcome was expressed as a binary variable according to whether the initial symptom improved ('absent' + 'improved'; n=43) or did not improve ('same' + 'worse'; n=30). Input variables were: age; sex; length of symptom history; number of symptoms; whether symptom was single; number of signs at examination; previous history of illness behaviour; previous history of psychiatric treatment; previous diagnosed neurological illness; concurrent neurological diagnosis; concurrent provisional neurological diagnosis; concurrent diagnosis of anxiety or depression; concurrent diagnosis of personality disorder; concurrent psychotropic medication; concurrent non-psychotropic medication.

Logistic regression analysis was carried out using SPSS for Macintosh version 6.0 (Norusis, 1996). Stepwise forwards and backwards analyses were conducted using conditional and likelihood ratio paradigms with items being progressively added or discarded provided entry or removal had a probability for the score statistic of less than 0.1. The solution obtained by each method was identical, yielding an equation from the same 4 variables which successfully predicted 84% of the observed frequencies, with a slight bias in favour of predicting a better outcome than that which was observed:

Probability of the symptom staying the same or being worse after 10 years is given by the equation:

\[
\frac{1}{1 + e^{-Z}}
\]

where: \( Z = 0.084(a) + 0.008(h) + 2.362(m) - 2.162(n) - 3.931 \)

and:

- \( a = \) age in years
- \( h = \) history of symptom in months
- \( m = 1 \) if non-psychotropic medication was being prescribed at assessment
- \( n = 1 \) if depression or anxiety neurosis had been diagnosed.

The goodness of fit of this solution is as follows:

<table>
<thead>
<tr>
<th>Predicted:</th>
<th>% correct:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improved</td>
<td>Not improved</td>
</tr>
<tr>
<td>Improved</td>
<td>38</td>
</tr>
<tr>
<td>Not improved</td>
<td>7</td>
</tr>
</tbody>
</table>

Overall: 83.56%

Chi-square (4 d.f.) = 37.00 (p < 0.000).
2.43 Diagnostic Outcome

Detailed investigation of the later medical records of patients for whom a change in diagnosis was reported confirmed that illness accounting for the original symptom was subsequently diagnosed in 11 of the 73 cases (15%). Salient clinical facts including the diagnoses ultimately made are presented in table 2.23. Comparison of column 6 and column 9 of the table shows that subsequent rediagnosis involved confirmation of a diagnosis suspected at presentation in 5 cases, and diagnosis of an abnormal finding thought to be related to the presenting complaint in a sixth (case no. 5). Where there had been any improvement in the symptom, this reflected treatment in the light of rediagnosis. Although the patient whose aphonia was rediagnosed as carcinoma (no.39) had this reported as 'absent', his efforts at speech after surgical removal of his larynx had not necessarily restored his previous performance. Another patient had been rated as 'unchanged' overall after weakness had worsened but aphonia had disappeared.

The relationship between these two clinical outcome measures is summarised in table 2.24. In nearly all cases where there was a diagnostic change, the original symptom had persisted or worsened.

Patients presenting with seizures were no more likely to have been rediagnosed than those with other initial symptoms (table 2.25).
<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age</th>
<th>Sex</th>
<th>Presentation</th>
<th>Length of history</th>
<th>Additional neurological diagnosis</th>
<th>Additional psychiatric diagnosis</th>
<th>Symptomatic outcome</th>
<th>Final diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>59</td>
<td>M</td>
<td>Weakness + pain</td>
<td>30 years</td>
<td>Probable myelopathy</td>
<td>PTSD</td>
<td>Worse.</td>
<td>Definite myelopathy.</td>
</tr>
<tr>
<td>34</td>
<td>21</td>
<td>M</td>
<td>Falling attacks</td>
<td>5 years</td>
<td>Probable epilepsy.</td>
<td>Nil</td>
<td>Worse.</td>
<td>Epilepsy (and pseudoseizures)</td>
</tr>
<tr>
<td>41</td>
<td>35</td>
<td>F</td>
<td>Multiple weakness</td>
<td>2 weeks (?)</td>
<td>Possible myaesthesia.</td>
<td>Nil</td>
<td>Improved.</td>
<td>Mymasthenia Gravis.</td>
</tr>
<tr>
<td>51</td>
<td>21</td>
<td>M</td>
<td>Seizures</td>
<td>7 years</td>
<td>Probable epilepsy.</td>
<td>Personality disorder.</td>
<td>Unchanged.</td>
<td>Epilepsy.</td>
</tr>
<tr>
<td>5</td>
<td>32</td>
<td>F</td>
<td>Blackouts</td>
<td>16 months</td>
<td>Abnormal scan thought relevant</td>
<td>Nil</td>
<td>Unchanged.</td>
<td>Vascular malformation.</td>
</tr>
<tr>
<td>76</td>
<td>59</td>
<td>F</td>
<td>Loss of sensation.</td>
<td>5 years</td>
<td>Carpal tunnel syndrome.</td>
<td>Depression.</td>
<td>Worse.</td>
<td>Multiple Sclerosis.</td>
</tr>
<tr>
<td>77</td>
<td>35</td>
<td>F</td>
<td>Aphonia + limb weakness.</td>
<td>7 years</td>
<td>Nil</td>
<td>Probable Briquet's syndrome. Weakness worse. (Aphonia absent).</td>
<td>Spinal muscular atrophy.</td>
<td></td>
</tr>
<tr>
<td>39</td>
<td>45</td>
<td>M</td>
<td>Poor balance + slurred speech.</td>
<td>3 years</td>
<td>Nil</td>
<td>Depression.</td>
<td>Absent.</td>
<td>(treated) Carcinoma of larynx.</td>
</tr>
<tr>
<td>10</td>
<td>60</td>
<td>F</td>
<td>Loss of coordination.</td>
<td>5 years</td>
<td>Nil</td>
<td>Nil</td>
<td>Worse.</td>
<td>Spino-cerebellar degeneration.</td>
</tr>
</tbody>
</table>

*Table 2.23: Clinical data for patients whose presenting symptoms were subsequently rediagnosed.*
Table 2.24: Comparison of symptomatic and diagnostic outcome.

<table>
<thead>
<tr>
<th>Diagnostic change</th>
<th>Symptom absent</th>
<th>Symptom improved</th>
<th>Symptom same</th>
<th>Symptom worse</th>
</tr>
</thead>
<tbody>
<tr>
<td>No diagnostic change</td>
<td>21</td>
<td>20</td>
<td>13</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

Table 2.25: Diagnostic outcome and presenting symptom.

<table>
<thead>
<tr>
<th>Presenting symptom:</th>
<th>Symptom rediagnosed</th>
<th>No rediagnosis.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor</td>
<td>6</td>
<td>25</td>
</tr>
<tr>
<td>Seizures</td>
<td>3</td>
<td>24</td>
</tr>
<tr>
<td>Amnesia</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Special senses</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Sensation</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>
Data from the neurological history and examination at presentation compared with diagnostic outcome are summarised in table 2.26. More signs were recorded on clinical examination among patients who were rediagnosed, and if a provisional diagnosis had been made for the complaint by discharge, rediagnosis was also more likely. An inter-relationship between these factors by which higher numbers of signs were recorded among patients given a provisional neurological diagnosis is illustrated in table 2.27.

Of the presenting factors hypothesised to predispose to change in diagnosis, no association was found in the case of age of onset, lack of history of abnormal illness, male sex, and left-sided unilateral symptoms (table 2.28).

Among psychological factors (table 2.29), neither additional psychiatric diagnoses nor psychometric measures of personality or mood were associated with rediagnosis. A recorded history of previous psychiatric treatment was more likely among patients who were rediagnosed. The "discriminant function" score from the Illness Behaviour Questionnaire (cf. section 2.33 (i) was higher among patients whose diagnoses were unchanged, although not to a statistically significant degree.
### Table 2.26: Diagnostic outcome by indicators of covert neuropathology.

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Rediagnosis</th>
<th>No rediagnosis</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (median) no. of signs at examination</td>
<td>3.5 (3.0)</td>
<td>1.77 (1.0)</td>
<td>Mann Whitney U=176.5 p=0.025</td>
</tr>
<tr>
<td>Provisional diagnosis for symptom at discharge</td>
<td>6/11</td>
<td>8/62</td>
<td>Chi-sq. (1 df)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>10.45 p=0.001</td>
</tr>
<tr>
<td>Independent neurological diagnosis</td>
<td>3/11</td>
<td>8/62</td>
<td>n.s.</td>
</tr>
<tr>
<td>Previous neurological illness.</td>
<td>5/11</td>
<td>23/62</td>
<td>n.s.</td>
</tr>
</tbody>
</table>
2: A 10 year follow-up study

<table>
<thead>
<tr>
<th></th>
<th>Neuropathology diagnosed (n=11)</th>
<th>No rediagnosis (n=62)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age at onset</td>
<td>32.6 (30)</td>
<td>28.5 (26)</td>
<td>U=275.5 (1-tailed p=0.161)</td>
</tr>
<tr>
<td>(median age)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absence of past atypical illness.</td>
<td>8/11</td>
<td>31/62</td>
<td>Chi-square 1.94 (1d.f.) p=0.164</td>
</tr>
<tr>
<td>Proportion of males.</td>
<td>4/11</td>
<td>12/62</td>
<td>Chi-square 1.56 (1d.f.) p=0.191</td>
</tr>
<tr>
<td>Proportion of unilateral symptoms on left.</td>
<td>2/4</td>
<td>20/31</td>
<td>Chi-square =0.32 (1d.f.) Fisher's 1 tail p =0.48</td>
</tr>
</tbody>
</table>

Table 2.28: Final diagnostic status and hypothetically associated initial variables.
2: A 10 year follow-up study

<table>
<thead>
<tr>
<th></th>
<th>Neuropathology diagnosed</th>
<th>No rediagnosis</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Past psychiatric treatment</strong></td>
<td>9/11</td>
<td>26/62</td>
<td>Chi-sq. (1df) 5.95 p=0.015</td>
</tr>
<tr>
<td><strong>Diagnosis of:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Personality disorder</td>
<td>3/11</td>
<td>14/62</td>
<td>n.s.</td>
</tr>
<tr>
<td>Anxiety neurosis:</td>
<td>0/11</td>
<td>10/62</td>
<td>n.s.</td>
</tr>
<tr>
<td>Depression:</td>
<td>3/11</td>
<td>17/62</td>
<td>n.s.</td>
</tr>
<tr>
<td><strong>Mean (median) questionnaire scores :</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI</td>
<td>14.1 (16)</td>
<td>15.4 (14)</td>
<td>n.s.</td>
</tr>
<tr>
<td>MACL depression</td>
<td>4.8 (4)</td>
<td>6.4 (5)</td>
<td>n.s.</td>
</tr>
<tr>
<td>MACL anxiety</td>
<td>3.6 (3)</td>
<td>4.6 (4)</td>
<td>n.s.</td>
</tr>
<tr>
<td>IBQ Discriminant function</td>
<td>52.1 (48.1)</td>
<td>59.1 (60.1)</td>
<td>U=247.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>p=0.074</td>
</tr>
</tbody>
</table>

*Table 2.29: Diagnostic outcome and psychopathology.*
An attempt was made to predict diagnostic change in this sample using logistic regression. The dependent variable was whether rediagnosis occurred. The covariates were the same as for the analysis to predict symptom persistence in section 2.32. Forwards and backwards stepwise regression were used for with a conditional paradigm adding and removing variables provided the probability of the score statistic was less than 0.1. The results were identical for either method, as follows:

where: \( Z = 0.111(a) + 4.322(p) + 2.705(i) - 8.024 \)

and:

- \( a = \) age in years
- \( p = 1 \) when a provisional neurological diagnosis had been made
- \( i = 1 \) when an independent neurological disorder was also diagnosed

The goodness of fit of this solution was given by:

<table>
<thead>
<tr>
<th>Predicted</th>
<th>No rediagnosis</th>
<th>Rediagnosis</th>
<th>% correct</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observed:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No rediagnosis</td>
<td>61</td>
<td>1</td>
<td>98.39%</td>
</tr>
<tr>
<td>Rediagnosis</td>
<td>6</td>
<td>5</td>
<td>45.45%</td>
</tr>
</tbody>
</table>

Overall: 90.41%

Chi-square (3 d.f.) 22.66 \( p < 0.000 \)

It is evident that the apparent goodness of fit is deceptive, as fewer than half the 11 cases requiring subsequent were predicted by the model.
2.44. Consultation behaviour and measures of morbidity at follow-up.

The 56 patients completing a follow-up interview provided detailed information about their consultation behaviour in and beyond the NHS. It is summarised in table 2.30, which illustrates the skewed distribution of uptake of each kind of service and their overall range in the sample. The skew by which small numbers of patients accounted for a majority of resources is evident from the final column. This is greatest in the case of relatively unpopular services, viz. use of alternative practitioners, in-patient facilities, and non-medical (“other”) NHS appointments. These "other" NHS professionals were nearly always district nurses or physiotherapists. Among the alternative practitioners consulted, osteopaths and homeopaths were the most popular (table 2.31).

At the same time, the interview requested information about current health, and illness in the intervening period. As table 2.32 shows, new intercurrent illness was no more likely among patients with a poor prognosis for the original symptom, but they were more likely to have had investigations for that symptom and to receive treatment. As tables 2.13 and 2.27 both show, patients with a poorer prognosis were older.
2: A 10 year follow-up study

<table>
<thead>
<tr>
<th>Measure</th>
<th>Range:</th>
<th>25%:</th>
<th>50%:</th>
<th>75%:</th>
<th>No. of patients accounting for 50% of total:</th>
</tr>
</thead>
<tbody>
<tr>
<td>GP visits</td>
<td>0-500</td>
<td>21.25</td>
<td>55</td>
<td>120</td>
<td>13 (32%)</td>
</tr>
<tr>
<td>OP visits</td>
<td>0-180</td>
<td>5</td>
<td>18</td>
<td>40</td>
<td>8 (14%)</td>
</tr>
<tr>
<td>General hosp. (wks)</td>
<td>0-155</td>
<td>0</td>
<td>2</td>
<td>8.5</td>
<td>4 (7%)</td>
</tr>
<tr>
<td>Psychiatric hosp. (wks)</td>
<td>0-60</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Other NHS consultations</td>
<td>0-900</td>
<td>0</td>
<td>3.5</td>
<td>12</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>&quot;Alternative&quot; consultations</td>
<td>0-90</td>
<td>0</td>
<td>0</td>
<td>1.75</td>
<td>2 (4%)</td>
</tr>
</tbody>
</table>

Table 2.30: Distribution of consultation rates within the sample.

<table>
<thead>
<tr>
<th>Type of practitioner</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteopath</td>
<td>9</td>
</tr>
<tr>
<td>Homeopathist</td>
<td>7</td>
</tr>
<tr>
<td>Acupuncturist</td>
<td>4</td>
</tr>
<tr>
<td>Herbalist/naturopath</td>
<td>3</td>
</tr>
<tr>
<td>Hypnotist</td>
<td>2</td>
</tr>
<tr>
<td>Chiropractor</td>
<td>1</td>
</tr>
<tr>
<td>Faith healer</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 2.31: Use of alternative practitioners. (n=19; 5 patients consulted more than one type).
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent (n=15)</td>
<td>7.0 (8)</td>
<td>2.67 (2)</td>
<td>2.7 (2)</td>
<td>2 (13%)</td>
<td>0 (0%)</td>
<td>3 (20%)</td>
<td>4 (27%)</td>
<td>8 (53%)</td>
<td>10 (66.7%)</td>
</tr>
<tr>
<td>Improved (n=19)</td>
<td>6.6 (7)</td>
<td>2.73 (2)</td>
<td>2.6 (2)</td>
<td>9 (47%)</td>
<td>3 (16%)</td>
<td>5 (26%)</td>
<td>8 (42%)</td>
<td>7 (37%)</td>
<td>12 (63%)</td>
</tr>
<tr>
<td>Unchanged (n=13)</td>
<td>5.77 (6)</td>
<td>4.85 (5)</td>
<td>5.23 (5)</td>
<td>13 (100%)</td>
<td>9 (69%)</td>
<td>8 (61.5%)</td>
<td>5 (38.5%)</td>
<td>6 (46%)</td>
<td>7 (54%)</td>
</tr>
<tr>
<td>Worse (n=9)</td>
<td>6.9 (8)</td>
<td>4.89 (5)</td>
<td>5.0 (6)</td>
<td>7 (78%)</td>
<td>4 (44%)</td>
<td>7 (78%)</td>
<td>6 (66.7%)</td>
<td>5 (57%)</td>
<td>2 (22%)</td>
</tr>
<tr>
<td>Chi square:</td>
<td>3.72</td>
<td>12.74</td>
<td>27.28</td>
<td>29.28</td>
<td>21.66</td>
<td>14.05</td>
<td>3.78</td>
<td>1.38</td>
<td>6.97</td>
</tr>
<tr>
<td>Significance:</td>
<td>0.29</td>
<td>0.004</td>
<td>0.000</td>
<td>0.000</td>
<td>0.001</td>
<td>0.002</td>
<td>0.28</td>
<td>0.71</td>
<td>0.073</td>
</tr>
</tbody>
</table>

Table 2.32: Findings at the end of the study period. (n=56)
(values of continuous variables given as mean and (median)).
Comparisons between consultation behaviour and the two clinical outcome measures showed that patients whose diagnoses were subsequently revised were likely to have made more outpatient visits, to have spent more weeks in hospital over the follow-up period, and to have had more visits to or from other NHS staff (Table 2.33).

Comparison of these consultation measures with symptomatic outcome shows that although there is a tendency for patients whose symptoms persist to have made more use of out-patient and in-patient facilities, they make greatest additional demand on general practitioners (table 2.34).
### Table 2.33: Use of services by diagnostic outcome (mean (median) totals for 10 year study period)

<table>
<thead>
<tr>
<th>Diagnostic Outcome</th>
<th>GP visits</th>
<th>Outpatient visits</th>
<th>Weeks in Hospital</th>
<th>Other NHS visits</th>
<th>&quot;Alternative&quot; visits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unchanged diagnosis:</td>
<td>77.6 (50)</td>
<td>30.0 (12)</td>
<td>10.6 (1.0)</td>
<td>42.6 (1.0)</td>
<td>3.3 (0)</td>
</tr>
<tr>
<td>Rediagnosis for presenting symptom:</td>
<td>89.4 (100)</td>
<td>51.1 (36.5)</td>
<td>16.6 (10.5)</td>
<td>78.4 (13.5)</td>
<td>17.6 (0)</td>
</tr>
</tbody>
</table>

#### Significance (2-tailed):
- \( U = 152; \ p = 0.35 \)
- \( U = 101; \ p = 0.032 \)
- \( U = 79; \ p = 0.007 \)
- \( U = 101; \ p = 0.026 \)
- \( U = 172; \ p = 0.57 \)

#### Table 2.34: Use of services over follow-up period by symptomatic outcome.

<table>
<thead>
<tr>
<th>Symptomatic Outcome</th>
<th>GP visits</th>
<th>OP visits</th>
<th>Weeks in hospital</th>
<th>Other NHS visits</th>
<th>Alternative practitioners</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>38.5(30)</td>
<td>22.5(5)</td>
<td>2.1(1)</td>
<td>40 (6)</td>
<td>3.1(0)</td>
</tr>
<tr>
<td>Improved</td>
<td>82.1(40)</td>
<td>32.5(16)</td>
<td>6.1(1)</td>
<td>1.8(0)</td>
<td>4.6(0)</td>
</tr>
<tr>
<td>Unchanged</td>
<td>127.7(120)</td>
<td>41.2(26)</td>
<td>32.2 (7)</td>
<td>143.1(12)</td>
<td>5.2(0)</td>
</tr>
<tr>
<td>Worse</td>
<td>71.7(45)</td>
<td>39.4(35)</td>
<td>9.2 (4)</td>
<td>19.6(3)</td>
<td>11.1(1)</td>
</tr>
</tbody>
</table>

#### Significance (Kruskall-Wallis):
- Chi-sq.14.29 (p=0.002)
- Chi-sq.7.03 (p=0.071)
- Chi-sq.8.39 (p=0.039)
- Chi-sq.12.12 (p=0.012)
- Chi-sq.1.92 (p=0.59)
Relatively few patients accounted for the use of in-patient and "other" NHS services and of "alternative" practitioners. The greater uptake of GP and OP services among the sample differed from one another in their distribution. Both were examined when surveying the factors at presentation and follow-up hypothesised to influence consultation behaviour.

Of the factors from the original clinical assessment, neither presence of an additional neurological or psychiatric diagnosis, nor of a provisional neurological diagnosis, nor concurrent treatment was associated with consultation behaviour (Table 2.35).

One final item from analysis of the patients' initial clinical data concerned the recorded prescription of non-psychotropic medication. As table 2.36 shows, patients already receiving medication on presentation were likely to have higher GP consultation rates than, but similar OP consultation rates to, patients who were not receiving such medication then over the follow-up period.
Table 2.35: Consultation behaviour (GP visits and Outpatient visits) and diagnostic status on entry.

<table>
<thead>
<tr>
<th></th>
<th>Mean GP visits (median)</th>
<th>Mean OP visits (median)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Independent neurological diagnosis:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present (n=9)</td>
<td>73.0 (90.0)</td>
<td>12.9 (10.0)</td>
</tr>
<tr>
<td>Absent (n=47)</td>
<td>80.5 (50.0)</td>
<td>36.8 (20.0)</td>
</tr>
<tr>
<td>n.s.</td>
<td></td>
<td>n.s.</td>
</tr>
<tr>
<td><strong>Provisional neurological diagnosis:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present (n=12)</td>
<td>92.1 (100.0)</td>
<td>36.5 (27.0)</td>
</tr>
<tr>
<td>None (n=44)</td>
<td>75.8 (50.0)</td>
<td>32.0 (12.0)</td>
</tr>
<tr>
<td>n.s.</td>
<td></td>
<td>n.s.</td>
</tr>
<tr>
<td><strong>Psychiatric diagnosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Additional diagnosis (n=36)</td>
<td>86.4 (50.0)</td>
<td>34.8 (13.5)</td>
</tr>
<tr>
<td>No other diagnosis (n=20)</td>
<td>66.5 (60.0)</td>
<td>29.65 (22.0)</td>
</tr>
<tr>
<td>n.s.</td>
<td></td>
<td>n.s.</td>
</tr>
</tbody>
</table>

Table 2.36: Consultation behaviour and initial medication.

<table>
<thead>
<tr>
<th></th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Receiving non-psychotropic medication.</td>
<td>102.8 (100)</td>
<td>33.7 (20)</td>
</tr>
<tr>
<td>Not receiving medication.</td>
<td>69.1 (40)</td>
<td>32.6 (12)</td>
</tr>
</tbody>
</table>

U=176;p=0.005 n.s.
Analysis of the relationship of initial measures of illness behaviour to subsequent consultation behaviour showed a disparity between questionnaire and historical indices. None of the original IBQ ratings appeared to predict subsequent consultation behaviour (table 2.37). However, clinical history of previous illness behaviour was positively correlated with GP attendance over the follow-up period (table 2.38).

Standardised assessments at follow-up had been in the form of Othmer and DeSouza's 'screening test' for SD used with 56 patients during the interview (cf. section 2.33(i)) and the three postal questionnaires subsequently returned by 52 of the sample. Overall scores on the screening test yielded a mean of 2.41 (median 2.0) and a range of 0 to 7.

Their association with the symptomatic and diagnostic outcome are summarised in tables 2.39 and 2.40 respectively. Somatisation index scores worsen with symptomatic outcome through the sample, although even lower scores were recorded for the group whose symptoms had improved rather than disappeared. In table 2.39, a non-parametric comparison between two groups, those whose symptoms did not improve and those whose symptoms did, showed the former's scores, with a mean and median of 3.0, to be demonstrably higher.

The scores of the 52 patients who completed the Illness Behaviour Questionnaire and the Beck Depression Inventory at follow-up are summarised in table 2.41. The rank correlation coefficients (column 3) confirm a close relationship between initial scores and follow-up scores except for the "affective inhibition" and "irritability" subscales.

Among the quantified items assessed at follow-up, the global assessments of disability by patients themselves and the interviewer showed an equally strong correlation with reported consultations, particularly out-patient consultations. A comparable positive correlation with consultations was obtained from scores on the 7-item "somatisation index", while patients' own ratings of their health showed a significant association with outpatient but not GP visits (table 2.42).
<table>
<thead>
<tr>
<th>IBQ scale</th>
<th>GP visits</th>
<th>Outpatient visits</th>
</tr>
</thead>
<tbody>
<tr>
<td>General hypochondriasis</td>
<td>-0.37 (0.39)</td>
<td>-0.11 (0.22)</td>
</tr>
<tr>
<td>Disease conviction</td>
<td>-0.25 (0.03)</td>
<td>-0.10 (0.23)</td>
</tr>
<tr>
<td>Psychological/somatic focus</td>
<td>0.05 (0.35)</td>
<td>0.04 (0.38)</td>
</tr>
<tr>
<td>Affective inhibition</td>
<td>-0.01 (0.46)</td>
<td>0.08 (0.27)</td>
</tr>
<tr>
<td>Affective disturbance</td>
<td>-0.07 (0.31)</td>
<td>-0.05 (0.35)</td>
</tr>
<tr>
<td>Denial</td>
<td>-0.02 (0.44)</td>
<td>0.06 (0.34)</td>
</tr>
<tr>
<td>Irritability</td>
<td>-0.07 (0.31)</td>
<td>-0.09 (0.25)</td>
</tr>
<tr>
<td>Affective state</td>
<td>-0.07 (0.30)</td>
<td>-0.11 (0.22)</td>
</tr>
<tr>
<td>Disease affirmation</td>
<td>-0.21 (0.056)</td>
<td>-0.10 (0.23)</td>
</tr>
</tbody>
</table>

*Table 2.37: Consultation behaviour and initial IBQ scores. Spearman correlation coefficients (1-tailed p).*

<table>
<thead>
<tr>
<th>Previous AIB</th>
<th>No previous AIB</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>GP visits</td>
<td>113.4 (100)</td>
<td>51.8 (40)</td>
</tr>
<tr>
<td>OP visits</td>
<td>33.0 (18)</td>
<td>31.0 (16)</td>
</tr>
</tbody>
</table>

*Table 2.38. Consultation behaviour and past illness behaviour.*
### Symptomatic outcome compared with somatisation index score

<table>
<thead>
<tr>
<th>Symptomatic outcome</th>
<th>Mean (median) S.I. score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent (n=15)</td>
<td>2.47 (2.0)</td>
</tr>
<tr>
<td>Improved (n=19)</td>
<td>1.63 (1.0)</td>
</tr>
<tr>
<td>Unchanged (n=13)</td>
<td>3.08 (3.0)</td>
</tr>
<tr>
<td>Worse (n=9)</td>
<td>3.0 (3.0)</td>
</tr>
</tbody>
</table>

Chi square = 7.115 (3df) p=0.068

| Absent or improved (n=34) | 2.01 (2.0) |
| Unchanged or worse (n=22) | 3.04 (3.0) |

U=250; 2-tail p = 0.035

*Table 2.39 Symptomatic outcome compared with somatisation index score.*

### Diagnostic outcome and somatisation index scores (n=56)

| Diagnosis revised (n=8) | 2.50 (2.5) |
| Diagnosis unchanged (n=48) | 2.40 (2.0) |

U=180 2-tailed p = 0.77

*Table 2.40 Diagnostic outcome and somatisation index scores.*
Table 2.41: Initial and final scores on Illness Behaviour Questionnaire and Beck Depression Inventory (n=52).
### Table 2.42 Consultation behaviour and quantified assessments at follow-up.
*(Spearman correlation coefficients)*

<table>
<thead>
<tr>
<th>Patients' health rating</th>
<th>GP visits</th>
<th>Out-patient visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-0.18 (p=0.08)</td>
<td>-0.33 (p=0.065)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Patients' disability rating</th>
<th>GP visits</th>
<th>Out-patient visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.37 (p=0.025)</td>
<td>0.47 (p=0.001)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Assessor's disability rating</th>
<th>GP visits</th>
<th>Out-patient visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.39 (p=0.001)</td>
<td>0.36 (p=0.003)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Score on 'Briquet' index.</th>
<th>GP visits</th>
<th>Out-patient visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.31 (p=0.08)</td>
<td>0.35 (p=0.004)</td>
</tr>
</tbody>
</table>

### Follow-up IBQ scores (n=52):

<table>
<thead>
<tr>
<th>1. General hypochondriasis</th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.17 (p=0.12)</td>
<td>0.10 (p=0.24)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>2. Disease conviction</th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.09 (p=0.26)</td>
<td>0.07 (p=0.31)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>3. Psychological/somatic focus</th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-0.07 (p=0.30)</td>
<td>-0.16 (p=0.13)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>4. Affective inhibition</th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.02 (p=0.45)</td>
<td>0.15 (p=0.16)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>5. Affective distress</th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-0.02 (p=0.44)</td>
<td>0.18 (p=0.11)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6. Denial</th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-0.24 (p=0.05)</td>
<td>0.11 (p=0.21)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>7. Irritability</th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.08 (p=0.30)</td>
<td>0.06 (p=0.34)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Affective state</th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.10 (p=0.25)</td>
<td>0.15 (p=0.15)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Disease affirmation</th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.11 (p=0.23)</td>
<td>0.14 (p=0.17)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Follow-up BDI score</th>
<th>GP visits</th>
<th>OP visits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.19 (p=0.09)</td>
<td>0.13 (p=0.18)</td>
</tr>
</tbody>
</table>

*Table 2.43: Consultation behaviour and follow-up IBQ and BDI scores (n=52).*
Other data was provided by the questionnaires returned by patients completing the follow-up. None of the scales of the Illness Behaviour Questionnaire showed a significant relationship to reported consultation in the expected direction, nor did the BDI scores (table 2.43). On the Nottingham Health Profile, several subscales correlated with attendance, notably "social isolation" and "physical mobility" with both GP and OP visits, and "pain" with OP visits. Its second part, a simple 6 item inventory of current perceived problems, was also able to discriminate well for high attenders (table 2.44).
<table>
<thead>
<tr>
<th></th>
<th>Mean score</th>
<th>Correlation with GP visits (p)</th>
<th>Correlation with OP visits (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;Energy&quot;</td>
<td>42.9</td>
<td>0.23 (0.052)</td>
<td>0.31 (0.016)</td>
</tr>
<tr>
<td>&quot;Pain&quot;</td>
<td>23.7</td>
<td>0.32 (0.014)</td>
<td>0.34 (0.008)</td>
</tr>
<tr>
<td>&quot;Emotional reactions&quot;</td>
<td>25.4</td>
<td>0.29 (0.02)</td>
<td>0.24 (0.049)</td>
</tr>
<tr>
<td>&quot;Sleep&quot;</td>
<td>30.2</td>
<td>0.26 (0.037)</td>
<td>0.12 (0.21)</td>
</tr>
<tr>
<td>&quot;Social isolation&quot;</td>
<td>18.2</td>
<td>0.38 (0.003)</td>
<td>0.33 (0.010)</td>
</tr>
<tr>
<td>&quot;Physical mobility&quot;</td>
<td>22.8</td>
<td>0.41 (0.002)</td>
<td>0.39 (0.003)</td>
</tr>
<tr>
<td>&quot;Part II&quot;</td>
<td>2.5</td>
<td>0.26 (0.037)</td>
<td>0.39 (0.003)</td>
</tr>
</tbody>
</table>

Table 2.44 Consultation behaviour and follow-up scores on the Nottingham Health Profile (n=52).
2.5 Discussion.

An appraisal of the methods used in the study (section 2.51) introduces a reconsideration of the hypotheses outlined in section 2.2 in the light of the findings summarised in section 2.4. The hypotheses concerning symptomatic and diagnostic outcome and subsequent consultation behaviour are discussed in turn in sections 2.52 to 2.54. Some further clinical implications of the study are summarised in section 2.55.

2.51. Method of the study.

Entry to the study followed neurological and psychiatric assessment, the latter being augmented by a small battery of psychological questionnaires. These will be commented on before the design and procedures of the follow-up phase are discussed with attention to implications for future studies.

(i) Initial assessments used in the study.
a) Neurological assessment. The present study benefitted from routine assessment procedures at the National Hospitals which ensured physical examinations were recorded in all cases in a standardised booklet in which the location of physical signs was marked on diagrams. This facilitates collection of consistent data concerning their quantity and laterality. Examiners had been assiduous in completing these booklets, and it is unfortunate from the perspective of the present study that the only item omitted with any frequency (being listed at the bottom of a page) concerned patients' handedness. Conversely, information on the number of symptoms that patients presented with depended upon the fulness of freehand notes made by their interviewer, although the standardised history sheet did require an enquiry after each major bodily system.

In collating neurological diagnoses - provisional as well as definitive ones - the admission summary at discharge provided the definitive diagnosis. The study indicates these diagnoses have considerable prognostic significance given the association between "provisional" diagnoses on discharge and a greater likelihood of subsequent unequivocal diagnosis of neurological disease. It might be argued that the consistency of the neurological diagnoses used here (particularly the extent to which provisional diagnoses would be hazarded in the absence of clear evidence) would vary between the 9 consultants whose patients had participated in the study. The circulation of staff in training between firms in this teaching institution could be expected to increase consistency between discharge diagnoses, but ideally all diagnoses would have been agreed by the same panel of trained neurologists at the time of the original admissions.
b) Psychiatric assessments. Although standardised diagnostic criteria were not used on psychiatric examination, the initial psychiatric diagnoses (table 2.5) were all made by the same psychiatrist. The diagnoses were recorded for the study after the original clinical notes were reinspected by the author. This confirmed only one diagnosis had been made in each of the cases having a psychiatric diagnosis additional to comments on "hysteria", "illness behaviour" or "psychogenic symptoms" except for two of the cases of suspected Briquet's syndrome, where abnormal personality was also commented on. Ideally, to maximise the reliability of the initial diagnoses, psychiatric examinations would have been recorded using a standardised schedule to allow diagnoses to be made in accordance with research criteria. A particularly significant lack turned out to be the absence of specific, structured enquiry into candidate symptoms for somatisation disorder.

The quantified assessments of mood, personality, and abnormal illness behaviour provided data of established reliability in addition to the initial clinical assessments of the same factors. In a previous study of the same subjects (Wilson Barnett and Trimble, 1985), these had failed to discriminate hysterical patients from those with neurological or psychiatric diagnoses. One explanation of this might be that the majority of these "hysterical" patients also had other psychiatric conditions. However, the questionnaire measures were unable to distinguish between those having diagnoses of personality disorder within the sample from those who did not, and the mood measures were unable to distinguish between those who had affective diagnoses and those who did not (table 2.8). One clue to the insensitivity of the Beck Depression Inventory here is the high correlation between measurements from a given individual despite a gap of 10 years (table 2.41) indicating it may be more of a trait than a state measure.

As this study shows that these initial clinical diagnoses of psychiatric disorder were of considerable prognostic significance, future follow-up studies might be advised to use instruments that correspond more closely with clinical diagnoses. The association here with clinical evidence of personality disorder suggests that, instead of the EPI and HOQ, inventories more closely aligned with diagnostic categories of personality disorder, such as the Personality Assessment Schedule (Tyrer et al, 1979) would be preferable. Although cumbersome, the latter has been successful in identifying personality deviations among chronic somatising patients (Stern et al, 1993).

The Illness Behaviour Questionnaire had failed to show a correlation with a recorded history of illness behaviour in the previous study on this cohort (Wilson Barnett and Trimble, 1985). Its failure here to associate with any of the three kinds of outcome in the present study (cf. tables 2.14; 2.29; 2.37) appears more significant alongside evidence that the clinical history of illness behaviour is of value when predicting higher rates of consultation behaviour (table 2.38). Even repeating the IBQ at the time that information about consultation behaviour was collected did not provide any evidence of a link between them. As table 2.44 confirms, the Nottingham Health Profile was able to predict attendance with considerable accuracy (as it
A 10 year follow-up study has in other contexts) suggesting that this was not a necessary defect of questionnaires, but that the IBQ simply does not measure the same parameters as clinical assessment of abnormal illness behaviour in these patients. The present study therefore provides further grounds for arguing that the validity of this questionnaire needs to be reassessed.

A major weakness of the study of Slater & Glithero (1965) -- the absence in most cases of a psychiatric opinion, let alone an independent prospective diagnosis -- has been remedied here. Optimally, all index diagnoses in a prognostic study would be made by consultation between the same neurologist and psychiatrist, a procedure developed in the diagnostic study of Creed et al (1990).

(ii) Design of study. The study was designed to provide a pragmatic way of assessing outcome and relating this to initial factors. The initial measures were already largely determined by the information available. Apart from the specific comments already made concerning limitations in the clinical and psychometric assessments, investigation of factors relating to subsequent consultation behaviour would have benefitted from more detailed information concerning past patterns of consultation among the cohort.

The follow-up procedures adopted in the study were also sub-optimal in important respects, although they allowed the study to proceed with relatively limited resources. The decision to base main assessment of clinical outcome on GP replies was vindicated by the high response rate. The quality of the information they provided was inevitably tempered by differences in GP's awareness of their patients evident from their responses. (Some were very recently acquainted with charges who had transferred to their list: others were unusually familiar, to the point of contempt). The difficulties frequently experienced in tracing a patient's current general practitioner introduced unexpected obstacles to the plan to standardise the length of the follow-up period. After attempting to trace GPs as far as possible through the transfer records of Family Practitioner Committees, it was still necessary in 19 cases to use the NHS central register. One consequence of this was that it could take up to a year longer to successfully make contact with a general practitioner by exhausting ordinary channels and then taking this route, compared with a GP who responded immediately to a first contact at the same time. This proved a major source of variation within the follow-up interval. Apart from differences in the time at which GPs were contacted, the effective length of follow-up was dependent upon the recency of a patient's last contact with their GP. The impact of this was restricted by excluding any patients who had not seen their GP in the previous 2 years. However, the fact that some patients may not have consulted their GP for periods up to 2 years prior to follow-up is likely to have biased reported symptomatic outcomes in favour of poorer outcomes as remissions were still being recorded as late as the 8th year of follow-up (cf. table 2.12). In all these cases, remissions were taken to be permanent, which may not have been the case as GPs were not questioned further about the time course of the index
2: A 10 year follow-up study

symptom. (None of the patients complying with later stages of follow-up did report a return of the original symptom after a period of total absence, however).

Reports from other agencies were always obtained when a change in diagnosis for the original symptom had occurred. The chief disadvantage of the telephone interviews (as used with most subjects at follow-up) over face to face meetings was loss of the opportunity to conduct detailed physical or mental state examinations. There was little doubt from subjects' responses to the initial questionnaire and on the telephone that this would have entailed an even larger drop-out rate than that experienced for the telephone interviewing stage. Nevertheless, such direct examinations would be necessary to exclude any cases where neuropathology had emerged that had not come to the attention of a patient's GP or other medical attendants. However, rediagnosis was always associated with persistence of the original symptom at least until the time of rediagnosis. The study has also linked symptom persistence to particularly high consultation rates with GPs and at out-patient departments (cf. table 2.34) whether or not diagnosis was changed, making it very unlikely such patients have failed to have a necessary diagnosis made as a result of medical neglect.

(iii) Follow-up measures. The study has attempted to reconcile some of the contradictions between previous studies, which concentrate either on changes in diagnosis (eg. Slater, 1965; Watson & Buranen, 1980) or symptomatic outcome (eg.Ljungberg,1957) to the relative exclusion of the other. However, with either of these, there has been little consistency among past studies as to how they should be assessed. The report of Watson and Buranen (1980), which reports 10 out of 40 men followed up to have been "false positives", is useful in identifying 4 different sub groups at follow-up within this population: cases may be judged by the raters to have changed or not changed diagnosis, and they may be judged by attending doctors to have changed diagnosis. Watson & Buranen's figures yield the following breakdown:

<table>
<thead>
<tr>
<th></th>
<th>Change made by patient's Dr.</th>
<th>No change made by patient's Dr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rater: diagnosis changed</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Rater: no change</td>
<td>4</td>
<td>30</td>
</tr>
</tbody>
</table>

Table 2.45: The nature of 'false positive' diagnoses in a follow-up of patients with hysteria (after Watson & Buranen, 1980) (n=40).

Adding together all cases where a diagnostic change is sanctioned either by raters or by the researcher carries a high presumption of change, and does not necessarily justify these authors' statement that 25% of diagnoses had changed. Alternative figures here would be 22.5% (attending clinicians' opinion); 15% (the researchers' opinion); or 12.5% (when
clinicians' opinion is confirmed by raters). Slater & Glithero's reports do not make their procedure for determination of diagnosis on follow-up plain. However, it is apparent that they accepted diagnostic changes that had been made, as well as feeling free to make their own presumptive diagnoses of systemic disorders on follow-up. This would strongly bias their judgments in favour of diagnostic change, as did their unquestioning tendency to attribute the original symptom to any serious illness that supervened. The error is particularly serious as they had not applied comparable diagnostic standards in making the index diagnoses.

In the present study, the changes in diagnosis recorded as such (table 2.23) respected current medical attendants' opinions. The involvement of the investigator was restricted to confirming that each GP's diagnostic statement that represented a change on the original diagnosis was supported either by specialists who had been consulted or by new investigations. (There were 2 cases in which GP presumptions of "epilepsy" contradicted specialist opinion and a further one in which longstanding intermittent weakness had been attributed to "fibromyalgia" without any additional evidence following a change of GP).

The relative lack of attention paid to psychiatric diagnoses at follow-up may appear to be a weakness of the study. Ideally, patients would have had a psychiatric assessment at follow-up to assess the nature and incidence of new psychopathology, and the circumstances in which new psychiatric illness was most likely to arise. However, it was quickly evident that, even if ethical approval had been obtained for detailed psychiatric enquiries, respondents would not have complied with attempts to reassess this directly as they had done for the other components of the investigation. In addition, several disparities between patients' reports and those of their GPs concerning subsequent psychiatric help (in either direction) suggested these historical data were less reliable.

It remains possible to comment on the data available. Of the 56 patients who completed the 2nd stage of the follow-up, 23 admitted to psychiatric consultation during the 10 year follow-up period. Of these, only 6 had not had an additional psychiatric diagnosis made when they were assessed on their index admission, and 4 out of these 6 had had psychiatric treatment in the past. Even if these 2 cases might be presumed to have developed psychiatric illness for the first time at presentation, they had retained their original symptom with the implication that any psychiatric treatment had not brought symptomatic relief while no neurological pathology had been diagnosed. There was little evidence that psychiatric illness occurring for the first time substituted for a symptom that improved or remitted. Although Slater & Glithero (1965) argue that primary psychiatric diagnoses were appropriate for 10 of the patients in their follow-up study, they did this on the basis that, in retrospect, it should have been diagnosed on presentation (rather than supervening at a later stage) in a quarter of those whose symptoms did not give way to neurological disease. (The presumption that psychiatric diagnoses were exclusive of neurological disease did not hold in the present study: see section 2.53(ii) below).
(iv) Analysis of results.

The sections that follow describe the evaluation of the hypotheses stated in sections 2.21 to 2.23 in the light of data comprehensively summarised in the "results" section. Because of the number of hypotheses independently tested, a lower probability than 0.05 should ideally be taken to indicate a significant finding. The results for symptomatic outcome have been presented to allow study of all four possible outcomes. Distribution of the data did not justify the use of parametric tests.

In order to assess the relative impact of all the initial variables on the two clinical outcome measures without reference to the study hypotheses, logistic regression analyses were performed, necessarily restricted to discrimination between cases with symptomatic improvement (including remission) and those without. The solution selecting 4 factors (age; length of previous history; use of non-psychotropic medication and a primary psychiatric diagnosis of depression or anxiety) produced a good fit in which the projected solution was sensitive and adequately selective. The logistic regression solution for rediagnosis, identifying a provisional neurological diagnosis; age; and a concurrent neurological diagnosis as joint predictors of diagnostic change was insufficiently sensitive. However, no logistic regression model predicted diagnoses would be changed in more than 5 cases, as opposed to the 11 that were actually amended, limiting their utility. This is likely to have reflected the same constraint that limited the testing of hypotheses concerning diagnostic outcome, viz. the relatively small number of cases in the subgroup whose diagnoses were changed.

2.52 Hypotheses concerning symptomatic outcome.

Table 2.46 summarises the implications of the results in section 2.42 for the hypotheses outlined in section 2.21. Individual hypotheses are discussed below:

(i) Symptoms would persist when previous history indicated the symptom's appearance was not a new event:

(I) The symptom was already chronic at the time of presentation.

As data in table 2.13 shows, the median duration of the symptom by the time of assessment increases as subsequent prognosis worsened. The table also demonstrates a similar tendency with increase in age at assessment. The inference that patients' age had an influence that was at least partly independent of symptom duration, is compatible with several explanations. These include the idea that different psychological factors account for selective symptom persistence as opposed to the appearance of symptoms (the hypothesis of secondary gain, Freud (1909)); that chronic somatic symptoms are an aspect of personality deviation rather than remitting illness (Kaminsky & Siauveney, 1976; Stern et al, 1993); or that symptoms become chronic as a consequence of enduring physiological change (Horvath et al, 1980).
Hypothesis: Finding:

Symptom persistence was expected when ...

(i) Previous history indicated the symptom's appearance was not a new event:
   (I) The symptom was already chronic at the time of presentation. ++
   (II) The patient had a prior history of "abnormal illness behaviour":
        abnormal attitudes to illness were detected by the Illness Behaviour Questionnaire at presentation: 0

(ii) There were relatively stronger grounds for suspecting neurological disease:
   (I) The primary symptom was seizures rather than weakness or loss of sensation. 0
   (II) There was evidence suggestive of CNS pathology either on initial physical examination, or in the diagnoses made on presentation 0

(iii) Psychiatric examination was consistent with a tendency to have chronic hysterical symptoms:
   (I) The patient complained of more than one medically unexplained symptom. 0
   (II) There was independent evidence of personality disorder. +
   (III) There was little evidence of affective distress at presentation. +

Table 2.46: Summary of hypothesis testing - symptomatic outcome.

| Strong support = ++ |
| Weak but significant support = + |
| No support = 0 |
The patient had a prior history of "abnormal illness behaviour" and abnormal attitudes to illness were detected by the Illness Behaviour Questionnaire at presentation.

The study appears not to support the hypothesis that symptomatic outcome can be predicted from initial assessments of "abnormal illness behaviour". The failure of a recorded history of episodes of undiagnosed illness to discriminate within these patients as a group is not wholly surprising, as they failed to distinguish them from a control group of patients whose symptoms were not diagnosed as hysteria in a previous study (Wilson Barnet & Trimble, 1985). The same could be said for the apparent similarity of initial scores on the Illness Behaviour Questionnaire (table 2.14) between the different outcome groups. There was a consistent but non-significant trend for the composite score for disease affirmation to be greater among patients whose symptoms persisted. (i.e. these patients were more likely to believe they were ill, and to feel their problems were somatic rather than psychological in nature).

The presenting symptom would persist when there were relatively stronger grounds for suspecting neurological disease:

(I) The primary symptom was seizures rather than weakness or loss of sensation.

The lack of difference in outcome between the most common kinds of presenting symptom (table 2.11) stands in contrast to previous work (table 2.2). In accounting for what seems to have been an unusually good prognosis for the patients with pseudo-epileptic seizures, an observation of Slater and Glithero (1965) is relevant. Most cases in their sample who were given provisional neurological diagnoses, and subsequently had these confirmed while their symptoms persisted, had epileptic seizures. In the present study, patients who had provisional neurological diagnoses confirmed were as likely to have presented with weakness as with seizures (table 2.25), removing this bias in data on symptomatic prognosis. Improvements in these patients' initial diagnosis relative to other studies may be responsible for this, perhaps through more accurate diagnosis of epilepsy, or greater caution in diagnosing hysteria.

(II) There was evidence suggestive of CNS pathology either on initial physical examination, or in the diagnoses made on presentation.

There was a trend among all 4 chosen indicators of CNS pathology for them to be associated with poor outcome (i.e. 'unchanged' or 'worse'). This was most marked for the number of signs present on initial examination (table 2.15). However, the association of these factors with rediagnosis was far stronger (cf. section 2.53 below) and as rediagnosis itself was associated with a poor symptomatic prognosis (table 2.24) this is taken to be the basis for association with poorer outcome here.
Symptom persistence was expected when psychiatric examination was consistent with a tendency to have chronic hysterical symptoms:

(I) The patient complained of more than one medically unexplained symptom.

The lack of association between prognosis and number of symptoms, or whether a single symptom only was present, did not support this hypothesis (table 2.16). Despite the absence of a clear link between polysymptomatology and chronicity in the sample as a whole, all 3 patients who were initially diagnosed as "Briquet's syndrome" had very poor symptomatic outcomes. However, as one of these subsequently developed illness to account for some of the symptoms attributed to Briquet's syndrome, they do not provide convincing evidence for the predictive validity of this clinical diagnosis either (cf. section 1.5).

One possible explanation by which a link between the number of reported symptoms and their persistence remains possible would be for it to apply within a well circumscribed subgroup of patients whose symptom count exceeded a high threshold. This of course would be consistent with Perley & Guze's (1962) findings, and Guze's subsequent distrust of the dilution of these criteria as Briquet's syndrome was reformulated as somatisation disorder (cf. discussion in section 1.5). However, the methodology in the present study could be at fault, as the independent clinical diagnoses of "Briquet's syndrome" made in three cases identified only one patient having a very high number of symptoms recorded in her notes, while the mean number of symptoms recorded among the three patients with the diagnosis was only 4.67. The use of a standardised symptom checklist in these initial assessments might have prompted redistribution of the symptom scores, although it seems unlikely that many further diagnoses of Briquet's syndrome would have been made.

The relevance of the study's findings to the diagnosis of somatisation disorder is discussed further in section 2.55 below.

(II) There was independent evidence of personality disorder.

Table 2.20 confirms that symptomatic prognosis was significantly worse among the patients given a psychiatric diagnosis of personality disorder. This was usually of unspecified type, references to "hysterical" or "histrionic" personality being rare in the original assessments. This bears out previous observations of the weakness of the latter's association with hysterical conversion symptoms (Kretschmer, 1926; Merskey & Buhrich, 1975). Given the comments on their abnormal personality, all three cases of "Briquet's syndrome" were treated as instances of personality disorder for the purposes of statistical analysis, but a strong association with poor prognosis for personality disorder remains if these cases are excluded. While the association observed here is consistent with Ljungberg's (1957) observations on the consequences of "abnormal personality" (see section 2.1 above), its utility is compromised by very similar considerations, viz. the probable lack of agreement.
between these observers and others as to the presence of "personality disorder" in the absence of standardised criteria for the diagnosis. The failure of the questionnaire measures of personality to confirm this tendency might be taken as further grounds for caution.

The finding may have important positive implications too. Clinical diagnoses of personality disorder are usually based upon evidence of repeated behaviour patterns in a patient's history. As other findings in this study (cf. section 2.54) also link poor prognosis to previous patterns of behaviour, further study seems desirable of specific pre-morbid behaviours that have prognostic significance.

(iii) There was little evidence of affective distress at presentation.

The greater likelihood for diagnoses of depression or anxiety disorders to have a good outcome here is compatible with the hypothesis, although the questionnaire measures of mood contradict one another. The specific association of diagnoses of anxiety disorder with a particularly good prognosis (table 2.22) are impressive. That this is more likely to represent an acute psychiatric episode is apparent as only 30% of the patients with anxiety, but 55% of those with depression, had a previous psychiatric history. As with personality disorder, it might be presumed that prognosis of the presenting symptom reflects the typical prognosis of the psychiatric condition. The lack of a sequential follow-up has precluded detailed examination of how far remission of affective symptoms has corresponded to improvement in the index symptom, which would be expected according to Roy (1980).

2.53 Hypotheses concerning diagnostic prognosis

The incidence of a change in diagnosis for the original symptom, 11 cases out of 73 (15%) was lower than previous studies of patients in medical settings would predict. Apart from Slater and Glithero's figure of 56% (of whom nearly half had provisional neurological diagnoses, usually epilepsy, for the hysterical symptom on assessment), Watson and Buranen (1979) quoted a 25% 'false positive' rate when they reviewed diagnoses. The criticisms already made of the latter's initial measures and their assessment of outcome identified likely sources of bias towards an excessively high figure (section 2.51(i)). However, the lower figure here is very likely to reflect improvements in diagnostic standards by the time of these patients' baseline assessments which were performed between 1978 and 1980.

Table 2.47 summarises the implications of the results in section 2.43 for the hypotheses outlined in section 2.22. Individual hypotheses are discussed below:
2: A 10 year follow-up study

**Hypothesis:**

Subsequent diagnosis of a neurological disease accounting for the presenting symptom was more likely when ..... 

(i) There was evidence of impaired neurological function, in the form of:
   - I) An independent neurological illness present at presentation 0
   - II) A previous history of neurological illness; 0
   - III) A provisional neurological diagnosis being made on the index admission. ++
   - IV) The presenting symptom was more susceptible to misdiagnosis, i.e. seizures or tremor. 0
   - V) Signs were present on neurological examination ++

(ii) The clinical presentation was untypical of patients with conversion disorder, in particular:
   - I) Onset of the symptom was late in life; 0
   - II) No history elicited of past unusual illness behaviour; 0
   - III) The patient was male; 0
   - IV) Unilateral symptoms did not favour the left side; 0
   - V) Initial IBQ scores did not follow Pilowsky's 'conversion' pattern 0

(iii) Psychiatric and psychological findings were not consistent with a diagnosis of a neurotic disorder, with:
   - I) No previous history of psychiatric treatment; –
   - II) A lower incidence of diagnosed psychopathology; 0
   - III) Lower scores on questionnaire rating of affective symptoms. 0

**Table 2.47: Summary of hypothesis testing - diagnostic outcome.**

- **Strong support** = ++
- **Weak but significant support** = +
- **No support** = 0

*Significant association in opposite direction* = –
Subsequent rediagnosis of neuropathology accounting for the original symptom would be more likely when the following possible concomitants of covert neuropathology had been present at presentation:

I) Independent neurological pathology on presentation;
II) Previous history of neurological illness;
III) A tentative neurological diagnosis being made on the index admission;
IV) The presenting symptom being more susceptible to misdiagnosis, i.e. seizures or tremor.
V) Signs were recorded on neurological examination.

Table 2.26 shows that at presentation an additional neurological disorder was no more likely to have been diagnosed among patients whose diagnoses changed than the remainder. Rediagnosed patients were also no more likely to have had a past history of neurological illness. However, despite the small number concerned, trends for patients whose diagnoses were revised to have been given a provisional neurological diagnosis, and for a greater number of neurological signs to have been recorded, proved significant (table 2.26).

The lack of association with concurrent neurological illness could be argued to be a false negative given the small number of cases this applied to (11), and the fact that this remained one of only three factors identified in the admittedly imperfect logistic regression analysis (cf. section 2.51(iv)). A lack of support for this and the association expected with past neurological illness would not be surprising given that Whitlock (1967) and Merskey & Buhrich (1975) document non-specific neurological changes among a majority of patients with hysteria. It would indicate that hysterical symptoms and neurological impairment can be indirectly related without the latter having to represent some kind of prodromal manifestation. However, other interpretations are also possible given the character of the previous illnesses recorded in this sample (table 2.6). Not only is there a preponderence of diagnoses frequently confused with conversion disorder or pain disorder (epilepsy, myasthenia, migraine), but the presenting symptom resembled a recognised symptoms of the previously diagnosed disorder in just over half of these cases. For some of these patients, subsequent diagnosis of a neurological disorder to account for the presenting symptom could have required an earlier diagnosis to be remade. That this was rare may be evidence that some of these previous diagnoses were unsound in the first place and would not necessarily stand up to retrospective scrutiny.

However, the study indicates the situation is different once a provisional neurological diagnosis has been entertained for the index symptom following the detailed assessments to which they were all subject before entering the study. A link between provisional neurological diagnoses and later rediagnosis was noted by Slater & Glithero (1965), having applied to nearly half of their rediagnosed cases, albeit three times more frequently than in the present study. Table 2.23 shows a further point of difference between the 6 patients whose
provisional diagnoses were substantiated here and the 19 in Slater's: unlike the majority of Slater's subgroup, only 2 of these 6 had diagnoses changed because suspected epilepsy was subsequently confirmed.

The association of rediagnosis here with a greater number of neurological signs on presentation is a unique finding. The count includes those that did and did not conform to patterns thought typical of neurological disorder. The implication that such a distinction is irrelevant to predicting future prognosis is consistent with reports that have cast doubt on absolute distinctions between hysterical and non-hysterical signs. Thus Merskey (1988) has taken Walters (1961, 1969) to task for identifying an atypical, 'regional' pattern of presentation with absence of neurological pathology when further research suggested that 'regional' patterns of changed function could themselves have a neurophysiological basis. The apparent incompatibility of hysterical and non-hysterical signs in the same patient has also been challenged by Gould et al (1986) who showed that atypical signs commonly accompanied those attributable to neurological disease, even if they are not always reported. The association here between greater numbers of signs of whatever type and a poorer prognosis is consistent with a continuum between 'organic' and 'functional' signs, with all signs being indicative of overt or covert neurological disorder. Indeed, analysis confirmed that subjects who had a provisional neurological diagnosis for their symptom were likely to have greater numbers of physical signs on examination, indicating that these associations were linked (table 2.27). Perhaps the presence of physical signs made the assessing physicians reluctant to discount a neurological pathogenesis completely, rendering patients more likely to enter the subgroup having provisional diagnoses for their symptoms.

(ii) Subsequent rediagnosis of neuropathology accounting for the original symptom would be more likely if the presentation was untypical of patients with uncomplicated conversion disorder, in particular:

I) Onset of the symptom was late in life:

II) There was no prior history of unusual illness behaviour;

III) The patient was male;

IV) There was no tendency for unilateral symptoms to favour the left side.

V) Initial scores on the IBQ subscales did not follow a pattern Pilowsky found in a 'discriminant function' for conversion patients.

The group of patients whose diagnoses were revised tended to differ from those whose diagnoses went unchanged on all 5 of these parameters, although not always to a significant extent. It was remarkable that patients whose diagnoses were unchanged were typical of hysterical populations in having a 3:1 female:male ratio and 65% of lateralised symptoms on the left, whereas there was less bias towards a particular gender or to lateralisation of symptoms, making this subgroup suspect as hysterics (table 2.2g). (cf. Stern,
While there may be no typical age of onset of hysteria, late onset has been linked with greater likelihood of misdiagnosis (eg Marsden, 1986) and the non-significant association here between later onset and rediagnosis is consistent with this (table 2.28). Although the overall incidence of past episodes of abnormal illness behaviour may be low in this sample (table 2.28) it was more likely to have been discovered in the histories of patients who kept their diagnoses than those who did not.

The final component of this hypothesised profile of the 'true hysteric' does not relate to routine clinical assessment but the analysis of responses to the Illness Behaviour Questionnaire. Higher scores on the discriminant function that Pilowsky derived from comparisons of pain clinic attenders with a general practice sample (Pilowsky & Spence, 1983) have been said by him to distinguish patients with conversion disorders. Although it failed to reach statistical significance, the difference is interesting (table 2.29). The discriminant function has been used in a study of newly assessed patients assorted according to clinical impressions of the "organicity" or "functionality" of their symptoms (Creed et al, 1990). Again, there was a clear trend in that study by which median scores on the discriminant function rose in line with attribution of "functionality" among a group of patients whose complaints were similar to those in the present study. Their raw scores would indicate that it is the patients whose diagnoses changed in the present study who have been typical of patients with neurological disease, as the median score of 60 among the remainder would appear to be typical of a group of "mixed" diagnosis. The specificity of Pilowsky's instrument here is easier to appreciate in comparison with a forerunner - the criterion keyed scales for 'hypochondriasis' and 'hysteria' on the MMPI. The latter was administered to patients in the diagnostic study of Watson and Buranen (1979), but they failed to observe any differences in scores between their false and true hysterics in their small sample.

(iii) **Subsequent rediagnosis of neuropathology accounting for the original symptom would be more likely when there had been little evidence of psychopathology at presentation, with:**

I) *No previous history of psychiatric treatment;*

II) *A lower incidence of diagnosed psychopathology;*

III) *Lower scores on questionnaire rating of affective symptoms.*

Comparison of the two groups at follow-up failed to substantiate any part of the hypothesis (table 2.28). Indeed, the findings are remarkable for a paradoxical discovery, that 9 of the 11 patients whose diagnoses had subsequently changed had had a previous history of psychiatric treatment rather than the reverse. One possible explanation of this could be that patients' previous psychiatric histories were known to the assessing team. If this knowledge led to a presumption in favour of psychological rather than physical attributions in doubtful cases, then evidence that might in other circumstances have led to a definitive neurological
diagnosis could have been less likely to be accepted or to have been sought. (This theme is discussed in relation to individual cases in section 2.55).

2.54 Hypotheses concerning subsequent consultation behaviour.

The study of consultation behaviour over the follow-up period poses some special problems of method. Several indices including hospital admission, clinic attendance, other NHS contacts and use of alternative practitioners were available, whose inter-relationships have been charted in table 2.29. From these it is evident that a progressively smaller proportion of the sample accounted for variations in in-patient and non-clinic visits, and so visits to general practitioners and out-patient clinics have been used as the basis of hypothesis testing across the sample. A tendency to high attendance at one of these did not automatically lead to high use of the other (the Spearman correlation coefficient for the 56 cases for whom data is available was 0.49 (p=0.000)) and in individual cases patients could be extremely frequent attenders of out-patient departments while rarely seeing their general practitioner and vice-versa. Discussion here of contributory factors, therefore, will consider the two indices independently.

The limited nature of the initial data constrains discussion of predictors of subsequent clinic use, as several potentially significant kinds of data were not available. These include each patient's past use of primary and secondary care, as well as measures such as the General Health Questionnaire which have a documented relationship to consultation practice (Goldberg & Huxley, 1980). The hypotheses that could be investigated were relatively specific. The balance of evidence is summarised in table 2.48 with respect to GP and outpatient consultation rates. Individual hypotheses are considered below.

(i) Future consultation rates were expected to be higher when the initial symptom had persisted over the follow-up period, with consultation rates being highest in cases where the initial diagnosis was changed.

Where symptoms persisted, use of GPs and some NHS out-patient and in-patient facilities was significantly greater (table 2.34). The greatest difference is found among those whose symptoms were reported as 'unchanged' rather than 'worse'. As the 'unchanged' group was not distinguished by other factors likely to be associated with greater demand (table 2.35) it would appear that very severely affected patients may retreat from medical contact.
Hypothesis: Higher consultations were expected when ...

Findings:

<table>
<thead>
<tr>
<th></th>
<th>GPvisits</th>
<th>OPvisits</th>
</tr>
</thead>
<tbody>
<tr>
<td>(i) The initial symptom had persisted over the follow-up period:</td>
<td>++</td>
<td>0</td>
</tr>
<tr>
<td>highest rates in cases where the initial diagnosis changed:</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>(ii) After initial diagnosis of an additional psychiatric disorder:</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>neurological disorder:</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>or provisional neurological diagnosis for the presenting symptom:</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>(iii) After evidence of abnormal illness behaviour at assessment,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>through a history of unexplained illness:</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>or evidence of disease conviction on the IBQ:</td>
<td>(−)</td>
<td>0</td>
</tr>
<tr>
<td>(iv) With unfavourable follow-up self-ratings for health:</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>or disability:</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>or high scores on the screening test for SD:</td>
<td>0</td>
<td>+</td>
</tr>
</tbody>
</table>

Table 2.48: Summary of hypothesis testing - consultation behaviour.

<table>
<thead>
<tr>
<th>Strong support</th>
<th>++</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weak but significant support</td>
<td>+</td>
</tr>
<tr>
<td>No support</td>
<td>0</td>
</tr>
</tbody>
</table>

Weak association in opposite direction = (−)
Table 2.33 shows that outpatient attendance is significantly greater when a diagnostic change has been necessary. This trend extends to other facilities, these patients spending more time in hospital, and receiving a greater proportion of non-medical services through the National Health Service. It would appear that only general practitioner consultations are relatively immune from this trend, but GP consultation rates were in turn the most sensitive to persistence of the index symptom.

The linking of diagnostic change and out-patient attendances is likely to reflect the fact that diagnostic change is likely to follow re-investigation of the original complaint at a hospital, and possibly continuing contact with that hospital for treatment of an emerging condition. At the same time, continuing outpatient contact may in itself predispose to diagnostic change if it guarantees exposure to a relatively greater number of diagnosticians, and with this the chance of a reappraisal and reinvestigation that is not necessarily sought by the patient and which non-attenders would not receive. However, among the patients providing detailed information of their consultations by interview at follow-up, 16 reported their symptoms as being the same or worse, but had not received a new diagnosis for them. These patients had visited significantly more out-patient departments than the remainder of the sample, although reporting no more visits in total, suggesting out-patient assessment per se did not make rediagnosis more likely.

(ii) Future consultation rates were expected to be higher when initial assessment led to diagnosis of an additional psychiatric or neurological disorder, or a provisional neurological diagnosis for the presenting symptom.

Table 2.35 indicates that only one kind of initial diagnostic observation is associated with subsequent consultation rates: whether or not a provisional diagnosis for the original symptom was entertained. Although both general practitioner and out-patient consultations were subsequently more frequent among those where a provisional diagnosis was made, the trend was not significant.

The finding that clear-cut psychiatric and neurological illnesses did not in themselves increase consultation rates is important. It highlights the relative impact on consultation rates of persistence of the index symptom throughout the follow-up period. It suggests that if a reduction in consultation rates is adopted as a goal, this might be achieved by devising interventions for patients whose index symptoms persist irrespective of other medical needs.

(iii) Future consultation rates were expected to be higher when there had been evidence of abnormal illness behaviour at assessment, either through a history of previous episodes of unexplained illness, or an unjustified degree of conviction about the patient's ill health had been recorded on the Illness Behaviour Questionnaire.

As table 2.38 shows, a link between past episodes of illness behaviour and subsequent consultation rates appears to hold for general practitioner consultations, but not
for outpatient attendances. Section 2.54 (i) has already indicated how these each reflect different factors, and why GP attendance rates may be more elastic. Episodes of past abnormal illness behaviour, being occasions in which a patient successfully elicited medical attention but no apparent diagnosis, can be expected to reinforce a tendency to repeat the experience from the patient's side, and possibly for further referral not to be initiated from their doctors.

The measures of illness behaviour yielded the only questionnaire measure that correlates with consultation behaviour, disease affirmation on the Illness Behaviour Questionnaire. Although it approaches significance at the 0.05 level, this is likely to be meaningless, as it forms one of 18 linked analyses performed at the same time (table 2.37). Furthermore, the association is in the opposite direction to that expected, with greater disease affirmation seeming to accompany lower consultation rates. The situation in which a crude clinical indicator (past history of unusual illness behaviour) proves superior to a questionnaire measure in its association with prognosis is reminiscent of the difference between clinical and questionnaire assessments of mood and personality on symptomatic outcome (cf. section 2.52(ii) above). Its association here with future consultation rates, apparently independently of diagnosis, certainly suggests it is a dimension that should not be ignored in future clinical studies of illness behaviour.

(iv) Future consultation rates were expected to be higher among patients who rated their health and disability least favourably on follow-up, or who appeared to be candidates for somatisation disorder on a short screening questionnaire given at follow-up.

A number of measures were used to assess patients' general health and functioning at the follow-up interview. As table 2.42 reports, global ratings of health and disability both varied in the expected direction with attendance rates in primary care and outpatient clinics, although only significantly so for disability ratings which showed little discrimination between GP and outpatient attendance. It is interesting that scores on the 7-item screening test for somatisation disorder also tended to show stronger correlations with outpatient attendance, as the North American cohort on whom it was tested were heavy users of specialist as opposed to primary care services (cf. section 2.33(ii)).

The results for the Illness Behaviour Questionnaire that 52 of the subjects completed are strikingly neutral by comparison. Even scores on the disease conviction subscale failed to correlate with attendance of either sort in contrast to the expectation of the research of Pilowsky, Smith & Katsikitis (1987). Their findings for prognosis over one year among men attending primary care do not appear to apply to this predominantly female, tertiary sample.

By contrast, when a cross-check was performed between attendance and scores at follow-up on the Nottingham Health Profile, a widely used measure of distress and disability whose 6 subscales have been correlated with likelihood of consultation in primary care (cf. section 2.33(ii)), none of 4 symptomatic subscales correlated with GP and outpatient
consultations at a significance level better than 0.01, but subscales for social isolation and loneliness both did (table 2.44). A further 7 point scale that corresponds to disability over several areas of functioning corresponded only with outpatient attendance. The greater correlation here with outpatient attendances compared to other indices of consultation behaviour was shared with other variables at follow-up, indicating perhaps that clinics were more uniform in their responses across the sample than general practitioners. The fact that good correlations can be obtained with simple questionnaires such as part II of the NHP suggests that the lack of correlation between attendance and IBQ scores reflects weaknesses in the IBQ rather than a general inability of self-report questionnaires to detect and quantify factors relevant to consultation behaviour.

2.55 Clinical implications of the study.

The study has several implications for future research and practice, beyond the prognostic factors identified through the analyses described in sections 2.52 to 2.54. The study's finding that only 15% of the cases followed up developed neuropathology to account for the original symptom contradicts the much higher proportion of Slater & Gilthero's (1965) influential study, and challenges the view that chronic pseudoneurological symptoms can and should always be subsumed under other diagnoses. However, there is no room for complacency in the cases where other pathology does supervene. The small numbers involved here does compromise the usefulness of statistical analyses for common factors, as does the division, noted by Slater, between those patients in whom the eventual diagnosis was strongly suspected initially, and those where it was relatively unexpected. The diagnostic changes in these two sub-groups, here comprising the patients in table 2.23, raise slightly different concerns for clinicians. For the six patients where a provisional neurological diagnosis was made and subsequently confirmed, why was psychogenesis suspected so strongly and neurological diagnosis withheld? In the case of the remainder, where unsuspected pathology supervened to account for the presenting symptom, why was the possibility of neurological disease dismissed at the outset?

The six patients having provisional diagnoses confirmed were very mixed in their presentations and histories. Case no.7 (table 2.23) had a long and continuous history of multiple symptoms including psychiatric symptoms dating from traumatic experiences as a prisoner-of-war of the Japanese. There was a strong presumption of psychogenesis in the absence of definitive neurological findings. Case 34, whose seizures were hard to classify among the recognised forms of epilepsy, came with a past history of hysteria in the sense that this diagnosis had been strongly suspected during previous investigations rather than being raised for the first time. Cases 51 and 71 were both viewed as having a marked tendency to
exaggerate in the course of assessment, the former being seen as a manipulative and exhibitionistic man with suspected mild learning disabilities; the latter as hypochondriacal with a history of inconclusive investigation for other symptoms. Case 5’s case is instructive in this respect, as she had been extremely demanding of attention on presentation and follow-up. Discharge had been arranged following a psychiatric assessment supportive of a diagnosis of abnormal illness behaviour, before an abnormal scan result was noted. The tentative diagnostic conclusions therefore reflected some incompleteness in neurological investigation at the point of discharge and her blackouts were subsequently attributed to a vascular lesion at the site of the scan abnormality. This leaves case 41, a lady who remained irritated as she quoted her diagnosis of "conversion hysteria" at follow-up. She had had a clear diagnosis of myasthenia gravis and thymectomy 8 years before presentation, as well as a history of "neurotic" depression. While tests had been equivocal, her speech problems had suggested an hysterical aphonia, although an accompanying dysphagia only remitted once she had treatment from an endocrinologist a year later.

The cases where the final neurological diagnosis was unsuspected included two where some initial neurological diagnosis had been made. Case 48 was felt to have bizarre features to his gait that were not typical of the unspecified extrapyramidal disorder he had been diagnosed as having for many years before myotonia was diagnosed. Case 76 was an elderly and very depressed woman whose numbness had been attributed to a mixture of carpal tunnel syndrome and hysteria, whereas the later unique diagnosis of multiple sclerosis proved correct.

Among the three cases where no neurological diagnosis of any kind was made initially, an unusual gait was confidently attributed to hysteria following a temporary response to behaviour therapy although progressive spino-cerebellar degeneration was diagnosed subsequently (Case 10, table 2.23). The remaining two are particularly instructive, as reversible pseudoneurological symptoms were undoubtedly present, but a diagnosis of conversion disorder alone proved insufficient. In both, gait and speech abnormalities were present. In one, an apparently psychogenic aphonia remitted but spinal muscular atrophy supervened (case 77, table 2.23). In the other, the gait abnormality responded to simple psychological measures, but carcinoma of the larynx was diagnosed a year later (case 39, table 2.23).

Some tentative diagnostic lessons might be drawn from this experience, for instance: diagnose the illness and not the patient; do not retrospectively modify diagnoses in the light of therapeutic responses alone; above all, consider each symptom separately, before attempting to reduce the whole to a single diagnosis.

Further clinical implications concern the diagnosis of the patients, and whether it was changing in another sense. If the majority whose symptoms persisted continued to have "hysteria", was this necessarily the syndrome they started with? It was remarkable that only three (4%) of the patients were felt to have "Briquet's syndrome" on admission (table 2.5)
given earlier North American claims for a prevalence of 12% in referred general medical patients (Purtell et al, 1951) or 11% in psychiatric outpatients (Woodruff et al 1971). However, the findings from patients on follow-up using the Othmer and DeSouza/DSM-III-R screening test for somatisation disorder, in which 36 (64%) scored 2 or more, and 23 (41%) scored 3 or more suggest most patients interviewed would have qualified by then for the diagnosis. On the basis of their observations on the test's sensitivity and specificity (section 2.33(ii)), Othmer and DeSouza (1985) suggested a score of 2 or more makes the diagnosis of SD "highly probable". Although the method of the present study could be argued to predispose to high scores because a medical aetiology could not be excluded for all symptoms reported in response to the probe, it provides evidence that many cases of somatisation disorder were developing in the course of the study. The contrast with the initial diagnoses also suggests that progressively greater reporting of past symptoms with the progress of time does not necessarily reflect a change in the number of symptoms experienced, but a behavioural disposition that develops when cases become chronic and medical exposure is increased. Ewald et al (1994), in comparisons between somatisers on neurology wards, observed that the number of symptoms reported reflected the extent of their "doctor shopping" as well as the duration of their illness.

Irrespective of the precise number of patients qualifying for diagnoses of Briquet's syndrome or somatisation disorder at either end of this study, the findings strongly suggest that reporting of multiple symptoms is a relatively late development in patients' careers, at least in those presenting primarily with pseudoneurological symptoms, and this is therefore artefactual to a prior behavioural disposition to seek medical help to an abnormal degree. A point that is frequently overlooked concerning Guze's own seminal retrospective and prospective analyses of polysymptomatic hysteria (Purley & Guze, 1962) is relevant here. When 25 of 28 patients admitting to gross polysymptomatology at presentation then kept their presenting symptoms in the absence of systemic disease to account for them over a period of 3 to 10 years, they had already had their symptoms for an average of over 13 years before the study began. Thus, the patients in the present study who would have been comparable with them would be those retaining their symptoms at follow-up, rather than the entire cohort at the time of presentation.

The suggestion that reporting of multiple symptoms is a late artefact following established behavioural patterns is consistent with associations observed between somatisation disorder and recognised personality disorders (Stern et al, 1993) and a long tradition that views SD as a personality disorder in its own right (Kaminsky & Slavney, 1976). The present study, by associating subsequent chronicity with prior evidence of personality disorder and a history of unusual consultation behaviour in preference to the number of symptoms reported at an early stage (cf. section 2.52) adds to the case to base diagnosis more securely on features which are currently listed only as "associated features" in DSM-IV (APA, 1994). Weaknesses of the present study, notably the failure both to record the extent
of prior consultation behaviour, and the number of potential symptoms representing somatisation by the same systematic method at presentation and at follow-up would need to be rectified in future studies aiming to specify the sequence of these developments more precisely. Ideally, any future naturalistic study would not only include clinical assessment of specific personality traits at its onset, but would chart the progressive development of somatic focussing, symptom reporting, and consultation behaviour through repeated prospective assessments.

The persistent morbidity uncovered in this study is evidence of the need for effective treatments. As poor symptomatic outcome appears to prompt high rates of consultation behaviour, apparently without result, this adds to the case. The study appears to indicate two potential foci for intervention from the association of symptom persistence with subsequent reporting of many other symptoms and with high levels of perceived disability (table 2.42). This suggests that therapeutic measures that aim to refocus attention on non-somatic discomfort and that challenge the patient's sense of their incapacity would offer rational means not only to seek immediate relief, but by which long-term handicap in this population may be prevented.
2.6 Conclusions

This study has confirmed that after patients receive assessment for symptoms at a neurological hospital, and these are not attributed to neurological disease, the prognosis remains poor for a significant minority. Symptom persistence is more likely when symptoms have been present for a considerable period prior to assessment, when patients are older, and when there is independent psychiatric evidence of a personality disorder rather than an affective disorder. The likelihood of diagnosis being revised in favour of that of an underlying neurological disorder within 10 years is less than has often been believed, but remains a significant possibility. This is more likely to occur when tentative diagnoses were entertained and when signs were recorded on physical examination (irrespective of their apparent diagnostic significance). There may be a greater danger of initial misdiagnosis if a patient's history of past psychiatric treatment is known to her clinical assessors.

Subsequent consultation behaviour can be expected to be more frequent if the patient had a past history of episodes of unexplained illness, or a provisional neurological diagnosis was considered during the index admission. Indeed factors promoting both diagnostic change and symptom persistence should be expected to favour higher consultation rates subsequently.

There have been a number of significant limitations to the study described here, including a lack of standardisation in the initial clinical assessments, omission of potentially useful data in those assessments, and lack of a consistent and independent medical re-examination of all patients at follow-up.

It would appear that future research should pay more attention to past patterns of consultation in predicting prognosis, and attempt to devise more discriminatory questionnaires than the Illness Behaviour Questionnaire to identify patients who do not qualify for diagnoses of personality disorder but whose symptoms can be expected to persist without neurological disease supervening. Nearly all the patients in the study received no specific therapy for their symptoms following their assessment. The extent to which their symptoms may be amenable to these remains open. As the study confirms the poor prognosis of the condition, this seems worthy of further study.
Chapter 3: Experimental studies of attention in patients with pseudoneurological symptoms.

Aims:

- To evaluate hypotheses that patients with hysterical symptoms share subtle defects of attention.
- To develop methods of assessment that realise the benefits of microcomputers over conventional methods of testing divided attention.

3.1 Background to the study.

3.11 Janet and "retraction of the field of consciousness".

This chapter describes two contrasting studies that aimed to identify replicable psychological deficits among patients with hysterical symptoms. They belong to a tradition launched by Pierre Janet who, as professor of psychology at Charcot's Salpêtrière, had described characteristic changes in higher mental functions among hysterical patients. The precise pathogenesis of these were obscure, but their effects were pervasive. Janet, like Hughlings Jackson (and their common mentor, Ribot) had subscribed to a hierarchical model of mental operations that held lower "automatic" functions to be subordinate to higher "synthetic" ones. Higher level failure inevitably entailed loss of integration and co-ordination between lower level operations, leading to the symptoms and signs from which higher level failure was inferred. Hysteria was especially worth of study for Janet because it represented a very pure case of impairment of this integrative capacity. His exhaustive early studies of hysterics' pathology (Janet, 1894, 1897) are summarised in this definition offered in his 1907 Harvard lectures:

"Hysteria is a form of mental depression characterized by the retraction of the field of personal consciousness and a tendency to the dissociation and emancipation of the systems of ideas and functions that constitute personality."

Janet's concept of a "retraction of the field of personal consciousness" peculiar to hysterical patients was more than an explanatory generalisation. He made very specific statements about performance that were consistent with this analysis. His descriptions confirm that he was studying phenomena modern psychologists would attribute to variations in attention, although his clinical observations have excited remarkably little attention from clinicians or researchers since. In "the mental state of hystericals" (Janet, 1907) he describes how a "retraction of the field of consciousness" leads to suggestibility, absentmindedness, and what he termed "alternation" in which the attention was forced to shift more rapidly between objects which could no longer be entertained in consciousness alongside one another. Janet held that this was an easily demonstrable finding, explaining how:
"They can perform but one action at a time. You ask one of these patients to make a certain movement continually, for instance to make on the table with her right hand the movement of playing on the piano. It is agreed that she must not discontinue this little movement, whatever may happen. At the same time, you ask her to perform some other simple acts, to open her mouth, to shut her mouth, to recite numbers. You always remark that the first movement, the piano playing, stops as soon as the second begins, and that it only recomences at the end of this second movement. Yet the subject had made up her mind to continue this movement, she had this idea in her head, but it became impossible for her as soon as she tried to do something else." (Janet, 1907 p.296).

The "retraction" of which Janet writes represented a restriction on the number of operations of any sort that could be consciously attended to at one time in this passage. It could also affect the sensitivity of an individual sensory field. Thus:

"Normal consciousness, as philosophers say, is always a fully illuminated point, surrounded by a strong penumbra. With the hysterical, the penumbra is wanting. This fact is brought into evidence by their quite peculiar visual field; you do not find in any normal individual that odd vision, which sees very clearly in one point and sees nothing around this point." (Janet, 1907 p.298).

Janet's observations suggest at least two specific hypotheses about attentional processes in patients with hysterical symptoms that stand independent of his own conceptual framework. These are:

1. Patients with hysterical symptoms are impaired in their ability to simultaneously perform repetitive motor and verbal tasks.

2. Patients with hysterical symptoms are less able to divide attention between objects simultaneously presented within their field of vision.

In developing appropriate clinical experiments to test these assertions, three sets of more recent work are particularly relevant. The first represents previous attempts to develop a neo-Janetian hypothesis accounting for symptom formation and psychological features among patients with conversion hysteria. The concept of "corticofugal inhibition" has been introduced as a unifying idea in physiological and psychological studies of these patients (Ludwig et al, 1972). However its limitations (cf. section 3.12 below) confirm the need for an alternative strategy of investigation such as the one to be proposed here.

The second set of work has sought a rationale for differences in performance on dual attention tasks through a concept of "functional cerebral space" compatible with more recent theory in neuropsychology (cf. section 3.13 below). This has informed a tradition of experimental work into the impact performance of one test can have on another. It has been used almost entirely in studies of non-clinical populations, but will be argued to have special relevance to patients with hysterical symptoms.

A third set of work concerns attempts to measure the number of objects that can be simultaneously registered within the visual field, the so-called "apprehensive." or "perceptual
3: An investigation of attention span. This methodology has been employed in groups of subjects with diagnosed psychopathology in the past, although not people with hysteria.

They will be reviewed in turn in sections (3.12 to 3.14) below, and their implications for the present study summarised. The potential contribution of microcomputer technology to experimental studies is also reviewed.

3.12 Attention in hysteria: corticofugal inhibition.

Two groups of psychiatrists have acknowledged debts to Janet in their speculations about attentional problems in patients diagnosed as conversion hysteria, (Ludwig et al., 1972; Meares et al., 1985) and have used these ideas to inform their own empirical studies (Bendefeldt, Miller & Ludwig, 1976; Meares and Horvath, 1972; Horvath et al., 1980). One concept they each refer to and elaborate in different ways is that of "corticofugal inhibition". Hernández Peón (1963) coined this term to account for the results of a classic experiment in which hysterical anesthesia was associated with a measurable reduction in afferent potentials. The hypothesis of "corticofugal inhibition" attributes this to centrally generated efferent activity which then selectively inhibits transmission from peripheral afferent systems. In an influential review of hysteria that emphasised the high incidence of neurological disease among patients with hysterical symptoms, Whitlock (1967) cited corticofugal inhibition as a possible neurophysiological mechanism they shared. The concept informed a number of isolated clinical studies of evoked potentials in patients with hysterical anesthesia (Levy & Mushin, 1973), although the extent to which these actively supported the hypothesis was equivocal. (These patients' evoked responses being reduced only at low levels of stimulation).

The interest and experiments of the two neo-Janetian groups took different directions from this point. Ludwig (Ludwig et al., 1972), in formulating his "neurobiological" theory of hysteria, proposed a neuropsychological study of the correlates of "corticofugal inhibition". He predicted cognitive impairments of attention and, secondarily, memory. In order to derive testable assumptions from his model, Ludwig classified the predicted attentional deficits according to a scheme of Silverman (1968). Hysterical patients were expected to have an increased sensory threshold, reduced scanning with low saccadic eye movement and field dependence with a tendency toward hyperdistractibility. With respect to memory, Ludwig predicted not only that deficits will be co-terminous with illness, greater with stress and for tasks requiring significant degrees of attention, but that they would affect recall rather than registration, and be more marked for emotion-laden material. His team's results, based on clinical testing of 17 patients with conversion hysteria and comparisons with neurotic controls, indicated significant differences in performance on tests of "suggestibility" (by inducing arm levitation) and "field-dependency" (on a hidden figures test), as well as
impaired "recent memory" (a guessing technique and paired associates test); "attention" (mental set shifting) and "vigilance" (a continuous performance test) (Bendefeldt, Miller & Ludwig 1976).

However, although Ludwig felt he was putting the "astute clinical observations of Janet" to direct scientific test in this way, the implications of his findings for the hypothesis of "corticofugal inhibition", or indeed Janet's "retraction of the field of consciousness", were not clear. "Hyperdistractibility" still has to be inferred as none of Ludwig's tests assessed it directly, while individual findings from the tests of memory and attention he used are quite non-specific. They could neither fully test Ludwig's predictions because of their limitations (for instance, although his memory tests had some selectivity for recall, they were unable to discriminate for emotional content), nor provide support for a corticofugal mechanism as opposed to any other cause of attentional impairment. The argument that the isolated deficits, and the emergence of heightened suggestibility, are adequate evidence of a failure of integrative processes may be Janetian, but it is weak in so far as almost any set of positive findings could be pressed to serve it. Ludwig's hypotheses about attention appear to have been insufficiently specific. Instead of using a schema developed for other purposes, it would have been preferable to attempt to demonstrate "retraction of the field of personal consciousness" by more specific indices.

Whereas Ludwig's group eschewed physiological measurement, Meares and his colleagues continued to employ it in extending and subsequently moving beyond the paradigm of corticofugal inhibition. Their key experimental finding concerned a failure of GSR responses to habituate to auditory stimuli among 11 patients having a clinical history of "chronic" hysteria (Meares and Horvath 1972; Horvath et al, 1980). This failure persisted even in the absence of concurrent conversion symptoms, or evidence of increased autonomic arousal. These patients contrasted with 6 others having acute conversion symptoms, who lacked their history of pre-morbid somatisation, and showed normal habituation. (They were therefore typical of other neurotic groups in this, as Lader and Sartorius (1968) describe).

To account for this finding, Meares followed Ludwig in speculating about corticofugal mechanisms, also citing Janet and Hernández-Peón. However, when Meares reconsidered his work in the light of Janet's ideas in a further review (Meares et al, 1985), he tended to play down Janet's own ideas on psychological mechanism. Meares thought his finding of impaired habituation was consistent with Janet's inferences about a failure of synthetic processes, but viewed the implications for attention differently. Whereas Janet had attributed overt distractibility ("alternation" in his own terminology) to an excessively focal and excluding restriction of attention, Meares postulated an impairment in what he termed "screening capacity". When the normal selective inattention to meaningless stimuli was compromised in this way, a "sensory overload" resulted.

Meares applied this idea in an apparently consistent way in further speculations on the pathogenesis of the chronic polysymptomatic syndrome of somatisation disorder (cf.
3: An investigation of attention

section 1.52). He postulated that patients with somatisation disorder share a common "subtle neurophysiological deficit" which accounts for their predisposition to somatising behaviour. A failure in these patients' screening capacity means their awareness of somatic sensations fails to habituate, prompting a conscious preoccupation with them. Subsequent studies by this group looked firstly at centrally recorded event related potentials in patients meeting criteria for somatisation disorder (Gordon et al, 1986). They were unable to replicate the findings found in the study of chronic conversion hysteria in this group (Meares & Horvath, 1972), the early N1 component alone showing evidence of exaggeration. This was taken to imply an impairment of initial filtering processes rather than subsequent responses. As their hypotheses were drawn from theories and studies concerning patients with conversion hysteria, these authors draw the reasonable inference that they may have obtained more convincing results from patients having pseudoneurological symptoms. Nevertheless, they have continued to examine patients meeting diagnostic criteria for somatisation disorder rather than (chronic) conversion disorder. Although they report a positive result from a still more sophisticated ERP paradigm, using mismatch negativity (James et al, 1989), there has been no comparative data from other groups apart from their own controls to evaluate this.

As the only previous attempt to base experimental investigation of hysteria on Janetian hypotheses, Meares' work has a number of significant implications for the present investigation. One of these is the lack of direct connection between the hypothesised failing, corticofugal inhibition, and the parameters that were measured. It is unclear in investigations relying on psychometric tests or ERPs how far positive findings would actually justify interpretation in terms of this mechanism. This suggests that any new "Janetian" studies would benefit from more clearly articulated hypotheses.

At the same time, it appears that the decision to select patients having somatisation disorder rather than chronic conversion symptoms for their experimental group, reflecting the clearer diagnostic criteria for that diagnosis, has been unproductive. The impressive results obtained with patients having chronic conversion symptoms alone suggest that future studies are justified in restricting investigations to patients with pseudoneurological symptoms.
3.13 The division of attention in hysteria.

The development of the theoretical concept of "functional cerebral space" illustrates a close integration between theory and experimental studies. In neuropsychological investigations of hysterical disorders, it offers a model which is specific, testable and amenable to elaboration.

3.13(i) The concept of "functional cerebral space".

Investigation of the limits of human attention has relied significantly on experiments that present competing demands between which attention is apparently "shared" or "divided". The way in which they and other experiments are interpreted can vary considerably according to concepts of what attention is, and what sort of constraints are involved. Since the 1950s, interpretations of data from studies of divided attention have referred to the "capacity" of an information processing system having several "channels". Its resources are presumed to be limited, due primarily to the restricted speed of the serial processing thought to correspond to conscious, attended operations. However, this has not necessarily been taken to imply that there are fixed limits to the total processing capacity available for the operations that require attention in that way. Both Kahneman (1973) and Pribram (Pribram and McGuinness, 1975) invoked a faculty of "effort" that was capable of expanding attended processing capacity to allow for variations in the demands that an individual's conscious attention could tolerate. However, this left it unclear how "effort" would itself translate into the terminology of information processing, and assumed that all types of processing demand were equivalent to one another in kind.

Kinsbourne was responsible for an alternative way of accounting for variations in attentional capacity that aimed to be more internally consistent (dispensing with injections of "effort" from without as a way of extending a limited system). His account sought compatibility with contemporary neuropsychology in hypothesising how the availability of attention for a given task would be affected by the nature and localisation of the brain processes involved, and was especially relevant when predicting the response to competing demands for attention. He suggested that the processing capacity available would depend upon the siting of those cerebral processes, with a relatively greater reduction in available capacity occurring if two simultaneously attended operations activated regions so proximate that processing in one impeded concurrent processing in the other. There was a presumption that this proximity would reflect patterns of cerebral localisation of different processes, although the map of "functional cerebral space" did not have to mirror that of anatomic localisation (Kinsbourne and Hicks, 1978). Nevertheless, a functional map of this kind would provide a blueprint from which specific hypotheses about the likely nature and size of interference between different tasks could be deduced.
3: An investigation of attention

3.13(ii) Use of dual task methodology in the dynamic testing of attention.

The development of Kinsbourne's model of a functionally differentiated cerebral space depended heavily on experimental evidence from interference effects between simultaneous tasks in general, and vocal-manual or verbal-manual interference in particular. He used two related experimental demonstrations of verbal-manual interference, each designed to highlight a differential impairment of right hand performance compared to left hand performance during verbal recitation.

The first of Kinsbourne's tests examined the length of time a dowel rod could be balanced, after considerable initial practice, by either hand alone and then while subjects continuously repeated a standard sentence aloud. (Kinsbourne and Cook, 1971). As might be predicted from his model of interference occurring within shared functional cerebral space, right hand balancing, consistently superior to left hand balancing alone, was significantly impaired during the verbal task. However, Kinsbourne and Cook also found that left hand balancing was unexpectedly improved during recitation, and to an even greater extent. They attributed this facilitation to an effective withdrawal of attention from the left hand during verbalisation. They hypothesised that withdrawal of attention could improve performance generally on tasks that had been sufficiently practised. The inference is a problematic one, contradicting many experimentalists' assumption that the quality of task performance reflects the attention actively devoted to it.

Such left-handed facilitation does not appear to have been replicated (Hicks, 1975) while right sided impairment remains a robust finding on this test. In any case, this particular method poses additional problems of interpretation because rod balancing, which requires visual monitoring at the same time, is a far from pure "manual" task.

The alternative methodology Kinsbourne used to assess the mutual impact of simultaneous task performance took the maximum number of finger taps that could be executed during a fixed 30 second time period as its manual task (Kinsbourne & McMurray, 1975). A convincing decrement in right hand performance when accompanied by rhyme recitations was found in normal children, an effect which apparently diminishes with age. Briggs (1973) reported that significantly more complex verbal tasks are required to produce interference effects in normal adults, which remain lateralised with significant impairment of right handed performance only. Subsequent work has tended to confirm this, with Hellige and Langdon (1981) showing a reciprocal effect by which a right hemisphere task, block design, selectively impaired tapping performance of the left hand.

Although their work was largely confined to normal subjects, Kinsbourne's team employed related tests on one possible neurological model for hysterical deficits: the commissurotomised patient. Thus, Kreuter et al (1972) reported that right handed performance on tapping tests during vocalisation was impaired in commissurotomised subjects at levels of task demand that had no effect on the performance of normal subjects. It was
concluded that callosal section led to increased interference once it was no longer possible for contralateral cortex to assist in the control of a function compromised through local competition from nearby homolateral sites. Kreuter's experiment had invited simultaneous tapping by either hand at as fast a rate as possible. It appeared that lateralised impairments in other situations were potentiated when both hands are required to perform together rather than in turn (Hicks et al 1974).

Variation in performance on these overtly simple experiments can also reflect the nature of the secondary task. Lomas and Kimura (1976) examined both dowel balancing and finger tapping (as well as arm tapping) while subjects were silent, recited aloud, or hummed ("non-verbal vocalisation"). They found that impairment of dowel balancing was lateralised during verbalisation only in males: males also showed bilateral impairments with humming. However, their protocol would indicate that the balancing had not been nearly as well practised before testing commenced in comparison with Kinsbourne and Cook's procedure. When Lomas and Kimura reviewed their results, simultaneous verbal recitation was associated with bilateral impairments on single finger tapping for subjects of either sex. The only tests that showed lateralised impairment specific to concurrent verbalisation were sequential finger tapping and sequential arm tapping. A similar procedure was used by Ikeda (1987) who was able to confirm the additional sensitivity conferred by a sequential as opposed to a simple tapping test. Ikeda also demonstrated a greater degree of interference between a vocalised verbal task as opposed to a silent one on right handed sequential tapping.

Sequential tapping tasks would therefore seem particularly promising for the kind of experimental investigation required here. Apart from this evidence of its potential sensitivity, it has three prime methodological virtues. One is that, like other tapping tests, it represents a pure form of manual task, without demanding other kinds of attended performance at the same time. Secondly, the sequential nature of the task makes for greater sensitivity as failure to complete sequences is likely to be evident before there is an impact on the overall tapping rate. Thirdly, the measurement of completed sequences in a pre-defined time is a highly replicable measure, whereas other protocols have been weakened by relying solely on measures of relative change in performance (eg. deviation from a baseline tapping rate).

The implementation of tests of sequential tapping progressed from the complexity of Hicks et al's (1975) procedure in which sequences of movements were copied using an adapted typewriter keyboard, to Lomas and Kimura's reliance on a row of four identical telegraph keys. Each of these was connected to a pen on a 4 pen recording drum, from which the experimenter counted the number of taps that contributed to correctly performed sequences from index to little finger. Their subjects were invited to complete as many sequences as they could in the 30 second period, while keeping their palm pressed to a board to allegedly minimise the effect of previous piano training on their performance. The same procedure was followed when subjects were required to add vocalisations at the same time. The resemblance between this sequential tapping task and the bedside test Janet
used to demonstrate retraction of consciousness in his hysterical patients (section 3.11) seems remarkable. It has not been used with patients with hysterical complaints before.

If, as the first study hypothesis suggests, there will be greater interference between performance on manual and verbal tasks interfere in subjects with pseudoneurological symptoms compared with appropriate controls, this body of work presents a sensitive and appropriate paradigm by which it can be tested. Moreover, the theory of functional cerebral space provides a way of explaining, and potentially mapping, consistent inter-group differences in such performance. The failure of an important earlier study to substantiate expected interference between homolateral manual and visual tasks (Dimond & Beaumont, 1972) is an additional reason for selecting investigation of manual/verbalisation interference for this purpose.

3.14 Perceptual (or "apprehensive") span.

The study's second hypothesis (section 3.11) requires an experimental paradigm concerning attention within the visual field. The concept of perceptual span has developed as experimental methods have grown in sophistication. It has been previously used in clinical studies of other groups, but not patients with hysterical symptoms.

3.14 (i) The history of perceptual span.

Psychologists had concerned themselves from Janet's own time with ascertaining the maximum number of objects could be attended to simultaneously. This measure has often been termed "apprehensive span" as well as "perceptual span". Its relatively long history, during which methods for its measurement have been progressively standardised, are described in the following paragraphs.

Early developments in the measurement of perceptual span have been reviewed by Woodruff (1938), from which the rest of this paragraph is abstracted. The first reported attempts to quantify apprehensive span coincide with a move from introspection towards experiment in psychology on the part of Sir William Hamilton around 1850. Hamilton would throw a set of marbles onto the floor in front of his subject, asking him to close his eyes as soon as they landed and then to report the number he could discern from the afterimage. Such methods were very crude, not least in leaving the duration of exposure to the objects in the subject's control. Subsequent modifications involved more accurate control of the size of the object field (W.S. Jevons introducing the use of a sand tray in the 1870s) then regulation of the duration of exposure to an array of objects by means of the tachistoscope as pioneered in the 1880s by Cattell. (This is an apparatus that requires subjects to rest their head in a predetermined position so that gaze is directed only towards a field of objects. A shutter between the object field and the subject's eyes has to be open for the objects to be seen, while the duration of its opening, usually very brief intervals, can be preset so it remains
Further modifications concerned the objects that were displayed. If these were all of the same sort, the ability to count the number present accurately could also reflect “chunking” effects - i.e., it would be possible to count to relatively high numbers by subdividing the objects within the field into groups, each containing the same recognisable number of objects, so long as the number of groups remained within the apprehensive span.

To counter this effect, variegated objects, such as numerals or letters, were employed. As measurement of span would not depend upon counting identical objects, but accurate identification of a number of dissimilar ones, the perceptual span could be equated with the number of correct identifications made. However, this would still leave the subject, once a target had been briefly presented, having to recite a list of identifying characteristics (e.g., the names of letters) in order. The accuracy of this kind of report would depend upon the decay characteristics of subjects’ short term memory as well as the span of their perception. In order to minimise the use of recall, further refinements were introduced by which the accuracy of subjects’ immediate reports concerning one part of the object field would be used to infer the completeness of their perception of the whole (Sperling, 1960). Thus, if letters were presented on rows on a tachistoscopic slide, a signal would be given immediately following their presentation that indicated which row’s letters should be recited. Naturally, for partial reports to provide an accurate measure of perceptual span for the field as a whole, multiple presentations for a given size of field are required in order to minimise the variation that sampling inevitably introduces.

The ultimate refinement of this trend towards partial reporting and repeated measures came with the method advocated by Estes and Taylor (1961). They proposed that an estimate of the perceptual span could be obtained from the accuracy with which a single target letter, embedded within an object field of known size, was reported over a series of trials. This target can occupy any position at random within the object field, being a letter that can take one of two forms (e.g., ‘T’ or ‘F’), neither of which are represented among the letters which make up the rest of the objects to which the subject is exposed. The subject responds to the presentation by naming whichever of these two alternative forms they believe the target to have taken. Once the size of the whole array exceeds the subject’s perceptual span, the proportion of guesses among their responses can be expected to increase. Provided correct and incorrect guesses are made with comparable frequency, a clear plateau will be evident as perceptual span fails to increase despite an increase in the number of objects in the arrays. For a series of presentations of a given size of letter array, the perceptual span, P, can be derived from the formula:

\[
\text{Span} = \frac{\text{No. of elements displayed} \times (\text{Proportion of correct reports} \times \text{Proportion of incorrect reports})}{\text{Proportion of correct reports} - \text{Proportion of incorrect reports}}
\]

This reduces to:

\[
\text{Span} = \frac{\text{No. of elements displayed} \times \text{Proportion of correct reports} - \text{Proportion of incorrect reports}}{\text{Proportion of correct reports} - \text{Proportion of incorrect reports}}
\]
3: An investigation of attention

\[ S = D (2P(C) - 1) \]

where:

- \( S \): Span
- \( D \): Display size
- \( P(C) \): Proportion of correct reports

Estimates of perceptual span obtained in this way appear to have demonstrated the "upper limit" effect that the concept of a finite span assumes, with consistency between the estimates obtained for a given individual. As the criticisms of previous memory-dependent methods would predict, the values of span obtained by Estes and Taylor's method have been significantly greater than those obtained by earlier techniques. However, as awareness has grown of the extent to which computed values of "perceptual span" are susceptible to experimental manipulation, the original quest of Hamilton and Jevons to establish an absolute figure for an apparently fixed capacity has been abandoned. It is curious that no studies appear to have been performed in "normal" subjects to establish how well an upper limit for span is maintained if the object field is progressively increased beyond about 11 objects.

Sophisticated methods of measuring perceptual span have been adopted with clinical populations other than patients with hysteria, and these are summarised in the next section. Although these all refer to the concept of perceptual or apprehensive span, they have tended to confine themselves to apparent differences in the number of objects perceived in a limited range of conditions, instead of investigating whether a reduced value for "span" was a relatively constant attribute of a group of patients across a range of circumstances.

3.14(iii) Clinical applications of perceptual span.

Most applications of assessment of perceptual span to abnormal psychology have been in schizophrenia research. In the 1960s John Neale had been severely critical of the experimental paradigms being used to investigate attentional deficits in schizophrenic patients. Believing methods such as the inference of visual scanning from measurements of object constancy to be both unnecessarily indirect in design and ambiguous in interpretation, he expected measurement of perceptual span among patient groups to be comparatively free from interference by aberrant motivational and response sets.

Neale reported that schizophrenic patients, when compared with institutionalised volunteers lacking a psychiatric history, tended to fail to extend their perceptual span as the size of an object field was increased beyond a quickly reached threshold (Neale et al, 1969). Neale felt this finding should prompt further investigation of its mechanism, offering some
tentative interpretations. Neale measured span by the tachistoscopic method of Estes and Taylor, presenting letters on cards for fields of 1, 4, 8 or 12 objects. By also asking each subject to respond to each card twice, over two testing sessions a day apart, he showed that correct target detection on the second occasion was significantly related to correct detection on a given card the first time around, to a similar degree for both schizophrenics and controls. This suggested that the schizophrenics' results could not be attributed to a simple defect in the consistency of their scanning style. In addition, Neale examined the possibility that the impairment of performance with the larger object fields represented a distraction effect dependent on the immediate proximity of other letters. However, for a given size of object field, the errors in detection seemed unrelated to how close the distractors were to the target within it, leaving the hypothesis that performance depended directly on the total number of objects present intact (Neale, 1971).

Neale's findings concerning a reduced span among schizophrenic patients received further support from a series of studies by Asarnow and MacCrimmon that, with minor differences in methodology, compared "clinically remitted" schizophrenic patients against a group with bipolar affective disorder as well as "normal" controls. They reported a less marked decrease in the maximum span among this other patient group (Asarnow & MacCrimmon, 1978, 1981). Asarnow and MacCrimmon also reported impairment of span among the first degree relatives of schizophrenic patients, and among children believed to be at an elevated risk of developing the illness, taking these as convergent evidence that this deficit was a "marker" for schizophrenia (MacCrimmon et al, 1980).

These claims were subsequently challenged. Strauss et al (1984) found comparable deficits among patients with schizophrenia and mania (as opposed to depression) and inferred that reduction in span was associated with acute psychosis rather than schizophrenia per se. However, Strauss' team not only ignored the way in which Asarnow and MacCrimmon had targeted clinically remitted patients, but failed to test their own patients' performances up to the size of display (10 elements) at which Asarnow and MacCrimmon had discovered the greatest discrimination between schizophrenic and bipolar groups. A more recent challenge to claims for the diagnostic specificity of reductions in perceptual span came from an investigation by Harris and Hanish (1987) into the relationship between span, anxiety and arousal. They replicated the procedure of Asarnow and MacCrimmon exactly, and in a college student population found that, although induced arousal failed to influence span, a decrement for the largest display similar to that reported for schizophrenic subjects was present among students with high trait anxiety on the MAS. They concluded that reduced span reflected cognitive aspects of anxiety rather than arousal per se, making no real attempt to link this finding to data from schizophrenia research. There, the most coherent explanatory hypothesis has remained that of Neale (1971) who attributed selectively poor performance on tests of span to a more rapid rate of decay of perceptual traces. Although Neale suggested an experimental test (such rapidly disappearing traces
should be particularly susceptible to "backward masking" as new material presented immediately afterwards interferes with processing of them) it appears never to have been attempted. If other clinical groups, such as patients with conversion symptoms, proved to share a similar pattern of restriction in perceptual span, more detailed work on contributory processes might result.

Harris and Hanish's study of anxiety was not developed by them, but has two significant implications for the proposed investigation. One is that changes in span are more likely to be found among populations with established pathology rather than those exhibiting induced transient changes. The second is that "neurotic" conditions would repay closer investigation than these have received in a field dominated by studies of psychosis. An investigation of perceptual span among patients with conversion hysteria would therefore complement and extend previous clinical studies.


The method of Estes and Taylor (1961) has been adopted in virtually all clinical studies of perceptual span since it was introduced. This requires letter arrays representing a range of sizes to be presented using a tachistoscope over brief exposures, typically 50 to 100 milliseconds in duration. At least one of the arrays used should be of a size calculated to exceed the expected span of any of the subjects. The subject receives an anticipatory signal as each presentation is due, and their subsequent report on the target letter is recorded by the experimenter. Because it relies on partial reports, many presentations are required in quick succession, usually well in excess of 100, making the procedure potentially tedious for both subject and experimenter, as well as being prone to error during the transcription of long sequences of verbal reports.

Although microcomputers have not been used previously in measurement of perceptual span, they recommend themselves for this on several counts. Subjects are likely to appreciate the shortening of experimental sessions that automation can bring, as well as technology that is more comfortable to use, and indeed more likely to be personally familiar to them. Experimenters should be able to capitalise on the fact that computer-administered object presentations could be coupled to the input of responses by subjects, allowing immediate sorting and storage of data ready for subsequent analysis. Both groups are likely to benefit from the greater portability of computerised tests.

Microcomputers also offer new possibilities for the assessment of perceptual span beyond transposition of existing methodology. Their graphic potential could allow changes in the format of an object field to be made without the necessity of producing a complete set of transparencies, facilitating precise experiment on the effect of changes in the detail and inter-relationship of objects within the object field on span. Further, partial report techniques of
the sort already discussed could be refined considerably once any part of the object field can be indicated electronically in order to cue an immediate report. This would abolish the need to distinguish between target and other objects, and reduce the high number of repeated observations that are required when correct reports occur frequently by chance.

The investigation outlined below (section 3.4) confined itself to transposing partial report techniques onto microcomputer in order to check first that results obtained in this way would be compatible with those obtained by traditional means.

3.2 Hypotheses for studies.

These surveys suggested that the two Janetian hypotheses proposed in section 3.11 could be recast in terms that were amenable to investigation using a dual task paradigm and a study of perceptual span respectively:

Hypothesis a: Patients with hysterical symptoms will show greater interference between the simultaneous performance of sequential manual tapping tasks and vocalised verbal tasks compared with patients with similar symptoms that are attributed to recognised neurological disease.

Hypothesis b: Patients with hysterical symptoms will also differ on tests of perceptual span, registering fewer objects on average when the object field is large, and demonstrating a greater variability in the number of objects seen for a given field size, compared to controls.

3.3 Study 1: The division of attention between verbal and sequential manual tasks in patients with pseudoneurological symptoms.

3.31 Method

3.31 (i) Subjects.

33 subjects completed testing for this study. They were all consenting patients attending the National Hospitals who had received investigation for a single salient presenting symptom. They were distributed among three groups as follows:

Group 1. Gait disorders not attributed to neurological disease (n=11). These patients had received investigation for weakness, or inco-ordination of the lower limbs that had impaired their mobility. Upper limb weakness had not been noted on routine examination by their neurologists. 3 of the sample were in wheelchairs.
3: An investigation of attention

Group 2. Pseudoseizures (n = 12). These patients were experiencing seizures which recent investigation indicated should not be attributed to epilepsy, irrespective of diagnoses that had been made in the past.

Group 3. Gait disturbance attributed to neurological disease (n=10). Patients were either ambulant or able to manoeuvre a wheelchair. The majority were in-patients of the hospital at the time of testing. Individual diagnoses included multiple sclerosis, lumbar spondylosis and tropical sprue (cf. appendix 2).

Nearly all of these patients also participated in the study of perceptual span to be described in section 3.4. A summary of the clinical diagnoses given to all patients in either study is contained in appendix 2.

Exclusions. During recruitment of patients, a small number of the patients recommended for consideration by their neurologists had to be excluded from the study. This would occur if there was clear evidence of upper limb weakness or inco-ordination likely to impair performance; or if the patient failed to complete the test through inability to master its basic procedures, subsequent refusal, or equipment failure. Unfortunately, the latter event prevented results being obtained from three patients participating in later testing sessions. Exclusions affected group 1 and group 3. The group whose lower limb weakness was attributed to conversion disorder included one patient who was unable to complete testing because she repeatedly insisted she could not move her fingers, and ended the session lying on the floor of the testing room. Another patient was unable to learn the test procedure despite coping with the demands of the perceptual span study. Two other patients' results were unobtainable due to equipment failure.

Among group 3, where patients' lower limb weakness was attributed to neurological disorders, two patients had clear evidence of upper limb weakness during neurological examination; another had been unable to complete the test complaining of mounting pain and wishing to stand; testing could not be completed due to equipment failure in a further case.

Handedness. Of the 33 patients studied, only 4 reported themselves as being left-handed. Because variations in the lateralisation of higher function might itself lead to observable differences in performance, a precise assessment of laterality was attempted by administering the Edinburgh Inventory (Oldfield, 1971) to all subjects. This assesses relative hemispheric dominance from responses to an inventory of 20 actions for which a preference for either left or right is usual. (A score of 100 indicates left sided dominance across all items, 0 equipotentiality and a negative score a right hemisphere dominance). Characteristics of the three groups are summarised in table 3.1:
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<table>
<thead>
<tr>
<th></th>
<th>Mean age (median)</th>
<th>Sex (Male/total)</th>
<th>Handedness (Left-handed)</th>
<th>Laterality quotient (s.d.)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group 1 (n=11)</strong> (Hysterical gait)</td>
<td>42.5 (43)</td>
<td>1/11 (9%)</td>
<td>1/11 (9%)</td>
<td>61.4 (54.3)</td>
</tr>
<tr>
<td><strong>Group 2 (n=12)</strong> (Pseudoseizures)</td>
<td>33.7 (32.5)</td>
<td>3/12 (25%)</td>
<td>2/12 (17%)</td>
<td>62.5 (65.4)</td>
</tr>
<tr>
<td><strong>Group 3 (n=10)</strong> (Organic gait)</td>
<td>40.1 (38)</td>
<td>3/10 (30%)</td>
<td>1/10 (10%)</td>
<td>69.4 (48.0)</td>
</tr>
</tbody>
</table>

*Table 3.1 Subjects for the dual attention study.*
3.31(ii) Procedure.

Sequential tapping by either hand was recorded independently over a fixed 30-second interval, across three separate conditions: silence; repetition of a simple phrase; and repetition of a ‘tongue twister’. To determine the number of completed tapping sequences in any 30 second period, Lomas and Kimura’s method (described above in section 3.13(ii) ) was adapted for use with a BBC B microcomputer. This computer was chosen for programming and administration of the tests because of the ease with which custom-built peripheral devices can be used with it. The computer was linked to a box having adjacent contact strips for each of their four fingers, and a separate area that had to be touched by the ball of the hand for the system to operate. (This effectively replicated Lomas and Kimura’s use of independent telegraph keys that all 4 fingers tapped in turn, while contact between a subject’s palm and the supporting surface was maintained.) The programme that was written required each finger to touch and leave its strip before the adjacent one was touched for the sequence to count as a properly executed one. The direction of each sequence was always from forefinger to littlefinger, the computer programme reversing the sequence in which contacts were expected between each recording period so that right and left hands could be recorded alternately.

Once a subject had been introduced to the equipment and the recording programme commenced on the computer, each subject had a trial of tapping with either hand, during which the experimenter received visual feedback as to how successfully and quickly sequences were being completed. This allowed for gross errors in technique to be corrected before a standardised sequence of timed recordings began. This sequence is summarised in table 3.2. "Simple tapping" refers to periods over which the subject remained silent. In periods of recitation of an "easy phrase", subjects would repeat the phrase "red for stop and green for go" throughout the 30 seconds while tapping. In periods of recitation of a "demanding phrase", subjects had to continuously repeat the phrase "red leather yellow leather" while they tapped. (Both phrases contained seven syllables, and had been repeated at a comparable rate by volunteer subjects asked to recite them without tapping.) The sequence had been chosen, following advice from a neuropsychologist, to minimise the impact of practice. The order of 'easy' and 'difficult' phrases was reversed through the second half of the sequence so that a mean score for either of these within a given sequence would minimise the impact of order on performance under a given condition (cf. table 3.2).

The experimenter recorded the number of complete phrases uttered at the end of each of these 8 recitations. The computer recorded the number of completed sequences for each of the 18 periods in the test, and provided the experimenter with a written printout of these on completion of each set of 18 tests (The complete programme, in BBC basic, written entirely by the author, will be found in appendix 3).
Table 3.2: Sequence of experimental conditions in tapping test.

<table>
<thead>
<tr>
<th>Right hand</th>
<th>Left hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple tapping</td>
<td>1</td>
</tr>
<tr>
<td>Tapping plus recitation of easy phrase</td>
<td>3</td>
</tr>
<tr>
<td>Simple tapping</td>
<td>5</td>
</tr>
<tr>
<td>Tapping plus recitation of demanding phrase</td>
<td>7</td>
</tr>
<tr>
<td>Simple tapping</td>
<td>9</td>
</tr>
<tr>
<td>Tapping plus recitation of demanding phrase</td>
<td>11</td>
</tr>
<tr>
<td>Simple tapping</td>
<td>13</td>
</tr>
<tr>
<td>Tapping plus recitation of easy phrase</td>
<td>15</td>
</tr>
<tr>
<td>Simple tapping</td>
<td>17</td>
</tr>
</tbody>
</table>
3.32 Results

3.32(i) Tapping performance.

Table 3.3 summarises the variation in tapping performance through all the experimental recordings, irrespective of any additional tasks. The mean score for each subject across 9 right-handed trials and 9 left handed trials are given, together with their standard deviation as an indication of intra-subject variation. The subjects are listed according to membership of the 3 experimental groups, allowing the variability of performance within each group to be compared. Left-handed subjects (who all had a negative score on the laterality index) are indicated with an asterisk: the subject with a double asterisk had a laterality score of minus 100 indicating unusually strong lateralisation.

In all the groups, left hand mean scores (and standard deviation) are lower than the right hand scores. Although the mean scores for either hand of members of group 1 are considerably below those of either group 3 or group 2, the standard deviation across group 1’s scores is very little less than standard deviations for the other groups. The range is similar across all groups, although lower within group 1 for the left hand. Distribution of scores is skewed towards median values higher than the mean in groups 2 and 3.
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<table>
<thead>
<tr>
<th>Group 1</th>
<th>Right hand</th>
<th>Left hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject 1</td>
<td>5.44 (0.53)</td>
<td>3.56 (2.07)</td>
</tr>
<tr>
<td>Subject 2</td>
<td>0.67 (0.71)</td>
<td>3.00 (1.00)</td>
</tr>
<tr>
<td>Subject 3</td>
<td>3.33 (1.80)</td>
<td>3.56 (2.19)</td>
</tr>
<tr>
<td>Subject 4</td>
<td>2.89 (2.57)</td>
<td>3.33 (2.24)</td>
</tr>
<tr>
<td>Subject 5</td>
<td>11.11 (3.92)</td>
<td>2.11 (0.93)</td>
</tr>
<tr>
<td>Subject 6</td>
<td>7.89 (2.15)</td>
<td>6.67 (1.00)</td>
</tr>
<tr>
<td>Subject 7</td>
<td>8.56 (1.01)</td>
<td>8.56 (0.53)</td>
</tr>
<tr>
<td>Subject 8</td>
<td>4.44 (2.74)</td>
<td>5.56 (2.96)</td>
</tr>
<tr>
<td>Subject 9**</td>
<td>1.22 (1.99)</td>
<td>3.00 (1.00)</td>
</tr>
<tr>
<td>Subject 10</td>
<td>2.44 (2.19)</td>
<td>3.44 (1.33)</td>
</tr>
<tr>
<td>Subject 11</td>
<td>3.00 (1.87)</td>
<td>2.67 (1.41)</td>
</tr>
<tr>
<td><strong>Group mean:</strong></td>
<td>3.95 (2.91)</td>
<td>3.63 (1.73)</td>
</tr>
</tbody>
</table>

| Median (25%; 75%): | 4.0 (1.0;7.0) | 4.0 (2.0; 6.0) |
| Range: | 0 - 18 | 0 - 10 |

<table>
<thead>
<tr>
<th>Group 2</th>
<th>Right hand</th>
<th>Left hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject 12</td>
<td>2.67 (1.50)</td>
<td>3.33 (1.66)</td>
</tr>
<tr>
<td>Subject 13</td>
<td>8.33 (4.47)</td>
<td>8.33 (5.17)</td>
</tr>
<tr>
<td>Subject 14</td>
<td>9.33 (2.40)</td>
<td>8.11 (1.54)</td>
</tr>
<tr>
<td>Subject 15</td>
<td>13.0 (2.12)</td>
<td>9.00 (3.97)</td>
</tr>
<tr>
<td>Subject 16*</td>
<td>5.78 (1.93)</td>
<td>5.11 (1.76)</td>
</tr>
<tr>
<td>Subject 17</td>
<td>10.44 (1.94)</td>
<td>10.00 (3.35)</td>
</tr>
<tr>
<td>Subject 18**</td>
<td>8.56 (2.24)</td>
<td>9.22 (1.39)</td>
</tr>
<tr>
<td>Subject 19</td>
<td>10.44 (2.35)</td>
<td>8.00 (3.61)</td>
</tr>
<tr>
<td>Subject 20</td>
<td>10.0 (1.12)</td>
<td>8.00 (1.73)</td>
</tr>
<tr>
<td>Subject 21</td>
<td>10.56 (1.51)</td>
<td>10.00 (2.91)</td>
</tr>
<tr>
<td>Subject 22</td>
<td>6.44 (0.89)</td>
<td>7.33 (0.71)</td>
</tr>
<tr>
<td>Subject 23</td>
<td>2.67 (1.66)</td>
<td>4.00 (0.71)</td>
</tr>
<tr>
<td><strong>Group mean:</strong></td>
<td>7.19 (2.84)</td>
<td>6.56 (1.95)</td>
</tr>
</tbody>
</table>

| Median (25%; 75%): | 8.5 (5.5;11.0) | 8.0 (5.0;10.0) |
| Range: | 0 - 16 | 0 - 17 |

<table>
<thead>
<tr>
<th>Group 3</th>
<th>Right hand</th>
<th>Left hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject 24*</td>
<td>9.22 (6.51)</td>
<td>8.44 (4.16)</td>
</tr>
<tr>
<td>Subject 25</td>
<td>9.78 (3.23)</td>
<td>5.89 (3.22)</td>
</tr>
<tr>
<td>Subject 26</td>
<td>2.56 (0.53)</td>
<td>2.44 (0.53)</td>
</tr>
<tr>
<td>Subject 27*</td>
<td>7.78 (3.15)</td>
<td>8.56 (1.13)</td>
</tr>
<tr>
<td>Subject 28</td>
<td>8.22 (2.77)</td>
<td>8.00 (2.06)</td>
</tr>
<tr>
<td>Subject 29</td>
<td>10.56 (1.51)</td>
<td>9.11 (1.36)</td>
</tr>
<tr>
<td>Subject 30</td>
<td>3.33 (2.12)</td>
<td>3.78 (1.79)</td>
</tr>
<tr>
<td>Subject 31</td>
<td>10.11 (1.27)</td>
<td>10.22 (1.30)</td>
</tr>
<tr>
<td>Subject 32</td>
<td>8.22 (1.09)</td>
<td>7.22 (0.83)</td>
</tr>
<tr>
<td>Subject 33</td>
<td>8.78 (1.99)</td>
<td>5.56 (2.07)</td>
</tr>
<tr>
<td><strong>Group mean:</strong></td>
<td>6.82 (2.47)</td>
<td>6.09 (2.29)</td>
</tr>
</tbody>
</table>

| Median (25%; 75%): | 8.0 (4.0;10.0) | 8.0 (4.0;9.0) |
| Range: | 0 - 17 | 1 - 14 |

Table 3.3: Mean scores (and standard deviations) for number of completed sequences for individual subjects across all conditions.

*/* = non-dominant laterality: see text
3.32 (ii) Impact of simultaneous verbal tasks.

Table 3.4 summarises the mean number of sequences completed by each group for each of the three imposed conditions, viz. without vocalisation ("simple"); during recitation of the easy phrase ("easy") and during recitation of the demanding phrase ("hard"). Although the number of sequences completed when reciting the "demanding" phrase (columns 5 & 6) is not always lower than when reciting the "easy" one (columns 3 & 4), the mean number of sequences was always less under either of these conditions than for tapping in the absence of vocalisation (columns 1 & 2).

3.32 (iii) Verbal productions.

The mean number (and standard deviation) of completed phrases under each condition are recorded for each patient group in table 3.5. Patients were likely to complete between 5 and 8 phrases irrespective of the condition. The only indication of a tendency to complete fewer "hard" than "easy" phases was restricted to members of group I. All subjects showed a consistent tendency for scores on the second trial under a given condition to be higher than on the first.
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<table>
<thead>
<tr>
<th>Group 1</th>
<th>R simple</th>
<th>L simple</th>
<th>R+easy</th>
<th>L+easy</th>
<th>R+hard</th>
<th>L+hard</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4.9</td>
<td>4.53</td>
<td>4.18</td>
<td>3.82</td>
<td>4.32</td>
<td>3.45</td>
</tr>
<tr>
<td></td>
<td>(3.47)</td>
<td>(2.12)</td>
<td>(4.20)</td>
<td>(2.14)</td>
<td>(2.32)</td>
<td>(2.26)</td>
</tr>
<tr>
<td>Group 2</td>
<td>8.73</td>
<td>8.15</td>
<td>7.62</td>
<td>6.30</td>
<td>7.38</td>
<td>7.25</td>
</tr>
<tr>
<td></td>
<td>(3.65)</td>
<td>(2.67)</td>
<td>(3.54)</td>
<td>(3.03)</td>
<td>(2.92)</td>
<td>(2.60)</td>
</tr>
<tr>
<td>Group 3</td>
<td>8.54</td>
<td>7.56</td>
<td>6.95</td>
<td>5.80</td>
<td>7.05</td>
<td>7.45</td>
</tr>
<tr>
<td></td>
<td>(3.14)</td>
<td>(2.72)</td>
<td>(3.57)</td>
<td>(2.95)</td>
<td>(2.96)</td>
<td>(2.63)</td>
</tr>
</tbody>
</table>

Table 3.4. Mean number (standard deviation) of completed sequences by each hand across the three experimental conditions.

<table>
<thead>
<tr>
<th>Group 1</th>
<th>&quot;Easy&quot; phrase (Right)</th>
<th>&quot;Easy&quot; phrase (Left)</th>
<th>&quot;Hard&quot; phrase (Right)</th>
<th>&quot;Hard&quot; phrase (Left)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6.27</td>
<td>5.59</td>
<td>5.64</td>
<td>5.0</td>
</tr>
<tr>
<td></td>
<td>(2.57)</td>
<td>(1.67)</td>
<td>(2.11)</td>
<td>(1.32)</td>
</tr>
<tr>
<td>Group 2</td>
<td>6.79</td>
<td>7.0</td>
<td>6.91</td>
<td>6.92</td>
</tr>
<tr>
<td></td>
<td>(2.24)</td>
<td>(2.38)</td>
<td>(1.88)</td>
<td>(2.0)</td>
</tr>
<tr>
<td>Group 3</td>
<td>7.05</td>
<td>7.1</td>
<td>7.0</td>
<td>7.15</td>
</tr>
<tr>
<td></td>
<td>(1.95)</td>
<td>(1.97)</td>
<td>(2.11)</td>
<td>(2.30)</td>
</tr>
</tbody>
</table>

Table 3.5 Mean number (standard deviation) of completed easy and difficult phrases during each of the dual task conditions.
3.32 (iv) Analysis of covariance of tapping rate with other variables.

Sequential analysis of variance was performed using the Statistical Package for the Social Sciences (Norusis, 1995) to assess the significance of three variables in accounting for variance within the overall rate of sequence completion. These were: clinical group; concurrent verbal task; and the hand used.

Between groups: \( F=6.23 \) (2df); \( p=0.005 \)
Between hands: \( F=3.04 \) (1df); \( p=0.091 \)
Concurrent task: \( F=4.57 \) (2df); \( p=0.014 \)

Group v. hand. \( F=0.09 \) (3df); \( p=0.92 \)
Hand v. difficulty \( F=0.52 \) (3df); \( p=0.60 \)
Group v. difficulty \( F=0.21 \) (6df); \( p=0.93 \)

Group v. hand v. difficulty \( F=0.62 \) (8df); \( p=0.65 \)

The analysis indicates that the three factors are independent of one another, and that two of them do have a significant impact on tapping rate. Thus the tendency for the number of sequences to diminish with either kind of vocalisation (table 3) and the poorer performance of patients with hysterical gait disorders compared with the other two experimental groups both appear to be robust findings.

3.32 (v) Analysis of covariance of vocalisations with other variables.

Data concerning the number of completed vocalisations of easy and difficult phrases were also subject to analysis of variance to establish how far the observed variations could be attributed to group membership, the hand that was concurrently tapping, and the supposed difficulty of the seven-syllable phrase that was being repeated.

Between groups \( F=1.88 \) (2df) (\( p=0.17 \))
Between hands \( F=1.10 \) (1df) (\( p=0.30 \))
Difficulty of phrase \( F=0.82 \) (1df) (\( p=0.37 \))

Group v. hand \( F=3.11 \) (2df) (\( p=0.06 \))
Group v. difficulty \( F=0.92 \) (2df) (\( p=0.41 \))
Hand v. difficulty \( F=0.01 \) (1df) (\( p=0.91 \))

Group v. difficulty v. hand \( F=0.25 \) (2df) (\( p=0.78 \))
3: An investigation of attention

These indicate that the selective slowing in the vocalisation rate when members of group 1 recited either phrase to left hand tapping approached usual standards of statistical significance (cf. table 3.5), but the rate of vocalisation appeared completely unrelated to other factors.

3.33 Discussion.

It is evident from the results that the main hypothesis of the study, that a selectively greater decrement in sequential tapping would be observed among all patients with hysterical symptoms when attention was shared with a requirement to vocalise verbally, has not been substantiated. Instead, there are clear differences in sequential tapping performance between the two "hysterical" groups that were tested: patients with hysterical gait difficulties failed to do this as well as subjects from either of the other groups. This difference was evident whether or not there was a concurrent task, and did not significantly differ according to the hand that was tapping. Not only were the mean scores of patients in this group lower across all conditions, but the performance of individual patients varied greatly from trial to trial. In addition, while members of this group shared a tendency with members of the other two groups to improve their performance on the vocal tasks with practice, there was a trend for these patients to complete fewer phrases in their vocalisations than members of the other groups throughout, and for this to be more marked when they were simultaneously tapping with the left hand (table 3.5).

The appearance of a group-specific difference associated with impairment of performance in the simplest tasks attempted here (tapping without vocalisation) indicated that the attempt to search for more subtle effects, evident only in a situation of attentional competition, was premature. As table 3.3 indicates, there is considerable inter-individual difference among members of this group, particularly with respect to right hand performance, so the poorer performance of the group as a whole would not necessarily have emerged during the small number of trial runs that had been attempted before the formal study.

In trying to account for this inter-group difference, the fact that both tapping and vocalisation were consistently performed more poorly by subjects with lower limb weakness not attributed to neurological disease, the tapping task to a statistically significant extent, suggests that many of these patients may suffer an impairment of co-ordination or concentration not found among members of either of the other two groups. Individual members of this group were far more likely to fail to complete a single correct sequence over a 30 second period, particularly with the right hand, than members of either of the other 2 groups. Thus, out of the 99 30-second tapping trials the 11 patients in this group attempted across all conditions, no sequences were completed on 16 of them. Five patients accounted for one or more of these failed trials. This compares with a single failure in the 108 trials for group 2, and 2 across the 90 trials for group 3. (Chi-squared: 23.70; p=0.0000).
Inspection of table 3.4 confirms there were also more likely to be discrepancies of more than 50% between performance with one hand compared with the other for members of group 1, but that the direction of this would not always be predicted by the subject's laterality index.

An intermittent but marked impairment of performance confined to this group had not been expected, and the experiment is able to contribute little in specifying conditions in which it might not apply as this was not the principal focus of its design. Further experiments would be indicated to ascertain whether the impairment was sensitive to other parameters held constant in this protocol, eg. the length of the trial period or the nature of the manual task, and any adjustments that could improve or worsen the performance of this group that had no impact on the (better) performance of the control groups used here.

It is also difficult from the available data to know with which common factor(s) the poorer performance of this group may be most strongly associated. The pseudoseizure patients also had pseudoneurological symptoms but did not have this apparent deficit. As table 3.1 shows, there was no real difference in age, sex or laterality that distinguished the patients whose lower limb weakness was attributed to neurological disease (group 2) and those where it was presumed to be psychogenic (group 1). It is possible that other shared psychological characteristics, such as depressed mood, could have contributed. This is inherently unlikely given that a comparison of pseudoseizure patients and those presenting with motor weakness within the cohort described in chapter 2 has shown the former were more likely to have received an additional diagnosis of depression (cf. Mace, 1996), while patients with neurological disorders seen in a neurological hospital are equally likely to be depressed (cf. Wilson Barnett and Trimble, 1985).

A less tidy explanation would be that the group with aberrant scores was simply less homogenous as a group, containing members whose performance was more likely to be compromised by possibly undetected neuropathology. It may be relevant that Leonard et al (1988), in an investigation of the impact of cerebral injury on fine motor performance, discovered that sequential tapping was selectively impaired among patients with unilateral temporal and frontal lesions. This association does make the failure of the pseudoseizure group to share this impairment puzzling. Given the growing literature that has linked cases appearing to have pseudoepileptic seizures to lesions of this kind (e.g. Fusco et al, 1990; Kanner et al, 1990), patients in group 2 might have been expected to be more likely to have difficulties in sequential tapping as a result of covert neuropathology. This possibility also highlights how the study could have been strengthened by inclusion of a second control group who had confirmed seizure disorders, several of whom might have had poorer performances if Leonard's observations are correct.

The experiment had been designed to highlight interference between the two kinds of tasks. In showing a small but consistent decrement across all groups when vocalisations accompanied tapping compared to tapping alone (table 3.4), it succeeded in doing this.
However, as table 3.4 also shows, no significant differences were detected in tapping performance according to whether the 'easy' or 'hard' vocalisation tasks were selected. This suggests these may not necessarily have represented a valid or sufficiently sensitive method of presenting different levels of demand. This would be consistent with a series of recent dual-task experiments that pitted finger tapping against a wide range of speech tasks (Steiner et al 1992; Green et al 1994). These demonstrated that greater degrees of interference (with performance by either hand) were found as the cognitive complexity of a speech task increased, as in moving from recitation of unfamiliar technical material, through recitation of "interesting" material, to paraphrasing of material as it was read. Their condition of reading aloud "humorous" material, which corresponded the most closely with the "tongue twister" condition here, had comparatively little additional impact on tapping performance than impersonal recitation (Green et al, 1994) and was not viewed as a more complex task cognitively.

Interpretation of why interposition of a vocal task affected tapping performance, but the change of vocal task did not, can also benefit from the work of Heuer (1991). Heuer has argued that variations in performance in dual task experiments often need to be explained in terms of the specific response requirements. Very different degrees of interference can be obtained between the same pair of tasks when these requirements are altered. Heuer has been sceptical about explanations of interference based on functional capacity because factors such as the timing of tasks can be critical. According to Heuer, who cites evidence from studies of vocal as well as tapping tasks, the extent to which tasks interfere with one another can be explained by reference to their rhythmic incompatibility, rather than their intrinsic demands or cerebral location. In the present study, the "easy" and "difficult" verbal tasks were of exactly the same length. It was observed in the course of the experiment that subjects who maintained an efficient performance tended to synchronise their tapping and recitation cycles. Precise comparisons here are limited because the experiment was more tolerant in its assessment (by the experimenter) of whether each verbal sequence was completed correctly, compared with the rigorous monitoring of each tapping sequence (via the electronic keypad). Comparison of the mean sequence completion rates and phrase completion rates for each of the experimental conditions from figures 3.4 and 3.5 respectively indicates they are likely to have maintained a temporal coupling, such that fewer completed tapping sequences would be accompanied by fewer phrases in each trial and vice versa. Had the "difficult" speech task imposed a more demanding rhythm than the "easy" speech task, then a greater decrement of tapping performance under this condition could have been expected, irrespective of content.

The experiment does not exclude the possibility therefore that additional decrements in performance might be found in clinical groups with pseudoneurological symptoms if an alternative task that is more demanding by virtue of it cognitive or rhythmic complexity were to be used. However, Janet himself had claimed an uncomplicated vocalisation would be
sufficient to prevent co-ordinated finger movements in hysterical patients (cf. section 3.1).
The experiment clearly shows this not to be so. It is possible that his patients, who were
usually chronically hospitalised women exhibiting a wide variety of dissociative phenomena as
well as apparently reversible pseudoneurological symptoms, were not from a comparable
population to the subjects studied here.

Careful account had been taken throughout the experiment that performance might
differ according to the hand that was tapping when vocalisation was also required, and that
this was likely to be sensitive to differences in handedness. A significant contrast between
the findings here with the literature on normal subjects cited in section 3.13(ii) was the lack of
lateralised bias in the interference that did occur. There was neither greater interference of
right-handed tapping among right-handed subjects, nor facilitation among left handed ones
(cf Ikeda (1987); Lomas & Kimura (1976)). Furthermore, the finding that completed
vocalisations were likely to be fewer when the left hand was performing rather than the right
hand (table 3.5) is also contrary to the expected direction of any interference between the
tasks. No support is evident from these results for the idea that, in the terms of Kinsbourne's
theory of "functional cerebral space", vocalisation and motor sequences of the dominant
hand are functionally closer than the non-dominant hand, nor for thinking that this functional
map is different among patients prone to hysterical symptoms than other groups.

The positive implication of the experiment is that further work appears justified to try
and define the nature of the performance difficulty that these patients did have. This could
relate to a difficulty in simply maintaining attention on a task beyond a very brief interval,
whether or not it is shared. If this is so, a poorer performance on other tasks requiring
sustained attention, such as the one examined in the following experiment, would be
expected from patients with non-organic lower limb weakness.
3.4 Study 2. A comparative study of perceptual span in subjects with pseudoneurological symptoms and controls with neurological disease

3.41 Method.

3.41(i) Subjects.

Four groups of subjects were studied, distinguished according to the nature of the symptom for which they were currently receiving treatment or investigation, and whether or not this had been attributed to underlying neuropathology in the course of investigation. Criteria for three of the groups were as described under section 3.32(i). Other subjects who fulfilled the criteria outlined there took part in this study in addition to patients from the previous study included 2 patients with lower limb weakness and with functional gait disorders (cf. appendix 2).

The remaining group comprised:

Group 4. Epilepsy (n=13). Subjects had to be experiencing intermittent seizures in the absence of progressive neurological disease. It proved impossible to recruit sufficient subjects with primary generalised epilepsy, and several out-patients with partial and complex partial seizures are included in this group. (Further details of subjects in these studies are summarised in appendix 2).

Because impairments of vision could account for significant differences in outcome, all subjects had their visual acuity tested using a Snellen chart and perimetry performed by the experimenter at the outset of each testing session. 31 subjects had a visual acuity of 5/60; 14 of 6/60. The distribution of the 6 patients having a visual acuity of 9/60 and the 7 having a peripheral field defect of up to 25% are recorded in table 3.6. No patient had both a field defect and visual acuity worse than 6/60.
3: An investigation of attention

<table>
<thead>
<tr>
<th>Group 1</th>
<th>Mean (median) Age</th>
<th>Male Gender (%)</th>
<th>Visual Acuity</th>
<th>Visual Fields</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-organic gait (n=13)</td>
<td>47.5 (50)</td>
<td>2/13 (15%)</td>
<td>4 at 9/60</td>
<td>4 restricted</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group 2</th>
<th>Mean (median) Age</th>
<th>Male Gender (%)</th>
<th>Visual Acuity</th>
<th>Visual Fields</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pseudoseizures (n=12)</td>
<td>33.7 (32.5)</td>
<td>3/12 (25%)</td>
<td>1 at 9/60</td>
<td>1 restricted</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group 3</th>
<th>Mean (median) Age</th>
<th>Male Gender (%)</th>
<th>Visual Acuity</th>
<th>Visual Fields</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organic gait (n=12)</td>
<td>46.25 (51)</td>
<td>3/12 (25%)</td>
<td>1 at 9/60</td>
<td>2 restricted</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group 4</th>
<th>Mean (median) Age</th>
<th>Male Gender (%)</th>
<th>Visual Acuity</th>
<th>Visual Fields</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epilepsy (n=13)</td>
<td>25.1 (24)</td>
<td>2/13 (15%)</td>
<td>All 6/60 or better</td>
<td>No restrictions</td>
</tr>
</tbody>
</table>

*Table 3.6 Characteristics of subjects in perceptual span study.*
3.41(ii) Procedure.

Perceptual span was assessed for all subjects using arrays of 1, 4, 8, and 12 letters. The method adopted represented a modification of the tachistoscopic experiments of Estes and Taylor (1961). Instead of using a tachistoscope, 160 letter arrays were presented using an IBM AT computer with an EGA monitor. A programme devised and written by the experimenter in GW basic (appendix 4) allowed for the letter arrays to be presented in a series of 5 blocks. Each block comprised 8 arrays where a target letter for the subject to identify was the only consonant; 8 where a target letter was accompanied by 3 other letters; 8 where the target letter was accompanied by 7 other consonants; and 8 where there were 11 other consonants. Within each block, the sequence of presentations was randomised. The position of the target consonant, which occupied all the available positions with equal frequency, accordingly changed on each presentation.

An oscilloscope had confirmed that the presentation time was consistently maintained at 68msecs, equivalent to 4 screen cycles. The luminous intensity of the images produced, as white letters in courier typeface on a black backdrop, was 320 candelas per square metre at 1.5 metres; that of the darkened blank screen was 4 candelas per square metre. Background illuminance was 0.37 lux. The display subtended an angle of 2 degrees from where the subjects sat, and was arranged to present images at eye level at a distance of 1.5 metres.

The target letters chosen for the experiment were ‘L’ and ‘Z’ on account of the minimal orthographic confusion that has been reported between them (Townsend, 1971). The programme allowed subjects to respond to the target letter by pressing a key on a mouse. After each response, the subject would see a message on the screen confirming which key they had pressed. This gave them the opportunity to change their response if they recognised this was not the key they intended to press, although they were not allowed to see the previous display again.

After each subject had had the nature of the experiment explained to them and their consent had been obtained, they had their visual acuity assessed using a Snellen chart, and the intactness of their visual fields assessed using perimetry (cf. section 3.41(i)). The computer programme was then started. To acclimatise subjects to the experimental procedure, the programme began with a brief introductory section that introduced the format of the arrays, the countdown that always preceded their presentation, the task they were being set (i.e. to detect either letter ‘L’ or ‘Z’ in each array), the use of the mouse, and how they could change an incorrect response during the 3 seconds immediately following presentation of each field. After this, subjects proceeded to provide specimen responses to a trial block of 32 arrays. The experimenter received immediate feedback from the computer on these, so that any gross difficulties in complying with the terms of the experiment could be identified in advance.
After this trial block, subjects had a minute's break, ended by an audible alarm. Following this, they entered the experiment proper, comprising four further blocks of 32 arrays, each combining the same combination of 1, 4, 8 and 12 letter displays, with no display being repeated. The supporting programme (appendix 4) recorded the proportion of targets each subject successfully identified for each size of array during each of these blocks. These could be displayed to the experimenter at the end of each session, and were also printed out using an attached printer.

3.4.2 Results.

All groups showed a progressive decrement in the proportion of target letters that were correctly identified as the size of the letter array increased (table 3.7). Only scores for the 8 and 12 letter arrays approximated to a normal distribution, it being apparent from the standard deviations that no group showed a much greater spread of scores than another. As each column of table 3.7 shows, the Kruskall Wallace statistic indicated no significant difference between group scores for any of the array sizes.

The equation of Estes and Taylor (1961; section 3.1c above) was used to calculate mean and median values for "perceptual span" in each group. Mean values for span were consistently higher for arrays containing 12 letters than arrays containing 8 letters, but did not exceed 6.1 in either condition (table 3.8).
<table>
<thead>
<tr>
<th></th>
<th>1 letter</th>
<th>4 letters</th>
<th>8 letters</th>
<th>12 letters</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group 1</strong></td>
<td>31.00 (1.73)</td>
<td>28.00 (2.86)</td>
<td>23.46 (2.96)</td>
<td>22.54 (2.67)</td>
</tr>
<tr>
<td><strong>Group 2</strong></td>
<td>31.50 (0.90)</td>
<td>30.00 (2.00)</td>
<td>26.17 (3.74)</td>
<td>24.17 (3.10)</td>
</tr>
<tr>
<td><strong>Group 3</strong></td>
<td>31.50 (0.67)</td>
<td>29.41 (2.35)</td>
<td>24.56 (3.85)</td>
<td>23.42 (3.68)</td>
</tr>
<tr>
<td><strong>Group 4</strong></td>
<td>31.61 (0.87)</td>
<td>30.15 (1.72)</td>
<td>25.31 (2.25)</td>
<td>23.08 (3.45)</td>
</tr>
</tbody>
</table>

Chi-square: 1.66 (p=0.65) 4.92 (p=0.18) 4.35 (p=0.23) 1.84 (p=0.61)

*Table 3.7 Mean number (and s.d.) of correct detections of target letter (maximum: 32) on each size of letter array.*

<table>
<thead>
<tr>
<th></th>
<th>1 letter</th>
<th>4 letters</th>
<th>8 letters</th>
<th>12 letters</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group 1</strong></td>
<td>0.94 (1)</td>
<td>3.0 (3.0)</td>
<td>3.73 (3.5)</td>
<td>4.90 (4.5)</td>
</tr>
<tr>
<td><strong>Group 2</strong></td>
<td>0.97 (1)</td>
<td>3.50 (3.75)</td>
<td>5.08 (5.25)</td>
<td>6.12 (5.625)</td>
</tr>
<tr>
<td><strong>Group 3</strong></td>
<td>0.97 (1)</td>
<td>3.35 (3.5)</td>
<td>4.29 (5.0)</td>
<td>5.56 (4.5)</td>
</tr>
<tr>
<td><strong>Group 4</strong></td>
<td>0.98 (1)</td>
<td>3.54 (3.5)</td>
<td>4.65 (4.5)</td>
<td>5.31 (6.0)</td>
</tr>
</tbody>
</table>

*Table 3.8 Mean (and median) values for perceptual span for each size of letter array.*
3.43 Discussion

The performance here of either of the groups of patients with hysterical symptoms is not sufficiently different from the control groups with respect to accuracy of detection or variability to support the hypothesis that their visual attention on the letter detection task is selectively impaired. The possibility that the result represents a "false negative" needs to be considered.

There appears to be no difference in scores between patients with epilepsy (group 4) and patients with pseudo-epileptic seizures (group 2). Although scores for patients with non-organic gait disorders (group 1) were consistently lower than those of patients whose lower limb weakness was attributed to neurological disease, this was not to a statistically significant extent. It is possible that this might have emerged as a significant finding had each of these comparison groups been larger.

The experiment had been designed primarily to permit statistical comparisons between a group of 25 patients with conversion symptoms and 25 patients with neurological disease. If differences across the sample in the number of correct letter detections had discriminated between these two populations, and the standard deviation of these scores had been 2.5, then a difference in letter detection scores of 2 across each block of 32 would have appeared as a significant one, assuming a power of 0.8 and a 0.05 criterion of statistical significance (Altman, 1982). The experiment shows these assumptions to have been unjustified. Thus, the standard deviation across the sample for the detection scores on the 12 figure arrays was 3.18; if there is any consistent difference in the number of correct detections per block of 32 arrays, it appears likely to be smaller than 2; and the experiment indicates that separate account should be taken of patients with seizures of either type, and patients with lower leg weakness of either type, with separate comparison of 2 pairs of subgroups within this mixed sample. (This impression is reinforced by the divergence of results in the dual task study between patients with non-organic seizures and non-organic weakness - cf. section 3.33). For a new study to be capable of detecting significant differences in score of only 1 in the hit rate between 2 groups, with similar variance in the scores to that obtained here, each group in a two group comparison would need to contain at least 160 subjects at the stated power and significance level (Altman, 1982).

Any possibility that members of group 1 were distinct from patients having seizures as a pseudoneurological symptom (group 2) as well as the 'organic' control groups would require explanation. As in the dual attention study described in section 3.3, members of group 1 were distinctly older than members of group 2. This makes it likely that, apart from the difference in the presenting pseudoneurological symptom, their disabilities were of longer duration, and therefore representative of a population with "chronic" hysteria. Such patients have shown habituation deficits in the past that those whose symptoms were of more recent
onset did not share (Meares & Horvath, 1972).

Were the present study to be extended in this way, it would be important to reduce the impact of one confounding variable that might contribute to poorer performance among group 1. This concerns the higher, albeit small, incidence of minor visual defect among its members (table 3.5).

Other explanations of a 'false negative' finding here would involve aspects of the procedure that render the experiment relatively insensitive. Although the nature of the task and the number of letter presentations used was comparable with other investigations that have been productive, some features either did not, or could reasonably be suspected not to, predispose to higher scores.

Thus, the angle subtended at the eye in this procedure was less than the 3 to 12 degrees operating in some tachistoscopic procedures where the letter array is physically closer, and permitted the arrays to stay entirely within the foveal angle of vision. A restriction of visual attention that was preferential for objects in the periphery of the visual field would therefore be less likely to have been detected by this method. This explanation has been invoked to explain differences in the literature concerning the perceptual span of clinically remitted schizophrenics, where deficits have been claimed only to approach those of acute schizophrenics when larger visual angles are used (Dobson et al, 1987; Asarnow et al, 1991). This possible explanation of the lack of positive findings here is pertinent to some of Janet's statements on hysteria. He viewed peripheral visual inattention among his patients as a special instance of the stigmata of hysterical anesthesia, going on to make observations of how "the effort of visual attention contracts in a very notable way their visual field".

Another aspect of the procedure that could conceivably compensate for lower scores is the method by which letter arrays were projected onto a computer screen. VDU screens contain a phosphor which is intended to promote retention of an after image, extending the duration of exposure of any image projected onto it to promote an illusion of continuity. However, inspection of the letter traces using an oscilloscope confirmed decay was rapid and the exposure interval quoted in the method section was consistently maintained. The possibility remains that this form of projection allows greater retention of a retinal after-image in a way that viewing a slide screen may not, predisposing to higher scores.

Inspection of reports of past clinical studies of perceptual span reveals that, although microcomputers had not been used to control presentation times and collate results, cathode ray tubes have been used as a means of presenting back-illuminated letters on transparent slides in some tachistoscopic experiments. This has been the procedure of Asarnow and MacCrimmon (1978;1981) whereas other investigators such as Strauss et al (1984) continued to use front-lit arrays. While the much lower values that Strauss quoted for the perceptual span of psychotic patients might be attributable to the acute nature of their illnesses compared to subjects in other investigations, the much lower scores quoted for the perceptual span of his normal control subjects relative to other studies has not been
An investigation of attention commented on. Yet on an 8 letter array, Strauss' data suggest a "normal" subject has a perceptual span reduced to 5 letters; Neale's data suggest a value of about 4.5. This contrasts with the figure of over 7 objects (normals) or 5 objects (schizophrenics) registered from a 10 letter array by extrapolating from Asarnow & MacCrimmon's data for detection rates. When 5 schizophrenic patients took part in pilot studies of the present apparatus, they produced values for 'span' of between 4 and 5 on the 8 and 12 letter arrays, compatible with the 'high threshold' cathode ray projections of Asarnow rather than "low threshold" studies such as Strauss or Neale. There seems little doubt that back projection leads to significantly higher values for perceptual span.

In explaining the results of the present experiment, it is therefore possible that a deficit not detected using a back-lit procedure such as the present one could manifest using a front-projection method. Hanish and Hannah's (1987) finding of positive differences between anxious and less anxious normal subjects was obtained by methodology that, although claiming to replicate Asarnow and MacCrimmon's methods, actually used front projection, strengthening this conjecture. What is particularly remarkable about their work, however, is not the claimed difference between these non-psychotic groups, but the fact that calculations of span from their data yield values of 4 to 4.75, according to anxiety level, on the 10 letter array where the differences were most significant. This suggests that the method of projection is particularly influential on measurements of perceptual span across all populations.

The many discrepancies in the experimental literature suggest that, far from representing some absolute limit on attention, figures for "perceptual span" obtained by the target letter strategy are only consistent within a given testing context and do not represent an absolute perceptual property. A further methodological anomaly that any future studies will need to take careful account of came to light as a result of the sizes of letter arrays chosen for the present study. While the range of array sizes chosen (1,4,8 and 12) seemed a logical reflection of the demands of the investigation, the literature shows that use of arrays of more than 8 letters has been very uncommon, with Asarnow and MacCrimmon's repeated use of 10 letter arrays a rare exception. There is little data therefore to support the presumption that, once perceptual span fails to increase with an increase in the size of the array, this represents an absolute ceiling that is maintained. However, as figure 3.1 indicates (reproduced from Estes & Taylor 1961) a considerable increase in calculated span is evident of nearly 2 letters as letter array size was increased from 10 to 11. The present investigation has shown not only that the calculated span for members of all four groups falls off sharply in progressing from 4 letter arrays to 8 letter arrays, but that it is consistently greater on a 12 letter array than on an 8 letter array (cf. table 3.8). This is completely consistent with Estes and Taylor's data, although they failed to explain the increment they had observed. As they too back-lit their arrays using a cathode ray tube, it is unclear whether the phenomenon is unique to this method.
Figure 3.1 Variations in perceptual span in normal subjects using the detection method for different sizes of letter array (after Estes & Taylor, 1964).
One explanation of these observations is that Estes and Taylor's calculation becomes progressively less valid with larger array sizes. It can certainly be expected to become less reliable as the proportion of letter detections that are correct will inevitably decrease as the size of the array increases. If larger arrays are included in a protocol, more presentations of that array size within a block of arrays may be needed to achieve estimates of span whose confidence intervals remain comparable to those for smaller arrays. This does not explain the tendency for the estimates of span to increase rather than decrease in larger arrays.

3.5 Conclusions.

The experiments described have permitted differences in subjects' ability to divide attention between verbal and sequential manual tasks, and in the number of letters that they can simultaneously detect, to be recorded. Possible restrictions to their sensitivity have been attributed to the choice of verbal task in the former, and the use of back projection and a narrow foveal angle in the latter. Hypotheses that attention on either task would be selectively impaired in patients having pseudoneurological symptoms had to be rejected. Although patients with lower limb weakness of non-organic origin encountered difficulty in maintaining attention for a sequential tapping task under all conditions, their performance was less evidently impaired compared with the other groups on the perceptual span task. While not excluding the possibility, the studies did not provide positive evidence that patients with pseudoneurological symptoms share a common psychological deficit compatible with theories of dissociation.
Conclusions on the place of conversion symptoms.

The historical, clinical and experimental studies of chapters 1 to 3 can each contribute to a reconsideration of the question raised in the introduction and in section 1.6. How far should "conversion" symptoms remain the basis for a distinct diagnostic syndrome? All three studies had been expected to clarify features common to these patients, with the possibility of retaining the concept of a disorder characterised by pseudoneurological symptoms, while its diagnostic criteria are refined in the light of new findings. Overall, little was found of diagnostic significance from these studies. While they indicate that pseudoneurological symptoms aren't simply misdiagnosed neurological illness, they provide few grounds for regarding them as any different in kind from other medically unexplained findings. At the same time, one of the key findings of this work has been a clinical one - that prognosis for patients with pseudoneurological symptoms is worse if they have a number of features that can in principle be elicited on routine clinical (including psychiatric) examination.

The historical study showed that hysterical conversion arose as a concept before hysteria was identified with diseases of the nervous system. Subsequently, the theories of Freud became identified with a subset of hysterical symptoms attributed to a specific psychopathology. These were the symptoms of "conversion hysteria". All the essential elements of the pathogenic models of Freud and his analytic successors, viz. the appeal to unconscious motivation, the symbolic character of symptoms, and the dynamics of secondary gain, have been progressively abandoned in official definitions of conversion disorder. This has left an anomaly in diagnostic schemes whereby the syndrome of conversion disorder is supposed to have a distinct aetiology although this is no longer the case. This raises the question of whether a diagnostic distinction should be maintained between pseudoneurological symptoms and symptoms in other systems that may resemble the consequences of disease, but which are taken to be expressions of emotional distress. Official classifications are inconsistent on this point, maintaining distinctions not only between conversion symptoms and the chronic polysymptomatic syndrome of somatisation disorder, but its forme fruste, undifferentiated somatoform disorder, for which many "conversion" patients might also qualify. At the same time, the classical symptoms of conversion disorder, but not other recurring psychosomatic symptoms, have been allied with syndromes based on amnesias, fugues and other psychogenic alterations of identity in order to subsume them as subtypes of "dissociative" disorders in the current WHO classification. Nosology in this area currently depends upon precedent and convention rather than original epidemiological research or demarcations founded in pathology. There are parallels between this situation, and that prevailing in the 18th. century when "hysterical conversion" was originally coined by John Ferriar to take care of anomalies that did not appear to fit into the prevailing diagnostic matrix before its introduction. There is clearly a need for research into the actual clustering of symptoms across the entire range of what are currently termed the somatoform and
dissociative disorders if classificatory schemes here are to be valid. In their absence, the follow-up and experimental studies described here addressed issues whose resolution would greatly assist current diagnostic discussion.

The 10 year follow-up study described in chapter 2, despite considerable methodological limitations, indicates that many patients' pseudoneurological symptoms do persist. When they do, neurological disease supervenes less frequently than has been supposed. In most such cases, some clinical indications of neurological disease had been present at assessment, even if a firm diagnosis could not be made. It may be expected that continuing refinements in neurological assessment will further reduce the risk of subsequent diagnosis of a neurological disorder that accounts for the original symptom, while leaving a substantial group of patients whose symptoms will still persist. Insofar as this study replicated the classic study of Slater and Glithero (1965) with a cohort of similar patients, it contradicted their key finding concerning the frequency with which diagnostic change was to be expected. It might be used to argue that diagnosis of a distinct conversion syndrome is in fact justified on the basis of long-term outcome. Despite Slater's interpretations, his own results show several of his patients whose symptoms and diagnoses did not change to have resembled those who were then being diagnosed by Guze as having polysymptomatic "hysteria" (the forerunner of somatisation disorder). It seemed likely from the present study that patients with persisting pseudoneurological symptoms come to resemble other patients whose long histories of unexplained physical symptoms qualify them for diagnoses of somatisation disorder. Further studies would be required to determine what proportion of chronic patients might illustrate this development over an even longer interval of time. At present, it must remain uncertain how far the natural history of persistent conversion symptoms supports the case that they represent a distinct syndrome.

The detailed assessments which were undertaken to identify factors at initial assessment associated with subsequent outcome suggested clinical suspicion of personality disorder, and success in obtaining prescribed medication from medical attendants, were common among patients whose symptoms persisted. While their previous pattern of consultation was unknown, these patients also consulted widely during the follow-up period before retrospectively reporting a possibly increasing variety of past symptoms. All these features have been noted in studies of patients with somatisation disorder. A convergence hypothesis offers a plausible explanation of how this might arise. Patients who develop any form of chronic somatisation may demonstrate early tendencies to consult and evidence of maladaptive personality traits, while their adult careers lead, through frequent consultations, towards a common polysymptomatic end state at which patients may increasingly resemble one another. Before this, one or more symptoms, including pseudoneurological symptoms, are likely to be more prominent, the choice influenced according to co-morbidity with other systemic illnesses. The resemblance of presenting 'conversion' symptoms to symptoms of past neurological illness among a majority of the study patients who had had these would
Conclusions

This hypothesis, consistent with much other literature on somatisation, might shape future research. A fuller understanding of the natural history of chronic conversion symptoms needs to encompass a wide range of psychogenic symptoms, to apply standardised symptom inventories at each assessment, to assess personality dysfunction using measures that reflect clinical concepts of personality, and to pay close attention to changes in consultation behaviour in order to clarify the relationships between these.

The third study in this thesis attempted unsuccessfully to demonstrate neuropsychological deficits that might be common to patients with pseudoneurological symptoms. Historically, patients with conversion symptoms have been grouped together from the time of Charcot and Janet because the neurological character of their symptoms implied that, although there was no evidence of structural disease, the symptoms resulted from subtle impairments of psychological functioning. Success in finding a neuropsychological deficit characteristic of patients with conversion symptoms would allow arguments of this kind to be revived. By focussing on patients' capacity to divide their attention, the experiments described in chapter 3 also had the potential to demonstrate psychological features that could provide a marker for "dissociation". Positive findings would therefore have assisted the argument that "conversion" and "dissociative" symptoms belong to the same pool. The experimental studies gave no real support for either of these contentions, suggesting any further neuropsychological studies with similar aims should use different methods to test alternative models of altered psychological processing in these patients.

Both the follow-up study and the studies on attention touch on one further facet of recent diagnostic reform. This is the description of three major sub-groups of patients with pseudoneurological symptoms, including one for "motor" symptoms and one for "seizures" or "convulsions" (APA, 1994; WHO, 1992). The cohort for the follow-up study represented an unselected sample of referred patients in which these symptoms accounted for the two largest groups of patients, indicating the distinction's face validity. However, the groups could not be distinguished in terms of their prognosis, a finding which contradicts earlier studies, and statements in DSM-IV concerning the better prognosis of motor symptoms. As a further difference from earlier follow-up studies concerned the failure in the present study of patients with seizures to dominate those whose provisional neurological diagnoses were subsequently confirmed, it is possible that this difference in prognosis may continue to diminish as initial clinical assessments grow in accuracy.

Nevertheless, differences between the performance of these two subgroups of patients did emerge in the experimental studies described in chapter 3, especially the dual attention task. While these were inconsistent within the terms of these studies (failing to conform to hypothesised differences, and varying from one experiment to the next), potentially significant demographic contrasts were evident between these two sub-groups in the present sample. It suggests that future research should respect this new diagnostic
Conclusions

subdivision when recruiting subjects with pseudoneurological symptoms to clinical and experimental studies.

C.J. Mace, December 1997
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Conclusions


Conclusions


Conclusions


Conclusions


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Conclusions


'Hysteria', 'functional' or 'psychogenic'? A survey of British neurologists' preferences

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Keywords: hysteria; classification; functional disease; conversion disorder

Summary
The diagnostic preferences of British neurologists for patients who lack a physical explanation for their symptoms were assessed by means of a postal questionnaire. Analysis of 168 completed replies showed 'functional', 'psychogenic' and 'hysteria' to be the most popular terms in use. The number of different terms a clinician would use rose in line with the volume of such patients they encountered, but was unrelated to clinician factors such as the extent of their clinical experience in psychiatry. A specific enquiry into these respondents' interpretation of the term 'functional' revealed a clear consensus as to which syndromes it should apply to, although this consensus was not shared by a comparison group of psychiatrists.

Introduction
Controversy continues to surround the diagnosis of hysteria. If the literature is to be believed, clinicians should treat terms such as 'hysteria', 'functional', and 'psychogenic' with suspicion, and instead consider alternatives such as 'abnormal illness behaviour'. Diagnostic manuals are encouraging the abandonment of 'hysteria', and adoption of alternatives such as 'somatoform disorder'. However, amid a welter of prescription and counter-prescription, a complete dearth of information persists in the literature as to how clinicians cope in practice with the need to find suitable descriptive labels for patients presenting with pseudoneurological symptoms.

As a first step towards remedying this lack, we report on a postal survey of British neurologists designed to provide three kinds of data. It set out to record which terms were most popular among neurologists for the description of cases evidently lacking a physical cause, both in discussion and in written reports. It aimed also to provide some indication as to whether any patterns of preference that emerged reflected differences between clinicians' patient loads, their previous training, or their therapeutic policies.

Finally, it tried to establish whether the evident ambiguity of the term 'functional', by which it could refer to those disorders viewed as 'psychological' or those viewed as 'physiological', was reflected by a divergence between subgroups of the sample in the way they chose to apply it themselves.

Method
The sample was chosen to include as many doctors within the UK as possible who had undergone specialist training in neurology, and who continue to undertake clinical work in the specialty. The membership list of the Association of British Neurologists was examined, and only members of consultant or senior registrar status, and believed to hold clinical posts in neurology, were invited to participate. The questionnaire they were sent was brief, in order to maximize the response, and invited responses took the form of single words, figures or forced choices in order to ensure that the quantity of information obtained from each respondent was independent of constraints of time or motivation. The questionnaire comprised 10 items over two pages, and was sent to 275 selected ABN members with a stamped envelope and a guarantee that the anonymity of replies would be respected.

The questionnaire is appended (Appendix 1). It yielded basic data concerning respondents' training, their patient load, therapeutic policy, and psychological attribution as well as their terminological preferences and interpretation of the term 'functional'. Completed replies also provided four secondary items of data for each participant: the approximate number of new cases they saw lacking neurological pathology ('suspect cases'); the total number of different diagnoses they admitted to using from among the 11 proffered ('label count'); the number of different referral destinations they cited ('resources used') and the number of conditions they were each prepared to classify as 'functional'.

Rank correlation was examined between these variables, along with the measures of specialis experience, case load, and psychological attribution. For each of the 10 diagnostic terms, the chi square statistic was used to test the hypothesis of association with respect to: use of each of the other terms; possession of any full-time psychiatri experience; neurological experience greater than the median; readiness to implicate psychological cause; and the number of different disposal destinations the reported using.

In addition, a modified questionnaire incorporating questions 9 and 10 (cf. Appendix 1) was given individually to psychiatrists attending a conference on movement disorders, to allow direct comparison of the terms they preferred in diagnosis, and the interpretation of the term 'functional'.

Results
Of 275 questionnaires originally sent to the neurologists, 206 were returned with no further prompting a response rate of 74.9%. Of these respondents, 22 felt they should not complete the form as their duties were currently non-clinical, and 7 others because they were fully retired. Nine replies were incomplete (two deliberately so), leaving 168 complete replies as the basis of this report.
Table 1. Reported use of different referral terms (n=168)

<table>
<thead>
<tr>
<th>Destination</th>
<th>Up to 1/10 'occasional'</th>
<th>Up to 1/2 'regular'</th>
<th>More than 1/2 'most'</th>
<th>None</th>
</tr>
</thead>
<tbody>
<tr>
<td>Referrer only</td>
<td>16 (9.5%)</td>
<td>45 (27%)</td>
<td>99 (59%)</td>
<td>8 (5%)</td>
</tr>
<tr>
<td>Psychiatrist</td>
<td>119 (71%)</td>
<td>24 (14%)</td>
<td>4 (2%)</td>
<td>21 (13%)</td>
</tr>
<tr>
<td>Physical therapist</td>
<td>76 (45%)</td>
<td>28 (17%)</td>
<td>2 (1%)</td>
<td>62 (37%)</td>
</tr>
<tr>
<td>Psychologist</td>
<td>75 (45%)</td>
<td>27 (16%)</td>
<td>-</td>
<td>66 (39%)</td>
</tr>
<tr>
<td>Another physician</td>
<td>85 (51%)</td>
<td>7 (4%)</td>
<td>-</td>
<td>76 (45%)</td>
</tr>
<tr>
<td>Social worker</td>
<td>72 (43%)</td>
<td>17 (10%)</td>
<td>-</td>
<td>79 (47%)</td>
</tr>
<tr>
<td>Behaviour or psychologist</td>
<td>35 (21%)</td>
<td>3 (2%)</td>
<td>-</td>
<td>130 (77%)</td>
</tr>
<tr>
<td>Other</td>
<td>18 (11%)</td>
<td>7 (4%)</td>
<td>-</td>
<td>143 (85%)</td>
</tr>
</tbody>
</table>

Over half the respondents (52%) had received their undergraduate clinical training in London. Neurological experience ranged between 2 and 38 years, with a mean of 16 years. Twenty-seven of the respondents had experience of full-time psychiatric work: its median duration was 9 months. Forty respondents indicated that a majority of their patients had problems of a particular type, with headache (10 replies); paediatric neurology (6), epilepsy (5); neuromuscular (5) and movement disorders (4) being the most common.

The estimated number of new patients seen in a typical month ranged from 4 to 350. The latter figure was a flagrant outlier: the median was 85 new cases per month. Estimates of the percentage apparently lacking a neurological basis ranged from nil (n=7) to 80% (n=2) with a median of 20%. The derived estimate of the number of patients seen having symptoms, but for whom a negative diagnosis was considered, had a median value of 13 cases per month.

The percentage of cases where psychological factors were thought to be of etiological significance ranged from 1% to 90%, median 30%. A few applicants chose to give more than one figure, eg for 'primary' and 'secondary' contributions: in such cases the larger figure was used in analysis, the question being worded to include contributions of all degrees. This rating was closely correlated to respondents' estimate of the proportion of cases they saw that lacked neurological pathology. (Spearman correlation coefficient 0.58, P<0.001.) Conversely, no association was found with past psychiatric experience.

Table 1 summarizes the relative popularity of colleagues when patients who lacked evidence of neurological disease were referred elsewhere. Although only 7 (4%) claimed never to make other referrals, 60% reported sending at least a majority of these cases straight back to the referrer. Of the referrals made on an occasional basis ('up to 1 in 10'), psychiatrists remained the most popular single destination. Where patients were referred more regularly to others (ie the options of 'up to 1 in 4', or greater, were chosen), the relative importance of psychologists and physical therapists grew.

The reported use of the 11 terms supplied in the diagnosis of non-neurological cases is summarized in Table 2.

Only two respondents denied using any of these terms, and the mean number of terms selected in question 9 was 5.4. This figure was positively correlated with those extrapolated for the number of patients they saw requiring such diagnoses, (Spearman r=0.19, P=0.006) and the number of referral destinations that they used (Spearman r=0.23, P=0.002). Among the individual terms, only 'hypochondriasis' showed a correlation with recency of training, being more popular among the more experienced (corrected chi squared 8.15; P=0.004).

Table 2 shows that four distinct groups of terms emerge, according to their relative popularity, and whether use was 'informal' or 'formal'. Terms in group A are distinctly more popular; group B are less popular, and as likely to be used informally as formally; group C are relatively unpopular but likely to be used formally; group D is distinctly unpopular. The mean number of other terms likely to be chosen by users of a given diagnosis increased steadily down Table 2, the exception being 'abnormal illness behaviour' whose use was associated with that of relatively fewer other terms. Individually, use of each of the terms in group B was associated with both 'hysteria' and 'functional' from group A at a 5%
Table 3. Syndromes classified as 'functional' by 168 neurologists

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pseudoseizures</td>
<td>114</td>
<td>68%</td>
</tr>
<tr>
<td>Anxiety neurosis</td>
<td>104</td>
<td>62%</td>
</tr>
<tr>
<td>Munchausen's syndrome</td>
<td>103</td>
<td>61%</td>
</tr>
<tr>
<td>Paranoid schizophrenia</td>
<td>12</td>
<td>7%</td>
</tr>
<tr>
<td>Post-ictal psychosis</td>
<td>6</td>
<td>4%</td>
</tr>
<tr>
<td>Gilles de la Tourette's syndrome</td>
<td>5</td>
<td>4%</td>
</tr>
<tr>
<td>Primary generalized epilepsy</td>
<td>4</td>
<td>2%</td>
</tr>
<tr>
<td>Spasmodic torticollis</td>
<td>4</td>
<td>2%</td>
</tr>
<tr>
<td>Idiopathic torsion dystonia</td>
<td>3</td>
<td>2%</td>
</tr>
<tr>
<td>Idiopathic parkinsonism</td>
<td>1</td>
<td>1%</td>
</tr>
<tr>
<td>None of the above</td>
<td>28</td>
<td>17%</td>
</tr>
</tbody>
</table>

Significance level, whereas none of group B were linked with use of 'psychogenic'. Use of 'hysteria' and 'functional' were also strongly associated with one another (corrected $\chi^2$ 8.15, $P=0.004$).

Table 3 details the frequency with which each of the named disorders provided were subsumed under 'functional'.

Here, a total of 140 respondents ticked one or more options, including 22 who were not currently using the term themselves. Remarkably, no fewer than 136 respondents had ticked between 1 and 4 items, and in every single case their choices were confined to a quartet comprising anxiety neurosis, Munchausen's syndrome, pseudoseizures, and paranoid schizophrenia. Their distribution is summarized in Figure 1, which demonstrates that 'functional' was effectively reserved for three of these diagnoses, ie anxiety neurosis, Munchausen's syndrome and pseudoseizures by members of this sample.

This situation contrasted with the psychiatrists' replies, where although 27 of the 38 replies received also made their selection within the same four diagnoses preferred by those neurologists using four or fewer terms, a distinct distribution was evident in which 'paranoid schizophrenia' was admitted to be 'functional' by half of these psychiatric respondents (Figure 2).

Discussion

The high response rate indicated that the questionnaire could elicit unambiguous responses on a contentious subject. Despite the target population's fearsome reputation for meticulousness, fewer than 30% of the respondents had added any marginalia, and no one question received more attention than others on this score. Spontaneous comments were generally, if not exclusively, enthusiastic, and 40% of the sample wished to take up an offer to receive a report of the study's outcome.

Three terms remain the most popular among neurologists when diagnosing patients who lack a neurological basis for their symptoms: 'hysteria', 'functional' and 'psychogenic'. Despite the criticism that has been aimed at each of them in the past, these terms at least appear to remain as popular among recently trained neurologists as among older colleagues. Users were very likely to be using more than one of these terms, although this exploratory survey could not determine the extent to which respondents may use them interchangeably in individual cases. Nevertheless, 'psychogenic' appeared to be favoured by doctors who were less willing to use more pejorative terms (group B of Table 2), while users of 'hysteria' and 'functional' may be more judgmental in their outlook.

The use of a relatively large number of diagnostic terms by an individual clinician was found to be associated with them having a larger caseload of patients who were believed to lack a neurological cause for their symptoms, but not to a greater disposition to suspect psychogenesis per se, nor to their having had previous special clinical experience in psychiatry. And it was the number of diagnostic terms a doctor admitted to using that showed at least a weak correlation with the number of different agencies they made use of when referring such patients for help elsewhere. Thus although the breadth of diagnostic vocabulary seemed to reflect necessity rather than temperament, a more restricted
vocabulary seemed to accompany a relatively nihilistic attitude towards treatment.

Although the survey did not examine the selectivity with which terms other than 'functional' were being applied, there was little evidence that the neurologists' diagnostic habits had been affected by reforms advocated by psychiatrists in recent years. For instance, users of the term 'abnormal illness behaviour', recommended as an alternative to all such terms as 'hysteria', 'functional' or 'psychogenic', may be relatively more selective than users of other less common terms, but were found to use a wide range of alternative labels also. The users of 'conversion', which has been recommended for use independently of 'hysteria', were continuing to use 'hysteria' in all but 4 instances. Users of 'somatization' were distinctly uncommon, despite a report that the chronic poly-symptomatic syndrome of 'somatization disorder' should be diagnosable in 30% of female medical inpatients with complaints of over 5 years' duration. These are findings of which future would-be reformers might beware.

The strategy of providing a fixed list of alternative terms from which selections were invited had proved particularly satisfactory on two counts. Firstly, in all instances where possible additions to the list were volunteered, these remained unique to one contributor. (Suggestions of this sort were usually 'informal' in the extreme.) Secondly, only two terms provoked any objections to their inclusion: the contradictory 'supratentorial', and the ambiguous 'functional'. Nevertheless, it is evident from Table 2 that both terms remain highly popular in practice, and 'functional' exceptionally so.

The interpretation of 'functional' received special attention in this survey, and the remarkable consensus that reserved it for the three syndromes of anxiety neurosis, Munchausen's syndrome, and pseudoseizures has been described. However, as a clear opinion was evident among our sample of psychiatrists that the term should extend to paranoid schizophrenia, its interpretation by either group is evidently distinct.

As this sample of psychiatrists shared a common interest in neurological aspects of psychiatry, it is likely that even greater range of use would be found among other groups of doctors. It would appear that the apparent neutrality of 'functional' belies much potential confusion, and its diagnostic use in particular should be discouraged.

While the primary aim of the study was to report on current terminology in a diagnostic no-man's-land, several figures were also extrapolated from the retrospective estimates contained in the participants' responses that should not be ignored were they to be confirmed by more rigorous methods. For instance the replies of the 168 neurologists suggested that between them they were seeing over 36,000 new patients a year whose symptoms lacked a neurologica basis. This would indicate the presence of a vast patient group that would seem deserving both of further research in their own right, and for guideline on their management to become a high priority in the training of neurologists. Very low proportions of these patients were referred by the participating neurologists to other specialists for further diagnosis (the vast majority saying they sent fewer than one in 10 such patients for psychiatric assessment, with an even lower proportion of patients reaching another physician, see Table 1). It would be reasonable to expect that, however arrived at, the neurologists' opinions in these cases were likely to be of lastin influence. This treatment contrasted with the view of the 38 psychiatrists who, when asked 'what percentage of patients who have neurological symptoms for which no physical cause is apparent would benefit from psychiatric assessment?' gave a median reply of '70%' (90% confidence interval, 50%-90%). Although it is likely that at least some of these patients will obtain psychiatric assessment by other routes in practice, there seems little doubt that neurologists would be justified in referring a greater proportion of such cases to psychiatric colleagues than that which they currently admit to.

Acknowledgment: The authors would like to thank all the doctors whose replies have contributed to this report, as well as the Raymond Way Fund for financial support.

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(Accepted 31 October 1990)
Appendix 1: The Questionnaire

1. At which medical school did you train? (school/city/country.)

2. How many years have you spent working in full-time clinical neurology?

3. Have you also ever done any full-time clinical jobs in psychiatry? (If so, for how long did you work in psychiatry?)

4. Approximately how many new cases (all sources) do you currently see in a typical month?

5. Do a majority of these tend to be of a particular clinical type? (If so, please specify it briefly here.)

6. In what percentage of all your new patients would you judge psychological factors to be of aetiological significance?

7. In what percentage of the new patients you see do the presenting symptoms have no apparent neurological basis?

8. For the cases you have seen in the last year where the patient's symptoms had no apparent neurological basis, please circle the reply that indicates roughly what proportion you then referred: (A grid was provided here. It listed the following frequencies horizontally: None; Up to 1 in 10; Up to 1 in 4; Up to 1 in 2; Up to 3 in 4; Up to 9 in 10; All. It listed the following destinations vertically: (a) directly back to the referrer (eg GP); (b) for a psychiatric opinion; (c) for another physician's opinion; (d) to a behaviour therapist or psychotherapist; (e) to a clinical psychologist; (f) to a physiotherapist; (g) to a social worker; (h) to anyone else (please specify).)

9. A list of terms follows that are sometimes used in the diagnosis of patients lacking a neurological basis for their symptoms. Please indicate with a tick which, if any, you have used yourself in this way in the last year both informally (ie in ward or clinic discussions) and formally (ie in summary letters or reports).

<table>
<thead>
<tr>
<th>Term</th>
<th>Informal use?</th>
<th>Formal use?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal illness behaviour</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conversion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Functional</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypochondriasis (or hypochondria/ hypochondriacal)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hysteria (or hysterical/ hysteric/ hysterical)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malingering (or malingerer)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurotic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychogenic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychosomatic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Somatoform (or somatization)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supratentorial</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

10. Lastly, please place a tick against any of the following disorders you would classify as 'functional':

   Anxiety neurosis
   Gilles de la Tourette syndrome
   Idiopathic Parkinsonism
   Idiopathic torsion dystonia
   Multiple sclerosis
   Munchausen's syndrome
   Paranoid schizophrenia
   Post-ictal psychosis
   Primary generalized epilepsy
   Pseudoseizures
   Spasmodic torticollis
Appendix 2: Patients used in studies of attention.

### Group 1: Lower limb weakness without neurological disease

<table>
<thead>
<tr>
<th>Study no.</th>
<th>Age</th>
<th>Sex</th>
<th>Dual task study?</th>
<th>Perceptual span study?</th>
<th>Diagnosis from medical notes.</th>
</tr>
</thead>
<tbody>
<tr>
<td>306</td>
<td>57</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Weak legs. No neurological diagnosis.</td>
</tr>
<tr>
<td>307</td>
<td>49</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Conversion hysteria</td>
</tr>
<tr>
<td>308</td>
<td>50</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Weakness of legs. History of pseudoseizures.</td>
</tr>
<tr>
<td>315</td>
<td>69</td>
<td>m</td>
<td>y</td>
<td>y</td>
<td>Functional gait disorder (?cerebellar)</td>
</tr>
<tr>
<td>316</td>
<td>21</td>
<td>m</td>
<td>y</td>
<td>y</td>
<td>Functional gait disorder</td>
</tr>
<tr>
<td>318</td>
<td>24</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Hysterical paralysis</td>
</tr>
<tr>
<td>319</td>
<td>41</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Functional unilateral leg weakness</td>
</tr>
<tr>
<td>326</td>
<td>43</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Hysterical monoplegia</td>
</tr>
<tr>
<td>327</td>
<td>37</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Functional gait disorder</td>
</tr>
<tr>
<td>331</td>
<td>28</td>
<td>f</td>
<td>y</td>
<td>n</td>
<td>Hysterical weakness</td>
</tr>
<tr>
<td>337</td>
<td>48</td>
<td>f</td>
<td>y</td>
<td>n</td>
<td>Functional gait disorder</td>
</tr>
<tr>
<td>312</td>
<td>66</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Weakness of no known cause</td>
</tr>
<tr>
<td>320</td>
<td>58</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Hysterical paralysis</td>
</tr>
<tr>
<td>330</td>
<td>52</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Functional gait disorder</td>
</tr>
<tr>
<td>332</td>
<td>58</td>
<td>m</td>
<td>n</td>
<td>y</td>
<td>Non-organic gait disorder</td>
</tr>
</tbody>
</table>

### Group 2: Pseudoepileptic seizures

<table>
<thead>
<tr>
<th>Study no.</th>
<th>Age</th>
<th>Sex</th>
<th>Dual task study?</th>
<th>Perceptual span study?</th>
<th>Diagnosis from medical notes.</th>
</tr>
</thead>
<tbody>
<tr>
<td>311</td>
<td>40</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Pseudoseizures</td>
</tr>
<tr>
<td>317</td>
<td>23</td>
<td>m</td>
<td>y</td>
<td>y</td>
<td>Pseudoseizures (falling attacks)</td>
</tr>
<tr>
<td>321</td>
<td>44</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Falling attacks/depression</td>
</tr>
<tr>
<td>323</td>
<td>37</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Pseudoseizures</td>
</tr>
<tr>
<td>324</td>
<td>37</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Pseudoseizures</td>
</tr>
<tr>
<td>325</td>
<td>21</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Hysterical seizures</td>
</tr>
<tr>
<td>328</td>
<td>28</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Hysterical seizures</td>
</tr>
<tr>
<td>329</td>
<td>25</td>
<td>m</td>
<td>y</td>
<td>y</td>
<td>Dissociative attacks with jerking</td>
</tr>
<tr>
<td>334</td>
<td>23</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Pseudoseizures</td>
</tr>
<tr>
<td>335</td>
<td>28</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Pseudoseizures and anxiety neurosis</td>
</tr>
<tr>
<td>336</td>
<td>43</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Pseudoseizures</td>
</tr>
<tr>
<td>338</td>
<td>46</td>
<td>m</td>
<td>y</td>
<td>y</td>
<td>Falling attacks/pseudoseizures</td>
</tr>
</tbody>
</table>

### Group 3: Lower limb weakness due to neurological disease

<table>
<thead>
<tr>
<th>Study no.</th>
<th>Age</th>
<th>Sex</th>
<th>Dual task study?</th>
<th>Perceptual span study?</th>
<th>Diagnosis from medical notes.</th>
</tr>
</thead>
<tbody>
<tr>
<td>208</td>
<td>49</td>
<td>m</td>
<td>y</td>
<td>y</td>
<td>with sciatica</td>
</tr>
<tr>
<td>210</td>
<td>29</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td>213</td>
<td>60</td>
<td>m</td>
<td>y</td>
<td>y</td>
<td>Lumbar spondylosis</td>
</tr>
<tr>
<td>216</td>
<td>53</td>
<td>m</td>
<td>y</td>
<td>y</td>
<td>Lumbar spondylosis</td>
</tr>
<tr>
<td>219</td>
<td>39</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td>221</td>
<td>25</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Familial spastic paraparesis</td>
</tr>
<tr>
<td>229</td>
<td>22</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Lumbar spondylosis</td>
</tr>
<tr>
<td>230</td>
<td>37</td>
<td>f</td>
<td>y</td>
<td>y</td>
<td>Cerebellar syndrome with paraparesis</td>
</tr>
<tr>
<td>231</td>
<td>50</td>
<td>f</td>
<td>y</td>
<td>n</td>
<td>HTLV1 infection</td>
</tr>
<tr>
<td>234</td>
<td>37</td>
<td>f</td>
<td>y</td>
<td>n</td>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td>203</td>
<td>19</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Generalised epilepsy</td>
</tr>
<tr>
<td>204</td>
<td>53</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td>209</td>
<td>55</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Tropical spastic paraparesis</td>
</tr>
<tr>
<td>220</td>
<td>67</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Cervical spondylosis</td>
</tr>
<tr>
<td>232</td>
<td>66</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Cervical spondylosis</td>
</tr>
</tbody>
</table>
### Group 4: Epilepsy with continuing seizures.

<table>
<thead>
<tr>
<th>Study no.</th>
<th>Age</th>
<th>Sex (f/m/n)</th>
<th>Dual task study?</th>
<th>Perceptual span study?</th>
<th>Diagnosis from case notes.</th>
</tr>
</thead>
<tbody>
<tr>
<td>212</td>
<td>26</td>
<td>m</td>
<td>n</td>
<td>y</td>
<td>Primary generalised epilepsy (myoclonic)</td>
</tr>
<tr>
<td>211</td>
<td>40</td>
<td>m</td>
<td>n</td>
<td>y</td>
<td>Temporal lobe epilepsy</td>
</tr>
<tr>
<td>215</td>
<td>25</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Primary generalised epilepsy</td>
</tr>
<tr>
<td>217</td>
<td>24</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Focal (?)frontal) seizures</td>
</tr>
<tr>
<td>218</td>
<td>19</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Complex partial (?)frontal) seizures</td>
</tr>
<tr>
<td>222</td>
<td>24</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Generalised epilepsy</td>
</tr>
<tr>
<td>223</td>
<td>25</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Generalised epilepsy</td>
</tr>
<tr>
<td>225</td>
<td>21</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Partial seizures (?)frontal)</td>
</tr>
<tr>
<td>227</td>
<td>32</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Complex partial seizures</td>
</tr>
<tr>
<td>228</td>
<td>23</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Partial epilepsy</td>
</tr>
<tr>
<td>233</td>
<td>18</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Myoclonic epilepsy</td>
</tr>
<tr>
<td>235</td>
<td>25</td>
<td>f</td>
<td>n</td>
<td>y</td>
<td>Temporal lobe epilepsy</td>
</tr>
</tbody>
</table>
Appendix 3: BBC Basic programme for administration of tapping test.

5 REM ***************************************************************
6 REM * PROGRAM: TAPPING
7 REM * TAPPING PROVIDES AN AUTOMATED METHOD OF RECORDING
8 REM * PERFORMANCE ON A SEQUENTIAL MANUAL MOTOR TASK THAT CAN BE
9 REM * GIVEN ALONGSIDE OTHER NON MANUAL TASKS. ITS CHIEF USE IS TO BE
10 REM * BE IN THE EXPERIMENTAL INVESTIGATION OF THE DIVISION OF
11 REM * ATTENTION. THE PROGRAM ENABLES AN EXPERIMENTER HAVING THE
12 REM * AID OF ONSCREEN FEEDBACK, TO INTRODUCE A SUBJECT TO THE CORRECT
13 REM * USE OF AN ATTACHED FOUR DIGIT TELEGRAPH KEYPAD BY EITHER
14 REM * HAND AND TO ADMINISTER PRACTICE SESSIONS WHOSE NUMBER AND
15 REM * FIXED DURATION CAN BE PRESET AT THE OPENING OF THE PROGRAM
16 REM * LISTING. FEEDBACK TO THE SUBJECT IS PROVIDED BY A CODE OF THREE
17 REM * BUZZERS CORRESPONDING TO THE THREE TYPES OF COMMON ERROR
18 REM * (DELAY, HITTING THE WRONG BUTTON, HITTING TWO OR MORE
19 REM * BUTTONS) AND THESE CAN BE INTRODUCED AT THE START OF A SESSION
20 REM * FROM A SPECIAL MENU. AFTER THE INTRODUCTION, ANY NUMBER
21 REM * SEQUENCES IS COUNTED AND RECORDED, CAN BE ADMINISTERED,
22 REM * ALLOWING FOR TAPPING PERFORMANCE TO BE ASSESSED UNDER A
23 REM * VARIETY OF CONDITIONS. BOTH HANDS ARE ASSESSED IN TURN FOR
24 REM * EACH ONE. THE RESULTS, STORED IN AN ARRAY, CAN BE FED TO
25 REM * PRINTER AND SCREEN. IT IS ANTICIPATED THAT ANOTHER MACHINE
26 REM * WILL BE USED FOR DISK STORAGE OF TAPPING SCORES TOGETHER WITH
27 REM * SCORES FROM ANY AUXILIARY TASKS.
28 REM ***************************************************************

100 REM *** INITIALISATION SECTION ***
110 LE = 1500 : REM LENGTH OF EACH TIMED BLOCK
120 SE = 8 : REM NO. OF INTRODUCTORY SEQUENCES FOR EACH HAND IN
130 DEMONSTRATION.
140 TB = 0 : REM NO. OF DEMO BLOCKS (NO BUZZERS) FOR EACH HAND.
150 CO = 9 : REM NO. OF CONDITIONS TO BE ADMINISTERED DURING TEST ROUTINE
200 NO80 = 0 : REM INPUT WHEN NO BUTTONS ARE Pressed
210 NO81 = 128 : REM INPUT WHEN FIRST BUTTON IS Pressed
220 NO82 = 64 : REM INPUT WHEN SECOND BUTTON IS Pressed
230 NO83 = 32 : REM INPUT WHEN THIRD BUTTON IS Pressed
240 NO84 = 16 : REM INPUT WHEN FOURTH BUTTON IS Pressed
300 DIM DEMOB (BB,2) : REM ARRAY FOR DEMO RESULTS FROM BLOCKS WITH
310 DIM DEMO (TB,2) : REM ARRAY FOR DEMO RESULTS FROM BLOCKS WITH
320 DIM TEST (CO,2) : REM ARRAY FOR TEST RESULTS FROM EXPERIMENTAL
330 DIM BLOCKS.

500 REM ***************************************************************
501 REM * MASTER ROUTINE
502 REM * THE MASTER ROUTINE CALLS THE CHIEF BLOCKS OF THE PROGRAM IN
503 REM * TURN(PARA, DEMO, TEST AND RESULT)DURING THE ADMINISTRATION
504 REM * OF A COMPLETE RUN.
505 REM ***************************************************************
510 CLS
520 GOSUB 700 : REM SUBROUTINE PARA
530 GOSUB 1000 : REM SUBROUTINE DEMO
540 GOSUB 3000 : REM SUBROUTINE TEST
550 GOSUB 4000 : REM SUBROUTINE RESULT
560 END
* SUBROUTINE PARA
* PARA RECORDS SUBJECT PARTICULARS FOR THE RESULT PRINTOUT,
* AND DISPLAYS THE CURRENT SETTINGS FOR THE PARAMETERS
* INITIALISED IN PROGRAM LINES 100-300.

INPUT \texttt{TAB(5,5) "NAME", NAMES}
INPUT \texttt{TAB(5,7) "DATE", DATES}
INPUT \texttt{TAB(5,9) "TEST NO.", TNO}
CLS : \texttt{PRINT TAB(15,4) "PARAMETERS"}
PRINT \texttt{TAB(5,7) "BLOCK LENGTH = "LE "CS."
PRINT \texttt{"DEMO: SEQUENCE NO. "SE}
PRINT \texttt{"BUZZ BLOCKS = "BB}
PRINT \texttt{"QUIET BLOCKS="TH}
PRINT \texttt{TAB(1,180 "TEST CONDITIONS "CO}
IF \texttt{GET \$=" "THEN 830
RETURN

* SUBROUTINE DEMO
* DEMO DEMONSTRATES THE THREE BUZZERS USED TO PROVIDE
* FEEDBACK ON ERRORS MADE IN USING THE KEYPAD: INTRODUCES
* EACH HAND IN TURN TO CORRECT TAPPING SEQUENCE; RUNS TRIAL
* BLOCKS FOR EACH HAND WITH AND WITHOUT BUZZERS; AND
* PRODUCES A SCREEN PRINTOUT SO THAT TIMED PERFORMANCE
* UNDER EITHER CONDITION CAN BE COMPARED
* UNDER EITHER CONDITION CAN BE COMPARED

VDU 23;8202;0;0;0;
TAB (5,8) "FOR WAITING' BUZZER PRESS 1"
TAB (7,11) "FOR 'WRONG' BUZZER PRESS 2"
TAB (6,14) "FOR 'DOUBLE' BUZZER PRESS 3"
TO PROCEED PRESS"*
A$=GETs
IF A$="1 "GOSUB 1120
IF A$="2" GOSUB 1130
IF A$="3"GOSUB 1140
IF A$="*"GOTO 1150
GOTO1070
SOUND 1,-15,49,10 : RETURN
SOUND 1,-15,1,10 : RETURN
SOUND 1,-15,29,10 : RETURN
CLS: \texttt{PRINT TAB(11,7) "USING THE KEYPAD"}
PRINT \texttt{TAB(11,10) "1. FOR RIGHT HAND"
PRINT \texttt{TAB(5,16) "PRESS SPACE BAR TO PROCEED"
IFGET$=""THEN1200
NI-NOBI
N2=NOB2
N3=NOB3
N4=NOB4
QE62=0
FOR X=1 TO SE
PRINT \texttt{TAB(10,22) X}
GOSUB 1500
NEXT X
CLS: \texttt{PRINT TAB(13,9) "2. FOR LEFT HAND"
PRINT \texttt{TAB(5,16) "PRESS SPACE BAR TO PROCEED"}
IF GETS$=" "THEN 1330
NI=NOB4
N2=NOB3
N3=NOB2
SUBROUTINE 'SEQUENCE' FOR HAND

\[
\begin{align*}
1360 & \text{N4=NOBI} \\
1365 & \text{?&FE62=0} \\
1370 & \text{FOR X=1 TO SE} \\
1375 & \text{PRINT TAB (10,22) X} \\
1380 & \text{GOSUB 1500} \\
1390 & \text{NEXT X} \\
1400 & \text{GOTO 2000} \\
1500 & \text{REM *** SUBROUTINE 'SEQUENCE' FOR HAND *****}
\end{align*}
\]

\[
\begin{align*}
1505 & \text{TAR=N4} \\
1510 & \text{GOSUB 1900} \\
1520 & \text{TAR=NI} \\
1530 & \text{BS="FIRST"} \\
1540 & \text{TIME=0} \\
1550 & \text{GOSUB 1800} \\
1560 & \text{CLS} \\
1570 & \text{BS="SECOND"} \\
1580 & \text{TIME=0} \\
1590 & \text{GOSUB 1900} \\
1600 & \text{CLS} \\
1610 & \text{TAR=N2} \\
1620 & \text{TIME=0} \\
1630 & \text{GOSUB 1800} \\
1640 & \text{CLS} \\
1650 & \text{BS="THIRD"} \\
1660 & \text{GOSUB 1900} \\
1670 & \text{CLS} \\
1680 & \text{TAR=N3} \\
1690 & \text{TIME=0} \\
1700 & \text{GOSUB 1800} \\
1710 & \text{CLS} \\
1720 & \text{BS="FOURTH"} \\
1730 & \text{TIME=0} \\
1740 & \text{GOSUB 1900} \\
1750 & \text{CLS} \\
1760 & \text{TAR=N4} \\
1770 & \text{TIME=0} \\
1780 & \text{GOSUB 1800} \\
1790 & \text{RETURN}
\end{align*}
\]

SUBROUTINE 'BUTTON' FOR REGISTERING BUTTON PRESSED

\[
\begin{align*}
1800 & \text{REM *** SUBROUTINE 'BUTTON' FOR REGISTERING BUTTON PRESSED} \\
1810 & \text{M=?&FE60} \\
1820 & \text{IF M=NOBO OR M=TAR OR M=48 OR M=144 THEN 1855 ELSE 1830 : REM KEYPAD} \\
1830 & \text{BY SOLUTION.} \\
1840 & \text{PRINT TAB (5,24) "WRONG." : BS "WANTED"} \\
1850 & \text{TIME=0 : GOTO 1810} \\
1855 & \text{IF M=TAR THEN 1885 ELSE 1860} \\
1860 & \text{IF TIME <300 THEN 1810 ELSE 1865} \\
1865 & \text{SOUND 1,-15,1,1} \\
1870 & \text{PRINT TAB (5,24) "WAITING FOR ": BS} \\
1880 & \text{TIME=0 GOTO 1810} \\
1885 & \text{RETURN}
\end{align*}
\]

SUBROUTINE 'GAP' FOR REGISTERING SPACE BETWEEN BUTTONS

\[
\begin{align*}
1900 & \text{REM *** SUBROUTINE 'GAP' FOR REGISTERING SPACE BETWEEN BUTTONS} \\
1910 & \text{M=?&FE60} \\
1920 & \text{IF M>TAR AND M<NOBO THEN 1930 ELSE 1960} \\
1930 & \text{SOUND 1,-15,29,1} \\
1940 & \text{PRINT TAB (5,24) "2 BUTTONS PRESSED"} \\
1950 & \text{TIME=0:GOTO 1910} \\
1960 & \text{IF M=NOBO THEN 1990} \\
1965 & \text{IF TIME<150 THEN 1910 ELSE 1970} \\
1970 & \text{SOUND 1,-15,49,10} \\
1975 & \text{PRINT TAB (5,24) "WAITING FOR A GAP"} \\
1980 & \text{TIME=0 : GOTO 1910} \\
1985 & \text{RETURN}
\end{align*}
\]

RETURN
Appendix 3

2000 REM *** THE FOLLOWING SECTION PROVIDES THE PRESET NUMBER OF
2001 REM *** TIMED BLOCKS WITH BUZZER FEEDBACK.
2010 CLS: PRINT TAB (8,10) "TIMED BLOCKS (BUZZERS)"
2020 PRINT TAB (8,15) "PRESS SPACE BAR TO PROCEED"
2030 IF GET$=" "THEN 2040
2040 FOR X=1 TO BB
2050 CLS: PRINT TAB (9,10) "GET RIGHT HAND READY (";X;")"
2060 PRINT TAB (8,20) "PRESS SPACE BAR TO PROCEED"
2070 N1=NOBI: N2=NOB2: N3=NOB3: N4=NOB4
2080 IF GET$=" "THEN 2090
2090 GOSUB 2200
2100 DEMOB (X,1)=D
2110 CLS: PRINT TAB (10,10) "GET LEFT HAND READY (";X;")"
2120 PRINT TAB (8,20) "PRESS SPACE BAR TO PROCEED"
2130 N1=NOB4: N2=NOB3: N3=NOB2: N4=NOB1
2140 IF GET$=" "THEN 2150
2150 GOSUB 2200
2160 DEMOB (X,2)=D
2170 NEXT X
2180 GOTO 2700
2190
gostab...
Appendix 3

2670 RETURN
2690 REM *** THE NO BUZZ SECTION IS OMITTED IN THIS VERSION
2700 REM *** THE FOLLOWING SECTION PROVIDES THE PRESET NUMBER
2701 REM *** OF TIMED BLOCKS WITHOUT BUZZER FEEDBACK.
2710 GOTO2880
2720 CLS : PRINT TAB (8,10) "TIMED BLOCKS (NO BUZZERS)"
2730 PRINT TAB (8,15) "PRESS SPACE BAR TO PROCEED."
2735 IF GET$=" "THEN 2740
2740 FOR X=1 TO TB
2750 CLS: PRINT TAB (9,10) "GET RIGHT HAND READY (";X;")"
2760 PRINT TAB (8,20) "PRESS SPACE BAR TO PROCEED"
2770 NI=NOBIp: N2=NOB2: N3=NOB3: N4=NOB4
2780 IF GET$=" "THEN 2790
2790 GOSUB 3200
2800 DEMO(X,1)=D
2810 CLS: PRINT TAB (10,10) "GET LEFT HAND READY (";X;")"
2820 PRINT TAB(8,20) "PRESS SPACE BAR TO PROCEED"
2830 NI=NOB4: N2=NOB3: N3=NOB2: N4=NOBI
2840 IF GET$=" "THEN 2850
2850 GOSUB 3200
2860 DEMO (X,2)=D
2870 NEXT X
2880 PRINT TAB (12,20) "OVER"
2890 CLS : PRINT TAB (12,5) "DEMO SCORES"
2900 PRINT TAB (8,8) "RIGHT": PRINT TAB (28,8) "LEFT"
2910 PRINT TAB (0,10) "DEMO:
2920 NEXT X
2930 IF X=1 TO BB: PRINT TAB (3) DEMOB (X,1);" ";DEMOB (X,2):PRINT
2940 NEXTX
2950 GOTO 2980 : REM ONLY ONE TYPE OF DEMO IN THIS VERSION
2960 PRINT "DEMO"
2970 FOR X=1 TO TB:PRINT TAB (4) DEMO (X,1);" ";DEMO (X,2):PRINT
2980 NEXT X
2990 PRINT:PRINT:PRINT TAB (8) "PRESS SPACE BAR FOR TEST"
3000 IF GET$=" "THEN RETURN
3001 REM * SUBROUTINE 'TEST'
3002 REM * 'TEST' ALLOWS THE SEQUENTIAL TAPPING RATE TO BE RECORDED
3003 REM * FOR THE PRESET NUMBER OF EXPERIMENTAL BLOCKS.
3004 REM *** SUBROUTINE FOR COUNTING NUMBER OF SEQUENCES
3005 REM * COMPLETED DURING AN EXPERIMENTAL BLOCK
3006 REM **********************************************************
3007 REM * SUBROUTINE TEST'
3008 REM * 'TEST ALLOWS THE SEQUENTIAL TAPPING RATE TO BE RECORDED
3009 REM * FOR THE PRESET NUMBER OF EXPERIMENTAL BLOCKS.3010 REM ************************************************************
3010 CLS: PRINT TAB(10,10) "EXPERIMENTAL BLOCKS"
3020 PRINT TAB(8,15) "PRESS SPACE BAR TO PROCEED"
3030 IF GET$=" "THEN 3040
3040 FOR X=1 TO CO
3050 CLS: PRINT TAB(9,10) "GET RIGHT HAND READY (";X;")"
3060 PRINT TAB(8,20) "PRESS SPACE BAR TO PROCEED"
3070 NI=NOBI: N2=NOB2: N3=NOB3: N4=NOB4
3080 IF GET$=" "THEN 3090
3090 GOSUB 3200
3100 TEST(X,1)=D
3110 CLS: PRINT TAB (10,10) "GET LEFT HAND READY (";X;")"
3120 PRINT TAB (8,20) "PRESS SPACE BAR TO PROCEED"
3130 NI=NOB4: N2=NOB3: N3=NOB2: N4=NOBI
3140 IF GET$=" "THEN3150
3150 GO5UB3200
3160 TEST (X,2)=D
3170 NEXT X
3180 GOTO3700
3190 REM *** SUBROUTINE FOR COUNTING NUMBER OF SEQUENCES
3200 REM * COMPLETED DURING AN EXPERIMENTAL BLOCK
3205 REM CLS: PRINT TAB (6,13) "TIMED BLOCK IN PROGRESS"
3240 IF TIME<LE THEN GOTO 3220 ELSE 3250
3250 D=C-1
3260 RETURN
3300 REM *** SUBROUTINE FOR REGISTERING PRESSES IN CORRECT SEQUENCE.
3310 TAR=N4
3320 G05UB3600
3330 TAR=N1 : B$="FIRST"
3340 GOSUB 3500
3350 GOSUB 3600
3360 TAR=N2 : B$="SECOND"
3370 GOSUB35000
3380 GOSUB 3600
3390 TAR=N3 : B$="THIRD"
3400 GOSUB 3500
3410 GOSUB 3600
3420 TAR=N4 : B$="FOURTH"
3430 GOSUB 3500
3440 RETURN
3500 REM *** SUBROUTINE FOR REGISTERING BUTTON PRESSED
3510 M=?&FE60
3520 IF M=NOBO OR M=TAR ThEN 3560 ELSE 3550
3550 GOTO 3510
3560 IF M=TAR THEN 3570 ELSE 3510
3570 RETURN
3600 REM *** SUBROUTINE FOR REGISTERING GAP BETWEEN PRESSES
3610 M=?&FE60
3620 IF M=TAR AND M~NOBO THEN 3650 ELSE 3660
3650 GOTO 3610
3660 IF M=NOBO THEN 3670 ELSE 3610
3670 RETURN
3700 CLS: PRINT TAB (10,8) "TEST OVER"
3710 PRINT TAB (8,18) "PRESS SPACE BAR FOR RESULTS"
3720 IF GET$=" "THEN RETURN
4000 REM
4001 REM * SUBROUTINE 'RESULT'
4002 REM * RESULT PROVIDES A SCREEN AND OPTIONAL PRINTER PRINTOUT OF
4003 REM * THE CONTENTS OF THE RESULT ARRAY OBTAINED DURING THE
4004 REM * EXPERIMENTAL SESSION.
4005 REM ******************************************************
4006 4005 REM
4020 CLS: PRINT TAB (11,5) "RESULT MENU"
4030 PRINT TAB (5,10) "FOR SCREEN PRINTOUT ONLY: 1"
4040 PRINT TAB (5,15) "IF PRINTER ALSO CONNECTED: 2"
4050 PRINT TAB (5,20) "TO FINISH SESSION: *
4060 4060 4060
4070 IF K$="1"GOSUB 4120
4071 IF K$="2"GOSUB 4100
4075 IF K$="*"RETURN
4080 GOTO 4020
4100 VDU2
4120 CLS: PRINT:PRINT TAB (5) NAMES$,DATE$,TND
4130 PRINT:PRINT:PRINT TAB (5) "BLOCK RIGHT LEFT": PRINT
4140 FORX=ITOCO
4150 PRINT TAB (2) X," ";TEST(X,1);" ";TEST(X,2)
4155 PRINT
4160 NEXTX
4165 VDU3
4170 PRINT:PRINT:PRINT TAB(7) "PRESS BAR TO RETURN TO END"
4180 IFGET$=" "THEN 4190
4190 RETURN
203
Appendix 4: GW Basic programme for administration of perceptual span test.

```
5 KEY OFF
10 REM***************************************************************************
11 REM* PROGRAM: SPAN
12 REM***************************************************************************
13 REM* SPAN IS A PROGRAM FOR THE AUTOMATED ADMINISTRATION OF A TEST
14 REM* OF PERCEPTUAL SPAN. IT INTRODUCES A SUBJECT TO THE TEST AND THE
15 REM* TYPES OF RESPONSE REQUIRED; ADMINISTERS A TRIAL BLOCK OF 32
16 REM* LETTER ARRAYS AND THEN A FULL TEST OF 128 LETTER ARRAYS;
17 REM* COMPUTES PERCEPTUAL SPAN FOR ARRAYS OF 1, 4, 8 AND 12 LETTERS.
18 REM* AND MAKES THE RESULT AVAILABLE FOR STORAGE TO DISKFILE.
19 REM* AND FOR SCREEN AND PRINTER OUTPUT. THE PROGRAM IS COMPILED IN
20 REM* GW BASIC, AND IS STRUCTURED IN MODULES TOGETHER WITH STATEMENTS
21 REM* FOR Initialise VALUES AND FUNCTIONS. THE MODULES ARE: (1) FLASH,
22 REM* FOR TRIAL PRESENTATION OF LETTER ARRAYS, RECORDING OF SUBJECT'S
23 REM* RESPONSE, AND CORRECTION OF ERRORS IN RESPONSE; (2) BLOCK, FOR
24 REM* PRESENTATION OF LETTER SETS IN SERIES OF 32 WITH A BREAK BETWEEN,
25 REM* STORAGE OF ACCUMULATING RESULTS AND COMPUTATION OF HIT RATE
26 REM* AND COMPUTATION OF HIT RATE AND SPAN SIZE FOR EACH OF THE 4
27 REM* SET SIZES (3) TEST, STORING THE VALUES FOR HIT RATE AND SPAN EACH TIME TO THE
28 REM* CHIEF ARRAY. "RESULT"; (4) TRIAL, WHICH USES BLOCK ONCE TO GIVE A
29 REM* PRACTICE RUN OF 32 LETTER ARRAYS, THE SCORE FROM WHICH IS
30 REM* INDEPENDENTLY STORED WITHIN "RESULT", (5) DEMO, PRODUCES AN
31 REM* INTRODUCTORY INTERACTIVE FAMILIARISATION ROUTINE; (6) SUBJECT,
32 REM* WHICH RECORDS PERSONAL DATA FOR FILE LABELLING; (7) FINAL, WHICH
33 REM* PRODUCES A FINAL SCORE FROM "RESULT" ARRAY, AND ARRANGES FOR
34 REM* DISK STORAGE AND DISPLAY. THEY ARE LINKED BY THE MASTER PROGRAM
35 REM* FOUND FROM LINE 500.
45 SCREEN 1
50 SCREEN 0: WIDTH 40
60 GAP= 167
70 ITIME=4
500 REM***************************************************************************
501 REM* MASTER SECTION
502 REM* THE "MASTER" SECTION OF THE PROGRAM CALLS SUBROUTINES IN TURN
503 REM* THAT RECORD SUBJECT'S DETAILS FOR FILE LABELLING, DEMONSTRATE
504 REM* PROCEDURES, RUN "TRIAL" AND "TEST" AND THEN HANDLE THE
505 REM* RESULTS.
506 REM***************************************************************************
560 GOSUB 700
565 REM*** FOR REGISTRATION OF PATIENT DETAILS BY EXPERIMENTER***
570 GOSUB 1000
575 REM*** CALLS SUBROUTINE "DEMO" ***
580 GOSUB 2500
590 REM*** CALLS SUBROUTINE "TRIAL" **
600 GOSUB 3000
610 REM*** CALLS SUBROUTINE "TEST"*
620 GOSUB 5000
630 REM*** CALLS SUBROUTINE "FINAL" *
640 END
500 REM***************************************************************************
700 REM* SUBROUTINE: SUBJECT
701 REM* "SUBJECT" RECORDS BASIC DATA CONCERNING THE SUBJECT FOR
703 REM* INCORPORATION INTO DISK DATA FILES AND PAPER PRINTOUTS.
704 REM***************************************************************************
705 LOCATE 2,8 : INPUT "CHECK MOUSE";MOUSE$
710 LOCATE 4,15 : INPUT "NAME";S$
715 L$=LEFT$(S$(,4)
720 LOCATE 6,12 : INPUT "INITIAL";I$
730 LOCATE 8,16 : INPUT "SEX";X$
```
LOCATE 10,7: INPUT "STUDY NUMBER"; Y$
LOCATE 12,14: INPUT "GROUP"; G$
LOCATE 14,10: INPUT "TRIAL NO." ; T$
LOCATE 16,3: INPUT "DATE AS DD.MM.YY"; D$
LOCATE 18,6: INPUT "TIME AS HH.MM"; T$
SFN$="S"+I$+L$+G$+T$
DFN$="D"+I$+L$+G$+T$
REM *** THE FILENAME TO BE USED FOR THE SUMMARY FILE ***
REM *** THE FILENAME TO BE USED FOR THE DATA FILE ***
CLS: LOCATE 13,2: PRINT "PRESS SPACE BAR WHEN READY TO PROCEED"
N$=INKEY$: IF N$=" " THEN
IF N$="" THEN 820 GOTO 820 RETURN
REM *** RETURNS TO MASTER SEQUENCE ***
CLN1: REM **************************************************************************
1001 REM * SUBROUTINE: DEMO
1002 REM * DEMO INTRODUCES SUBJECTS IN TURN TO THE FORMAT OF THE LETTER
1003 REM * ARRAYS, THE REQUIRED KINDS OF RESPONSE, AND TO THE METHOD OF
1004 REM * CORRECTING RESPONSES AFTER THEY ARE MADE. IT USES A GROUP
1005 REM * OF 4 STANDARD SLIDES FOR DEMONSTRATIONS OF PROCEDURES AT
1006 REM * EACH STAGE. PROMPTS ARE GIVEN AS APPROPRIATE. NO RESULTS ARE
1007 REM * RECORDED FROM THE OPERATION OF "DEMO".
1008 REM **************************************************************************
1020 DIM DAT$(32,6)
1030 DATA "AC","L P",L,B
1040 DATA "  7 7 Z 7 7 ",Z,A
1050 DATA "F Z Q"," S T P M","N F"," Q R Y ",Z,D
1060 DATA "XM","CTM ",FB","L",L,C
1070 FOR X=1 TO 4
1080 FOR Y=1 TO 6
1090 READ DAT$(X,Y)
1100 NEXT Y
1110 NEXT X
1120 CLS: NOW=TIMER
1125 COLOR 14
1130 LOCATE 11,8: PRINT"IN THE TEST THAT FOLLOWS"
1140 LOCATE 13,8: PRINT"YOU WILL SEE MAY SETS"
1150 LOCATE 15,8: PRINT"OF LETTERS."
1160 IF TIMER> (NOW+ITIME) THEN 1165 ELSE 1160
1165 CLS: COLOR 9
1170 LOCATE 11,8: PRINT"THESE WILL DIFFER IN SIZE."
1180 LOCATE 13,8: PRINT"BUT THEY WILL ALWAYS HAVE"
1190 LOCATE 15,8: PRINT"EITHER ONE 'L' OR ONE 'Z'."
1200 IF TIMER> (NOW+2*ITIME) THEN 1210 ELSE 1200
1210 CLS: COLOR 12
1220 LOCATE 13,8: PRINT"HERE ARE SOME EXAMPLES..."
1230 IF TIMER> (NOW+2.5*ITIME) THEN 1240 ELSE 1230
1240 FTIME=3000
1250 CLS
1260 FOR X=1 TO 4
1270 GOSUB 4330
1280 REM *** REFERENCES PROGRAM TO LETTER PRESENTATION IN "FLASH"
1290 NEXT X
1300 SCREEN 0: WIDTH 40
1305 CLS: COLOR 13: LOCATE 11,8: NOW = TIMER: PRINT"THE SETS OF LETTERS WILL"
1306 LOCATE 13,8: PRINT"ALWAYS APPEAR INSIDE THIS";
1307 LOCATE 15,8: PRINT"AREA OF THE SCREEN..."
1308 IF TIMER> (NOW+ITIME) THEN 1310 ELSE 1308
1310 SCREEN 1: NOW = TIMER: DRAW "BM128,72"
1312 DRAW "R64D56L64U56": DRAW "PL0";
1315 IF TIMER> (NOW+1TIME) THEN 1317 ELSE 1315
1317 SCREEN0: NOW = TIMER: KEYOFF :LOCATE,.0
1320 COLOR 14
1330 LOCATE 10,8: PRINT"FROM NOW ON, EACH TIME YOU"
Appendix 4

1340 LOCATE 12,8 : PRINT" SEE A SET OF LETTERS, YOU"
1350 LOCATE 14,8 : PRINT" SHOULD PRESS A BUTTON TO"
1360 LOCATE 16,8 : PRINT" SHOW IF YOU SAW 'L' OR 'Z'."
1370 IF TIMER> (NOW+1.5*TIME) THEN 1380 ELSE 1370
1380 CLS : COLOR 9
1390 LOCATE 12,9 : PRINT "TRY THIS NOW USING THE"
1400 LOCATE 14,9 : PRINT" BOX IN FRONT OF YOU."
1410 IF TIMER> (NOW+2.5*ITIME) THEN 1415 ELSE 1410
1415 CLS
1420 FOR X= 1 TO 4
1425 CLS : NOW=TIMER
1430 GOSUB4330
1440 T$=INKEY$ : IF T$="L" OR T$="Z" THEN 1460 : IF T$="" THEN 1450
1450 IF TIMER> (NOW+6) THEN 1500 ELSE 1440
1460 IF ASC(T$)=ASC(DAT$(X,5))THEN 1550 ELSE 1470
1470 SOUND 150,12; CLS: SCREEN 0: COLOR 20
1480 CLS :LOCATE 12,17 : PRINT "WRONG!": LOCATE 14,15 : PRINT "TRY AGAIN."
1490 FORM 1 TO 4000: NEXT M: GOTO 1425
1500 CLS: SCREEN 0 : COLOR 20
1510 LOCATE 13,4: PRINT "DON'T FORGET TO PRESS 'L' OR 'Z'!"
1520 NOW=TIMER
1530 GOTO 1440
1540
1550 NEXT X
1560 CLS: SCREEN 0:COLOR 14 : NOW=TIMER
1600 REM *** SEQUENCE FOR INTRODUCING ACTUAL RUNNING SPEED
1610 LOCATE 11,9 : PRINT"IN THE TEST THAT FOLLOWS,"
1620 LOCATE 13,8 : PRINT"THE LETTERS WILL COME AND"
1630 LOCATE 15,8 : PRINT" GO VERY QUICKLY."
1640 IF TIMER> (NOW+3.5*ITIME) THEN 1500 ELSE 1630
1650 CLS : COLOR 13
1660 LOCATE 11,11: PRINT"YOU WILL NEED TO KEEP YOUR"
1670 LOCATE 13,8 : PRINT"EYES ON THE MIDDLE OF THE"
1680 LOCATE 15,8 : PRINT" SCREEN..."
1690 IF TIMER> (NOW+2*ITIME) THEN 1700 ELSE 1690
1700 CLS : COLOR 3 : LOCATE 10,18: PRINT" ... AND TO"
1710 LOCATE 12,8 : PRINT"REMEMBER TO PRESS ONE OF"
1720 LOCATE 14,8 : PRINT"THE BUTTONS, EVEN IF YOU"
1730 LOCATE 16,10: PRINT"HAVE TO MAKE A GUESS."
1740 IF TIMER> (NOW+3.5*ITIME) THEN 1750 ELSE 1740
1750 CLS : COLOR 14: LOCATE 13,8 : PRINT "HERE ARE SOME TO TRY..."
1760 FOR M=1 TO 6000: NEXT M
1770 CLS: FTIME=GAP
1780 FOR X= 1 TO 4
1790 GOSUB 4200
1800 T$ = INKEY$ : IF T$="L" OR T$="Z" THEN 1850 : IF T$="" THEN 1810
1810 IF TIMER > (NOW + 9) THEN 1820 ELSE 1800
1820 SCREEN 0 : COLOR 20 : LOCATE 13,4 : PRINT"DON'T FORGET TO PRESS 'L' OR"
1830 NOW=TIMER; GOTO 1800
1840 REM *** LOOP FOR REMINDER WITH DELAYED RESPONSE. ERRORS IGNORED.
1850 IF X <= 3 THEN 1800 ELSE 1890
1860 SCREEN 0 : COLOR 2 : NOW=TIMER
1870 CLS : LOCATE 13,8 : PRINT"NEXT ONE-follows...
1880 IF TIMER> (NOW+2)THEN 1890 ELSE 1880
1890 NEXT X
1900 CLS: SCREEN 0 : COLOR 9 : NOW=TIMER
1910 LOCATE 11,10: PRINT" DURING THE TEST, EACH"
1920 LOCATE 13,10: PRINT"RESPONSE YOU MAKE WILL"
1930 LOCATE 15,10: PRINT"BE SHOWN LIKE THIS..."
1940 IF TIMER> (NOW+1TIME) THEN 1950 ELSE 1940
1950 CLS: COLOR 2 : LOCATE 13,10: PRINT"YOU HAVE PRESSED: L"  
1960 IF TIMER> (NOW+2ITIME) THEN 1970 ELSE 1960
1970 CLS : COLOR 12 : LOCATE 11,7 : PRINT" TRY THIS OUT BY PRESSING"
1980 LOCATE 13,9 : PRINT"A BUTTON AFTER EACH OF"
Appendix 4

1990 LOCATE 15,9: PRINT" THESE EXAMPLES..."
1995 IF TIMER>(NOW+3*ITIME) THEN 2000 ELSE 1990
2000 FOR X = 1 TO 4
2010 GOSUB 4000
2020 NOW=TIMER: IF X <=3 THEN 2030 ELSE 2050
2030 CLS: COLOR 2: LOCATE 13,10: PRINT "NEXT ONE FOLLOWS..."
2040 IF TIMER>(NOW+2) THEN 2050 ELSE 2040
2050 NEXT X
2060 CLS: COLOR 9: NOW=TIMER: LOCATE 10,10: PRINT"WHILE YOU SEE THE"
2070 LOCATE 12,10: PRINT" GREEN MESSAGE, IT"
2080 LOCATE 14,10: PRINT" IS POSSIBLE TO CHANGE"
2090 LOCATE 16,10: PRINT" THE LETTER YOU CHOSE."
2100 IF TIMER>(NOW+1.5*ITIME) THEN 2110 ELSE 2100
2110 CLS: COLOR 14: LOCATE 10,10: PRINT" TO MAKE A CHANGE,"
2120 LOCATE 12,10: PRINT" PRESS THE BUTTON '\"'"
2130 LOCATE 14,10: PRINT" AND YOU SHOULD SEE"
2140 LOCATE 16,10: PRINT" THIS MESSAGE"
2150 IF TIMER>(NOW+3*ITIME) THEN 2160 ELSE 2150
2160 CLS: COLOR 2: LOCATE 12,5: PRINT" YOUR RESPONSE HAS BEEN CANCELLED."
2170 LOCATE 14,5: PRINT" PLEASE PRESS 'L' OR 'Z' AGAIN NOW."
2180 IF TIMER>(NOW+4*ITIME) THEN 2200 ELSE 2180
2200 CLS: COLOR 12: LOCATE 10,9: PRINT" NOW TRY CHANGING YOUR"
2210 LOCATE 12,10: PRINT" RESPONSES USING THE"
2220 LOCATE 14,11: PRINT" EXAMPLES YOU HAVE"
2230 LOCATE 16,12: PRINT" JUST TRIED..."
2240 IF TIMER>(NOW+5.5*ITIME) THEN 2300 ELSE 2240
2300 FOR X=1 TO 4
2310 GOSUB 4100
2320 IF X<=3 THEN 2330 ELSE 2350
2330 NOW=TIMER: LOCATE 13,10: PRINT "NEXT ONE FOLLOWS..."
2340 IF TIMER>(NOW+2) THEN 2350 ELSE 2340
2350 NEXT X
2360 CLS: NOW=TIMER: COLOR 9: LOCATE 10,4: PRINT" THAT IS THE END OF THE INTRO."
2365 LOCATE 12,4: PRINT" IF YOU REMAIN UNSURE OF THE WAY TO"
2370 LOCATE 14,3: PRINT" USE THE BOX, PRESS ** NOW TO SEE IT"
2375 LOCATE 16,17: PRINT" AGAIN."
2380 N$=INKEY$: IF N$="" THEN 2388: IF N$="" THEN 2384
2384 IF TIMER>(NOW+5) THEN 2385 ELSE 2380
2385 GOTO 2390
2386 REM *** CONTINUES PROGRAM IF NO REQUEST FOR REPEAT INTRO. ***
2388 CLS: GOTO 1130
2389 REM *** REPEATS INTRODUCTORY SEQUENCE IF REQUESTED ***
2390 CLS: NOW=TIMER: COLOR 3
2400 LOCATE 10,8: PRINT" YOU ARE NOW READY TO START."
2410 LOCATE 12,9: PRINT" THE TEST COMES IN 5 SHORT"
2420 LOCATE 14,8: PRINT" SESSIONS, WITH ABOUT 30 SETS"
2430 LOCATE 16,9: PRINT" OF LETTERS IN EACH ONE."
2440 IF TIMER>(NOW+1.5*ITIME) THEN 2450 ELSE 2440
2450 RETURN
2460 REM *** RETURNS TO MASTER ROUTINE
2500 REM
2501 REM SUBROUTINE: TRIAL
2502 REM * "TRIAL" CALLS UP BLOCK ONCE IN ORDER TO OBTAIN A BASELINE RECORD
2503 REM * OF THE SUBJECT'S RESPONSE TO A PRACTICE BLOCK OF 32 LETTER SETS. IT
2504 REM * IS NOT ANNOUNCED AS SUCH, BUT THE SCORES IT YIELDS ARE TREATED
2505 REM * SEPARATELY IN THE COURSE OF THE PROGRAM.
2506 REM
2560 REM
2600 CLS
2610 NOW=TIMER
2620 LOCATE 12,6: COLOR 4: PRINT" GET READY FOR THE FIRST SERIES"
2640 IF TIMER>(NOW+3) THEN 2650 ELSE 2640
2650 LOCATE 15,10: PRINT" COMING UP NOW...
2660 IF TIMER>(NOW+5) THEN 2670 ELSE 2660

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2670 GOSUB 3500
2680 RESULT(0, 1, 1)=HIT1
2690 RESULT(0, 1, 2)=FNSPAN1 (HIT1)
2700 RESULT(0, 2, 1)=HIT4
2710 RESULT(0, 2, 2)=FNSPAN4 (HIT4)
2720 RESULT(0, 3, 1)=HIT8
2730 RESULT(0, 3, 2)=FNSPAN8 (HIT8)
2740 RESULT(0, 4, 1)=HIT12
2750 RESULT(0, 4, 2)=FNSPAN12 (HIT 12)
2760 RETURN
2770 REM *** RETURNS TO MASTER ROUTINE
3000 REM ************************************************************************
3001 REM * SUBROUTINE: TEST
3002 REM * "TEST" CALL UP "BLOCK" 4 TIMES IN SUCCESSION IN ORDER TO ADMINISTER
3003 REM * SLIDE SETS FOR MEASUREMENT OF PERCEPTUAL SPAN. A WAITING
3004 REM * PERIOD PRECEDES EACH, THEN SUBJECTS CALLED TO RESUME THE TEST.
3005 REM * HIT RATES AND SPANS FOR EACH BLOCK OF 32 ARE STORED ON THE
3006 REM * ARRAY "RESULT" FOR FUTURE USE AND STORAGE.
3007 REM ************************************************************************
3008 FOR Z=1 TO 4
3009 NEXT Z
3010 GOSUB 3100
3070 NEXT Z
3080 CLS : LOCATE 12, 15 : PRINT 'REM *** NOT YETI
3090 RETURN: '*** RETURNS TO MASTER PROGRAM ***
3100 CLS
3110 NOW=TIMER
3115 LOCATE 11, 11: PRINT "THAT COMPLETES BLOCK NO. "Z","
3120 LOCATE 15, 10 : PRINT "PLEASE TAKE A 1 MINUTE REST."
3130 IF TIMER> (NOW+50) THEN 3140 ELSE 3130
3140 CLS : LOCATE 12, 10 : PRINT "PLEASE BE READY IN 10 SECONDS"
3150 SOUND 440,9
3160 IF TIMER> (NOW+55) THEN 3170 ELSE 3160
3170 CLS : LOCATE 11, 10 : PRINT "PLEASE BE READY IN 5 SECONDS"
3180 IF TIMER> (NOW+59) THEN 3190 ELSE 3180
3190 LOCATE 15, 10 : PRINT "NEXT SET FOLLOWS..."
3210 IF TIMER> (NOW+60) THEN 3220 ELSE 3210
3220 GOSUB 3500
3230 REM *** REFERS TO BLOCK *
3240 RESULT(Z, 1, 1)=HIT1
3250 RESULT(Z, 1, 2)=FNSPAN1 (HIT1)
3260 RESULT(Z, 2, 1)=HIT4
3270 RESULT(Z, 2, 2)=FNSPAN4 (HIT4)
3280 RESULT(Z, 3, 1)=HIT8
3290 RESULT(Z, 3, 2)=FNSPAN8 (HIT8)
3300 RESULT(Z, 4, 1)=HIT12
3310 RESULT(Z, 4, 2)=FNSPAN12 (HIT12)
3320 RETURN
3330 REM *** RETURNS TO 3060***
3500 REM ************************************************************************
3501 REM * SUBROUTINE: BLOCK
3502 REM * BLOCK READS DATA STATEMENTS IN BATCHES OF 32 INTO ARRAY DATA,
3503 REM * CALLS "FLASH" TO CONDUCT BRIEF PRESENTATIONS OF THE LETTER SETS
3504 REM * THEY CONTAIN, PRODUCES A SUMMARY SCORE FOR THE OVERALL HIT RATE
3505 REM * FOR SETS HAVING 1,4,8 AND 12 LETTERS, AND CALCULATES THE PERCEPTUAL
3506 REM * SPAN IN EACH CASE. "BLOCK" IS ACCESSED FROM BOTH "TRIAL" AND "TEST".
3507 REM ************************************************************************
3520 HIT1=0
3530 HIT1=0
3540 HIT8=0
3550 HIT12=0
3560 DEF FNSPAN1 (HIT1)=((2*HIT1/8)-1)
3570 DEF FNSPAN4 (HIT4)=((2*HIT4/8)-1)*4
3580 DEF FNSPAN8 (HIT8)=((2*HIT8/8)-1)*8

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DEF FNSPANI2(HIT12)=((2*HIT12/8)-1)*12
FOR X=1 TO 32
FOR Y=0 TO 6
READ DAT$(X,Y)
NEXT Y
NEXT X
FOR X=1 TO 32
GOSUB 4000
IF ASC(DAT$(X,6))=65 THEN HIT1 = HIT1 + HIT
IF ASC(DAT$(X,6))=66 THEN HIT4 = HIT4 + HIT
IF ASC(DAT$(X,6))=67 THEN HIT8 = HIT8 + HIT
IF ASC(DAT$(X,6))=68 THEN HIT12 = HIT12 + HIT
IF X<=31 THEN 3730 ELSE 3800
NOW=TIMER
LOCATE 13,10 : PRINT "NEXT SET FOLLOWS..."
IF TIMER>(NOW + 1)THEN 3760 ELSE 3750
NEXT X
REM *** SPAN IS CALCULATED BY CALLING INITIAL FUNCTIONS LATER
RETURN
REM SUBROUTINE: FLASH
GOSUB 4190
GOSUB 4500
GOSUB 4800
RETURN
REM *** RETURNS TO BLOCK ***
GOSUB 4200
CLS
NOW=TIMER
COLOR 2
LOCATE 8,15 : PRINT CHR$(218) : LOCATE 8,26 : PRINT CHR$(191)
LOCATE 18,15 : PRINT CHR$(192) : LOCATE 18,26 : PRINT CHR$(217)
LOCATE 13,20 : PRINT"S"
IF TIMER>(NOW + 1)THEN 4280 ELSE 4270
NEXT T=I TO FTIME
LOCATE 10,17 : PRINT"2"
LOCATE 12,17 : PRINT"1"
LOCATE 14,17 : PRINT"0"
LOCATE 16,17 : PRINT"
COLOR 7
LOCATE 10,17 : PRINT DAT$(X,1)
LOCATE 12,17 : PRINT DAT$(X,2)
LOCATE 14,17 : PRINT DAT$(X,3)
LOCATE 16,17 : PRINT DAT$(X,4)
FOR T=1 TO FTIME
LOCATE 10,17 : PRINT"
LOCATE 12,17 : PRINT"
LOCATE 14,17 : PRINT"
LOCATE 16,17 : PRINT"
COLOR 15
LOCATE 10,17 : PRINT DAT$(X,1)
LOCATE 12,17 : PRINT DAT$(X,2)
LOCATE 14,17 : PRINT DAT$(X,3)
LOCATE 16,17 : PRINT DAT$(X,4)
FOR T=1 TO FTIME
LOCATE 10,17 : PRINT"
LOCATE 12,17 : PRINT"
LOCATE 14,17 : PRINT"
LOCATE 16,17 : PRINT"
COLOR 7
RETURN
REM *** RETURNS DURING TEST TO 4100 ****
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4447 REM *** NECESSARY TO PREVENT CARRY OVER OF 'L' OR 'Z' PRESSED EARLY ***
4450 N$=INKEY$: IF N$="" THEN 4450
4460 IF N$="L" OR N$="Z" THEN 4470 ELSE 4450
4470 RETURN
4500 REM *** SUBROUTINE "ERROR" STARTS HERE ***
4510 NOW=TIMER
4520 SCREEN 0
4530 COLOR 2
4540 CLS: LOCATE 13,10: PRINT *YOU HAVE PRESSED: "N$*
4550 E$=INKEY$: IF E$="" THEN 4560: IF E$ ="" THEN 4560
4560 IF TIMER> (NOW+3) THEN 4570 ELSE 4550
4570 RETURN
4600 REM *** ROUTINE FOR CHANGING A RESPONSE BEGINS HERE ***
4605 CLS
4610 LOCATE 12,5: PRINT "YOUR RESPONSE HAS BEEN CANCELLED."
4620 LOCATE 14,5: PRINT "PLEASE PRESS 'L' OR 'Z' AGAIN NOW"*
4640 N$ = INNEY$: IF N$ = ""THEN 4640
4650 IF N$="L" OR N$="Z" THEN 4660 ELSE 4640
4660 NOW=(TIMER-1)
4665 CLS
4670 GOTO 4540
4800 REM *** SUBROUTINE "HIT" STARTS HERE ***
4810 CLS
4820 LET HIT=0
4830 IF ASC(N$) = ASC(DAT$(X,5)) THEN HIT = 1
4840 RETURN
4850 REM *** RETURNS TO 4130
5000 REM ******************************************************
5001 REM * SUBROUTINE: FINAL
5002 REM * FINAL USES COMPLETED ARRAY "RESULT" TO SUMMARISE THE RESULTS
5003 REM * FROM TRIAL AND TEST, PRINT THEM ON SCREEN OR PRINTER, AND STORE
5004 REM * TO A DISK FILE.
5005 REM ******************************************************
5040 CLS
5050 DEF FNMEAN(K,L,M,N)=(K+L+M+N)/4
5060 SPANA = FNMEAN(RESULT(1,1,2),RESULT(2,1,2),RESULT(3,1,2),RESULT(4,1,2))
5065 HITA = FNMEAN(RESULT(1,1,1),RESULT(2,1,1),RESULT(3,1,1),RESULT(4,1,1))
5070 SPANB = FNMEAN(RESULT(1,2,2),RESULT(2,2,2),RESULT(3,2,2),RESULT(4,2,2))
5075 HITB = FNMEAN(RESULT(1,2,1),RESULT(2,2,1),RESULT(3,2,1),RESULT(4,2,1))
5080 SPANC=FNMEAN(RESULT(1,3,2),RESULT(2,3,2),RESULT(3,3,2),RESULT(4,3,2))
5085 HITC = FNMEAN(RESULT(1,3,1),RESULT(2,3,1),RESULT(3,3,1),RESULT(4,3,1))
5090 SPAND=FNMEAN(RESULT(1,4,2),RESULT(2,4,2),RESULT(3,4,2),RESULT(4,4,2))
5095 HITD = FNMEAN(RESULT(1,4,1),RESULT(2,4,1),RESULT(3,4,1),RESULT(4,4,1))
5100 CLS
5105 NOW=TIMER: LOCATE 13,12: PRINT "THANK - YOU"
5110 IF TIMER=(NOW+10) THEN 5115 ELSE 5110
5115 CLS: COLOR 14: LOCATE 5,15: PRINT "RESULT MENU".
5120 LOCATE 9,3: PRINT "FOR SCREEN DISPLAY OF HITS PRESS:": LOCATE 9,37: PRINT "1*"
5125 LOCATE 12,2: PRINT "FOR SCREEN DISPLAY OF SPANS PRESS:": LOCATE 12,37: PRINT "2*"
5130 LOCATE 15,7: PRINT "FOR PRINT OUT OF HITS PRESS:": LOCATE 15,37:PRINT "3*"
5135 LOCATE 18,6: PRINT "FOR PRINT OUT OF SPANS PRESS:": LOCATE 18,37:PRINT "4*"
5140 LOCATE 21,6: PRINT "FOR SUBJECT DISK FILES PRESS:": LOCATE 21,37:PRINT "5*"
5145 LOCATE 24,6: PRINT "TO MAKE TAP DISK FILES PRESS:":LOCATE 24,37:PRINT "6*"
5150 N$ = INKEY$: IF N$ = "" THEN 5150
5150 IF N$ = "1" THEN GOSUB 5200
5160 IF N$ = "2" THEN GOSUB 5300
5162 IF N$ = "3" THEN GOSUB 5400
5163 IF N$ = "4" THEN GOSUB 5500
5164 IF N$ = "5" THEN GOSUB 5600
5165 IF N$ = "6" THEN GOSUB 5700
5170 CLS: LOCATE 13,1: INPUT; "PRESS ANY KEY TO RETURN TO RESULT MENU", M$
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5180 RETURN

5181 REM *** RETURNS TO MASTER PROGRAM

5200 REM *** SUBROUTINE FOR PRINTING HIT RESULTS TO SCREEN

5210 CLS

5220 LOCATE 10, 5; PRINT; RESULT(0, 1, 1); RESULT(1, 1, 1); RESULT(2, 1, 1); RESULT(3, 1, 1);
RESULT(4, 1, 1); " HITS (1) = " HITA

5230 LOCATE 13, 5; PRINT; RESULT(0, 2, 1); RESULT(1, 2, 1); RESULT(2, 2, 1); RESULT(3, 2, 1);
RESULT(4, 2, 1); " HITS (4) = " HITB

5240 LOCATE 16, 5; PRINT; RESULT(0, 3, 1); RESULT(1, 3, 1); RESULT(2, 3, 1); RESULT(3, 3, 1);
RESULT(4, 3, 1); " HITS (8) = " HITC

5250 LOCATE 19, 5; PRINT; RESULT(0, 4, 1); RESULT(1, 4, 1); RESULT(2, 4, 1); RESULT(3, 4, 1);
RESULT(4, 4, 1); " HITS(12) = " HITD

5260 LOCATE 22, 5; PRINT "PRESS SPACE BAR TO CONTINUE"

5270 N$ = INKEY$; IF N$="" THEN 5270

5275 IF N$ = " " THEN 5280 ELSE 5270

5280 RETURN

5300 REM *** SUBROUTINE FOR PRINTING SPAN RESULTS TO SCREEN

5310 CLS

5320 LOCATE 10, 5; PRINT RESULT(0, 1, 2); RESULT(1, 1, 2); RESULT(2, 1, 2); RESULT(3, 1, 2);
RESULT(4, 1, 2); " SPAN (1) = " SPANA

5330 LOCATE 13, 5; PRINT RESULT(0, 2, 2); RESULT(1, 2, 2); RESULT(2, 2, 2); RESULT(3, 2, 2);
RESULT(4, 2, 2); " SPAN (4) = " SPANB

5340 LOCATE 16, 5; PRINT RESULT(0, 3, 2); RESULT(1, 3, 2); RESULT(2, 3, 2); RESULT(3, 3, 2);
RESULT(4, 3, 2); " SPAN (8) = " SPANC

5350 LOCATE 19, 5; PRINT RESULT(0, 4, 2); RESULT(1, 4, 2); RESULT(2, 4, 2); RESULT(3, 4, 2);
RESULT(4, 4, 2); " SPAN(12) = " SPAND

5360 LOCATE 22, 5; PRINT "PRESS SPACE BAR TO CONTINUE"

5370 N$ = INKEY$; IF N$="" THEN 5370

5375 IF N$ = " " THEN 5380 ELSE 5370

5380 RETURN

5400 REM *** SUBROUTINE FOR PRINTING HIT RESULTS TO PRINTER ***

5410 LPRINT; " NAME: " 1$; " DATE: " DA$; " TRIAL: " Y$; " G$; " T$;
SUMMARY OF HIT SCORES ON SPAN TEST

5412 LPRINT

5415 LPRINT

5420 LPRINT 
RESULT(0, 1, 1); RESULT(1, 1, 1); RESULT(2, 1, 1); RESULT(3, 1, 1); " HITS (1) = " HITA

5425 LPRINT

5430 LPRINT
RESULT(0, 2, 1); RESULT(1, 2, 1); RESULT(2, 2, 1); RESULT(3, 2, 1); " HITS (4) = " HITB

5435 LPRINT

5440 LPRINT
RESULT(0, 3, 1); RESULT(1, 3, 1); RESULT(2, 3, 1); RESULT(3, 3, 1); " HITS (8) = " HITC

5445 LPRINT

5450 LPRINT
RESULT(0, 4, 1); RESULT(1, 4, 1); RESULT(2, 4, 1); RESULT(3, 4, 1); " HITS(12) = " HITD

5455 LPRINT

5460 RETURN

5465 REM *** RETURNS TO RESULT OPTIONS SEQUENCE ***

5500 REM *** SUBROUTINE FOR PRINTING SPAN RESULTS TO PRINTER ***

5510 LPRINT; " NAME: " 1$; " DATE: " DA$; " TRIAL: " Y$; " G$; " T$;
SUMMARY OF SPAN SCORES ON SPAN TEST

5512 LPRINT

5515 LPRINT

5520 LPRINT 
RESULT(0, 1, 2); RESULT(1, 1, 2); RESULT(2, 1, 2); RESULT(3, 1, 2); " SPAN(1) = SPANA

5525 LPRINT

5530 LPRINT
RESULT(0, 2, 2); RESULT(1, 2, 2); RESULT(2, 2, 2); RESULT(3, 2, 2); " SPAN(4) = SPANB

5535 LPRINT

5540 LPRINT
RESULT(0, 3, 2); RESULT(1, 3, 2); RESULT(2, 3, 2); RESULT(3, 3, 2); " SPAN(8) = SPANC

5545 LPRINT

5550 LPRINT
RESULT(0, 4, 2); RESULT(1, 4, 2); RESULT(2, 4, 2); RESULT(3, 4, 2); " SPAN(12) = SPAND

5555 LPRINT

5560 RETURN

5565 REM

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5550 PRINT";RESULT(0,4,2);RESULT(1,4,2);RESULT(2,4,2);RESULT(3,4,2);
RESULT(4,4,2);";SPAN(12)=";SPAND
5555 LPRINT
5560 RETURN
5565 REM *** RETURNS TO RESULT OPTIONS SEQUENCE***

5599 REM
***********************************************************************
5600 REM *** SUBROUTINE FOR PRINTING RESULTS TO DISK FILES
5601 REM *** THERE ARE TWO FILES SET UP FOR EACH INDIVIDUAL USING THIS
5602 REM *** ROUTINE: SF STORES A SUMMARY OF OVERALL HITS AND SPAN
5603 REM *** SCORES; DF KEEPS A LIST OF THE 20 "HIT" RATINGS
5604 REM *** RECORDED DURING EACH SESSION FOR FUTURE USE WITH
5605 REM *** STATISTICAL PACKAGES IN A MERGED FILE. BOTH ARE SEQUENTIAL
5606 REM *** DATA FILES, TO BE MERGED OR READ BY OTHER PROGRAMS.
5607 REM***********************************************************************
5620 REM *** ROUTINE FOR STORAGE OF SUMMARY DATA
5630 OPEN SF$ FOR OUTPUT AS 1
5640 PRINT#1,S|" ";G$|" ";I$;L$|" ";T$|" ";DA$|" ";T$|" ";HITA|" ";SPANA;
5650 PRINT#1," ";HITB|" ";SPANB|" ";HITC|" ";SPANC|" ";HITD|" ";SPAND
5660 CLOSE#1
5670 SAVE SF$,A
5700 REM *** SUBROUTINE FOR STORING FULL RESULTS FROM HIT PROGRAM TO DISK***
5705 OPEN DFN$ FOR OUTPUT AS 2
5710 PRINT#2,S," ";G$," ";I$,L$," ";T," ";DA$," ";T$;RESULT(0,1,1)
5720 PRINT#2.USING"##.##";RESULT(0,1,1);RESULT(1,1,1);RESULT(2,1,1);RESULT(3,1,1);
RESULT(4,1,1);
5725 PRINT#2," ";
5730 PRINT#2.USING"##.##";RESULT(0,2,1);RESULT(1,2,1);RESULT(2,2,1);RESULT(3,2,1);
RESULT(4,2,1);
5735 PRINT#2," ";
5740 PRINT#2.USING"##.##";RESULT(0,3,1);RESULT(1,3,1);RESULT(2,3,1);RESULT(3,3,1);
RESULT(4,3,1);
5745 PRINT#2," ";
5750 PRINT#2.USING"##.##";RESULT(0,4,1);RESULT(1,4,1);RESULT(2,4,1);RESULT(3,4,1);
RESULT(4,4,1);
5760 CLOSE#2
5770 SAVE DFN$,A
5775 REM *** RETURNS TO RESULTS *****
5780 RETURN

5995 REM **** DATA FOR "TRIAL" BEGINS HERE ****
6000 DATA 67,;Z;" ";" ";";";Z,A
6010 DATA 90,;C;" ";";Z Q;"V ";";Z,B
6020 DATA 110,;D B;"P M;"W X;"C Z;";Z,C
6030 DATA 61,;H Q S G ;"N W X;"S K P;"L F ";"L,D
6040 DATA 36,;M B L;"X K ;";G T ;"Q ";"L,C
6050 DATA 23,;V ;"K L ;";S ;"L,B
6060 DATA 86,;K ;"Z V ;";H ;"Z,B
6070 DATA 105,;P ;"Q X ;"Z K G B;"H ;"Z,C
6080 DATA 46,;C G ;"J N K ;";"R L ;"F;L,C
6090 DATA 7,;"L ;";";";L,A
6100 DATA 62,;J K B ;"S M;"F R D ;"W L H ;"T;L,D
6110 DATA 95,;M ;"D ;";N ;";Z;"Z,B
6120 DATA 39,;Q B ;"L P;"J Y ;"D X ;"L,C
6130 DATA 41,;K X V ;"S C P ;"L F ;";";";L,C
6140 DATA 74,;";";Z ;";";";Z,A
6150 DATA 87,;F ;"K ;"Z ;";X ;"Z,B
6160 DATA 121,;W C ;"H N J ;";Z PM ;"Y B Q N ;Z,D
6170 DATA 14,;";";";L ;";L,A
6180 DATA 22,;";YL ;";W ;";V ;";L,B
6190 DATA 58,;T D K ;"X Y ;"W L V Q ;"R G P ;"L,D
6200 DATA 69,;";Z ;";";";Z,A
6210 DATA 112,;D H S ;"W C ;"G ;";P Z ;"Z,C
6220 DATA 123,;C S J ;"M P ;"D Q X ;"N V H ;"Z,D
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6230 DATA 18,"L","J","C","S","L,B
6240 DATA 13,"L","L","L","L,A
6250 DATA 120,"N D H J","MRZ","V WG","Y P","Z,D
6260 DATA 64,"D W","G H S","X PM","J CKL","LD
6270 DATA 29,"QD","K","L","L,B
6280 DATA 1,"L","L,"LA
6290 DATA 68,"Z","Z","Z","ZA
6300 DATA 127,"P F T","RK","Q","B C Y","N","Z X","Z,D
6310 DATA 108,"SB","R K","QZ","Y F","Z,C
6320 REM *** DATA FOR FIRST "TEST" RUN STARTS HERE ***
6330 DATA 43,"WG H","X","L","L","LC
6340 DATA 81,"ZT","DK","Z","ZB
6350 DATA 69,"Z","Z","Z","ZA
6360 DATA 60,"N F","M C GT","JHL","YQR","L,D
6370 DATA 119,"DY T","RC ZG","D K","Q","MP","Z,D
6380 DATA 15,"L","L","L","LA
6390 DATA 26,"L","L","L","LA
6400 DATA 38,"J","LGK","X","MY","LC
6410 DATA 50,"N LY","M WD","F V H","Q SP","L,D
6420 DATA 95,"M","D","M","ZB
6430 DATA 121,"WC","HN J","Z PM","Y BQB","Z,D
6440 DATA 106,"DS","MG","HZ","F","W","Z,C
6450 DATA 47,"Z","M","X","YH","QC","Z,C
6460 DATA 46,"C","G","JNK","RL","F","LC
6470 DATA 10,"L","L","L","LA
6480 DATA 21,"W","L","Y","V","L,B
6490 DATA 65,"Z","Z","Z","ZA
6500 DATA 99,"V ZK","LDR","N M","Z,C
6510 DATA 126,"P B","GMVQ","C RK","Y ZN","Z,D
6520 DATA 29,"QD","K","L","L,B
6530 DATA 110,"D B","P M","W X","C Z","Z,C
6540 DATA 1,"L","L","L","LA
6550 DATA 80,"L","L","L","ZA
6560 DATA 37,"W B","LQ","G N","DS","L,C
6570 DATA 19,"LS","J","H","LB
6580 DATA 117,"GN H","ZQD","WRKB","JY","Z,D
6590 DATA 62,"JKB","S M","FR D","WLH","T","L,D
6600 DATA 73,"Z","Z","Z","ZA
6610 DATA 93,"G","F W","Z","ZB
6620 DATA 6,"L","L","L","LA
6630 DATA 92,"G","C Z","P","ZB
6640 DATA 63,"Y MK","CW B","X Q","P FLD","L,D
6650 REM *** DATA FOR SECOND "TEST" RUN BEGINS HERE ***
6660 DATA 91,"T","J","ZW","ZB
6670 DATA 103,"J","Y ZC","F M","S","Z,C
6680 DATA 49,"LN W","C DB","P YV","S G F","L,D
6690 DATA 84,"Z","V","DG","ZB
6700 DATA 3,"L","L","L","LA
6710 DATA 25,"G","Y L","R","LB
6720 DATA 39,"Q B","LP","JY","DX","L,C
6730 DATA 125,"CM X","Q G","P DW","Z JRT","Z,D
6740 DATA 72,"Z","Z","Z","ZA
6750 DATA 90,"C","Z Q","V","ZB
6760 DATA 59,"NY VG","J M","QLK","HCS","L,D
6770 DATA 109,"SB","R K","QZ","Y F","Z,C
6780 DATA 34,"X L K","W P","MT","S","L,C
6790 DATA 18,"L J","C S","L","LB
6800 DATA 114,"C Z J R","G W","BDS","PK V","Z,D
6810 DATA 79,"Z-Za","Z-Za","Z-Za","Z-Za
6820 DATA 16,"A","A","L","LA
6830 DATA 82,"G Z R","Q","ZB
6840 DATA 101,"W TX","Z K Q","G","R","Z,C
6850 DATA 31","K","B LW","L,B
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6860 DATA 42,"V","K","M","GL","C","SW","LC"
6870 DATA 120,"N,D,H,J,""MRZ","V","WG","Y","P","ZD"
6880 DATA 13,"""","L","LA"
6890 DATA 61,"HQS","G","NW","X","SK","P","LF","LD"
6900 DATA 112,"DHS","WC","G","PZ","ZC"
6910 DATA 44,"G","PB","R","L","TC","CM","LC"
6920 DATA 12,"""","L","LA"
6930 DATA 68,"Z",""","ZA"
6940 DATA 118,"RBY","TW","J","X","J","PSDV","ZD"
6950 DATA 53,"FKQD","L","CP","WTB","G","N","LD"
6960 DATA 30,"H",""","LT","LB"
6970 DATA 76,"""","Z",""""""""""""""""""""""""""""""""
6980 REM *** DATA FOR THIRD TEST RUN BEGINS HERE ***
6990 DATA 9,"""","L","LA"
7000 DATA 51,"SLT","BP","M","N","DFK","Q","K","LD"
7010 DATA 76,"""","Z",""""""""""""""""""""""""""""""""
7020 DATA 86,"K","ZV",""H","ZB"
7030 DATA 89,"W","Z","T","G","ZB"
7040 DATA 27,"Q","C","L","X","LB"
7050 DATA 48,"WF","JR","C","N","QL","LC"
7060 DATA 55,"WR","GVL","QCD","WT","J","LD"
7070 DATA 102,"PV","ZG","S","JFY","ZC"
7080 DATA 66,"Z",""",""","ZA"
7090 DATA 4,"L",""","LA"
7100 DATA 36,"MBL","XK","GT","Q","LC"
7110 DATA 111,"K","GS","X","J","M","ZP","ZC"
7120 DATA 56,"TFBW","MR","L","N","PK","DG","LD"
7130 DATA 123,"CJS","M","P","QZX","NVH","ZD"
7140 DATA 33,"LS","T","BJ","YPV","LC"
7150 DATA 85,"Z","YB","K","ZB"
7160 DATA 7,"L","LA"
7170 DATA 22,"YL","W","V","LB"
7180 DATA 108,"SB","R","K","QZ","Y","F","ZC"
7190 DATA 122,"GNJ","YSQ","HZMP","B","K","ZD"
7200 DATA 11,"L","LA"
7210 DATA 96,"P","W","V","ZB"
7220 DATA 124,"WN","CDP","GV","Z","YX","K","ZD"
7230 DATA 100,"MD","Z","X","Y","RGN","ZC"
7240 DATA 71,"Z","Z","ZC"
7250 DATA 40,"J","HTML","QY","B","LC"
7260 DATA 24,"CS","S","L","J","LB"
7270 DATA 116,"GZ","X","PR","N","MD","VTS","Y","ZD"
7280 DATA 17,"L","Q","M","W","LB"
7290 DATA 58,"TDK","X","Y","WL","QV","RGP","LD"
7300 DATA 74,"Z","ZA"
7310 REM *** DATA FOR FOURTH TEST RUN BEGINS HERE ***
7320 DATA 128,"DHB","X","TK","P","SB","F","R","QZ","ZD"
7330 DATA 87,"F","K","Z","X","ZB"
7340 DATA 32,"NS","PL","LB"
7350 DATA 67,"Z","ZA"
7360 DATA 78,"Z","ZA"
7370 DATA 45,"XBJ","V","SN","LC"
7380 DATA 14,"L","LA"
7390 DATA 64,"D","W","GH","S","X","PM","JCK","L","LD"
7400 DATA 104,"H","Z","P","WX","S","NF","ZC"
7410 DATA 127,"PF","T","RK","Q","BC","Y","N","ZX","ZD"
7420 DATA 75,"Z","ZA"
7430 DATA 20,"L","R","Q","Y","LB"
7440 DATA 52,"TDKL","K","VS","R","F","B","NX","LD"
7450 DATA 5,"L","LA"
7460 DATA 105,"P","QX","ZKGB","H","ZC"
7470 DATA 28,"H","QX","L","LB"
7480 DATA 23,"V","K","L","S","LB"