

Panic Disorder : The strength and influence of selected variables as predictors of Comorbidity

and

Research Portfolio

**Submitted in partial fulfilment of the Degree of Doctor of Clinical Psychology within the
Faculty of Medicine, University of Glasgow**

Therese Carney

September 1995

ProQuest Number: 11007761

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 11007761

Published by ProQuest LLC (2018). Copyright of the Dissertation is held by the Author.

All rights reserved.

This work is protected against unauthorized copying under Title 17, United States Code
Microform Edition © ProQuest LLC.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

Ther
10408
C971

GLASGOW
UNIVERSITY
LIBRARY

Acknowledgments

The author would like to thank all those who have advised on and supported this project. In particular :

Prof. Colin Espie, Department of Psychological Medicine, University of Glasgow

Dr Jim White, Department of Clinical Psychology, Landsdown Clinic

John and Saoirse

Contents

Section	Title	Page
	Acknowledgments	
	Contents	
	List of Appendices	
	List of Tables	
	List of Figures	
1	Literature Review	
	Classification of Psychological and Psychiatric Disorders	1
	Impact of Classification on Comorbidity	1
	Assigning principal and Comorbid diagnoses	3
	Research and Clinical Implications	4
	Rates of Comorbidity	5
	Comorbidity and Anxiety Disorders	5
	Patterns of Comorbidity	6
	Symptom Comorbidity	7
	Panic Disorder and Comorbidity	8
	Future directions for Research	9
	Reference List	10
2	Research Proposal	
	Introduction	15
	Summary	16
	Aims and Hypothesis	17
	Subjects	18
	Measures	19
	Research Method and Design	21
	Procedure	22
	Settings and Equipment	22
	Data Analysis	22
	Purpose	23
	Reference List	24
3	Major Research Proposal	
	Abstract	
	Introduction	28
	Method : Subjects	31
	Measures	33
	Procedure	34

Section	Title	Page
3 (cont'd)	Results	35
	Discussion	39
	Reference List	43
4	Improving Health Care in Scotland guided by “Framework for Action”. An evaluation of Psychologists health attitudes, behaviour and role.	
	Abstract	
	Introduction	48
	Method : Subjects	52
	Materials	52
	Design and Procedure	52
	Results	53
	Discussion	56
	Reference List	59
5	Group Management of Chronic Pain. An outcome Study.	
	Abstract	
	Introduction	61
	Method : Subjects	64
	Measures	65
	Procedure	66
	Results	67
	Discussion	69
	Reference List	73
6	Specific Language Impairment. A Single Case Report.	
	Abstract	
	Introduction	76
	Clinical Presentation	78
	Procedure	78
	Measures	79
	Results	80
	Discussion	81
	Reference List	84
7	Adrenoleukodystrophy : A Psychological Profile. Case Study of a thirteen year old male.	
	Abstract	
	Introduction	87

Section	Title	Page
7 (cont'd)	Clinical Presentation	88
	Formal Assessment	91
	Results	93
	Discussion	94
	Reference List	97
	Appendices	

List of Appendices

Number	Title
1 1	Instructions to Authors. Journal of Consulting and Clinical Psychology
2.1	Instructions to Authors. Journal of Consulting and Clinical Psychology
2.2	Rate and Pattern of Comorbid Diagnosis
2.3	Subject variables
2.4	Symptom and Adjustment variables
2.5	Ways of Coping
2.6	Mean FSAQ Scores
2.7	Discriminant Analysis U. Significant Variables. D. A. Selects the best
2.8	Correlation Matrix
2.9	Raw Data Set
3.1	Guidelines for Contributors. Health Education Journal
3.2	Health Questionnaire
4.1	Manuscript Preparation. International Journal of Rehabilitation Research
4.2	Course Evaluation Questionnaire
5.1	Notes for Contributors. Journal of Child Psychology and Psychiatry
6.1	Notes for Contributors. Journal of Child Psychology and Psychiatry

List of Tables

Section	Number	Title	Page
3	1	Demographic Characteristics	32
	2	Results of Between-groups difference testing on symptomatic and adjustment measures using the Mann-Whitney procedure	36
	3	U. Significant variables remaining in analysis	38
	4	Classification Table (selected cases only)	38
	5	Classification Table for subjects withheld from model construction	39
4	1	Health Attitudes	53
	2	Health Behaviours	54
	3	Health Promotion	55
5	1	Demographics	64
	2	Outcome Measures	68
7	1	Wechsler Intelligence Scale for Children Revised (Scottish) (WISC-RS)	92

List of Figures

Section	Number	Title	Page
5	1	Partner's Responses	68
	2(a)	General Health Questionnaire Results	69
	2(b)	Hospital Anxiety and Depression Scale Results	69
7	1	Bender Visual Motor Gestalt Test Results	91
	2	Benton Visual Retention Test Results	92
	3	Neale Analysis of Reading Ability - Revised British Edition Results	92

Panic Disorder : The strength and influence of selected variables as predictors of Comorbidity

Literature Review

Therese Carney

Department of Psychological Medicine

University of Glasgow

Classification of Psychological and Psychiatric Disorders

Classification systems have been in existence since the earliest times in an attempt to put some order into the chaos of mental illness. The two major taxonomic systems used in the mental health field at present include the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV, American Psychiatric Association, 1994) and the International Classification of Diseases (ICD-10, World Health Organisation, 1992). The development of these objective and reliable systems represented a change of paradigm from the previous medical model in terms of the concept of multiple discrete mental disorders; the use of operational criteria; structured interviews; diagnostic algorithms; reliability and validity (Klerman, 1990). Disquiet with the medical model came into focus with challenges to the view that mental illness was a disease (Szasz, 1961), and the growing evidence that mental illness included social adjustment problems and personal issues. Impetus for the development of new classification systems was driven by two further changes. Firstly, the increased involvement of Social Scientists during the 1960's and 1970's who were not primarily concerned with the search for biological causes for mental illness; and secondly, the input of Psychologists and Statisticians who questioned the nature of the medical model's diagnostic systems and the poor reliability of psychiatric diagnosis. The resulting classification systems have helped to define disorders, improve reliability, clinical communication and ability to generalise from and across research studies. Discussion within this Literature Review will concentrate on DSM criteria.

Impact of Classification on Comorbidity

One of the consequences of the development of classification systems has been the interest generated in the area of comorbidity. This term has been defined as 'any distinct additional clinical entity that has existed or that may occur during the clinical course of a patient who has the index disease under study' (Feinstein, 1970). The broad coverage of both psychological

and psychiatric disorders increases the number of individuals likely to meet the criteria for one or more disorders. In addition, the severity thresholds for many conditions are relatively low which encourages frequent diagnosis and comorbidity. DSM-111-R (APA, 1987) allows for multiple diagnoses, with each condition being assessed on its own merit and being diagnosed independently, when present with another comorbid condition.

This however, has not always been an option. The previous DSM-111 (APA, 1980) operated on a system of an *apriori* diagnostic hierarchy in which the most severe condition took precedence over the other(s), when more than one was present simultaneously. Therefore, an additional diagnosis was not made as the symptoms were assumed to belong to the more severe or principal disorder (Brown & Barlow, 1992). Most of the anxiety disorders were excluded when presenting with major depression, as this was considered to be of greater severity. Criticism with these exclusionary criteria soon became widespread. Boyd, Burke, Gruenberg, Holzer, Rue, George, Karno, Stoltzman, McEvoy, & Nestadt (1984) reported that patients diagnosed with having one DSM-111 disorder were likely to meet the criteria for at least one additional DSM-111 disorder when the exclusion criteria were dropped. In addition, Lechman, Weissman, Merikangas, Pauls, & Prusoff (1983) reported that relatives of individuals with major depression and panic disorder had markedly increased rates of major depression, anxiety disorders and alcoholism compared to relatives of depressed individuals without an anxiety disorder. However, the hierarchical rules present in DSM-111 obscured this result as a diagnosis of depression and panic disorder could not have been made using this classification system. The introduction of DSM-111-R solved many of these problems by withdrawing the aforementioned exclusionary criteria. Changes in classification however, were reflected by the changes in reported rates and types of comorbidity which in many instances increased. This in turn, has stimulated recent interest and research.

Assigning Principal and Comorbid Diagnoses

In the field of mental health a comorbid condition is a symptom cluster which is concurrent with the symptoms of another disorder, but is not a typical feature of that disorder (Spitzer & Williams, 1983). For example, a patient who meets the criteria for panic disorder but who also reports unrealistic and excessive worry about her children, her health or financial matters would be given the additional diagnosis of generalized anxiety disorder (GAD). Employing DSM-111-R criteria often results in assigning at least one comorbid diagnosis and in some cases several. In this situation, it is necessary to assign a primary diagnosis for the purpose of treatment planning. The primary-secondary distinction can be made in three ways (Klerman, 1990).

Firstly, the primary condition is the one which comes first chronologically. Researchers use this criteria in the main as it assists in the generation of homogeneous groups which reduces variability in subject selection and contributes to the knowledge of aetiology. No causation is implied nor is there any reference to which condition requires initial clinical attention. The second distinction carries with it causal implications. Secondary conditions are usually regarded as having a 'due to' causation. The DSM-111-R incorporates this version of the primary-secondary distinction in its criteria for a number of its conditions, for example, where the clinical syndrome is clearly the consequence of an organic mental disorder. The third distinction emphasizes the predominance of the clinical features. This requires the Clinician's judgement as to which condition is of most importance for treatment decisions. It is usually the condition which causes greatest life interference and prompts referral for treatment. Studies on comorbidity can be conducted cross-sectionally or longitudinally. In studies using the former design, disorders which are present at one point in time are recorded, whereas longitudinal studies take in a specified length of time and record the disorders which present over this time frame.

Research and Clinical Implications

Comorbidity has important implications for research and clinical practice because of its influence on diagnosis, classification, treatment and prognosis. The clinical course for a patient with a principal and comorbid diagnosis may be quite different from those with no additional diagnosis. For example, patients with panic disorder and social phobia have a greater lifetime risk of major depression than those with panic disorder alone (Stein, Shea, & Uhde, 1989). Also, patients with major depression and panic attacks are less likely to recover over a two year period than those without panic attacks (Coryell, Endicott, Andreasen, Keller, Clayton, Hirschfeld, Scheftner, & Winokur, 1988). Several studies have indicated the importance of recognising comorbidity when planning treatment and knowing when to refer to psychiatry. For example, Foa, Grayson & Steketee (1987) found that Imipramine was of little benefit to obsessive patients who were not depressed but was clearly effective in treating depressed obsessive patients. Coryell et al. (1988) reported that patients with panic disorder and major depression have more severe symptomology and poorer response to antidepressant medication than patients with panic disorder or major depression alone. Lifetime rates of suicide attempts are also reportedly higher when panic and major depression both occur, e.g. 19.5% compared with those with panic disorder alone (7%), or major depression alone (7.6%) (Johnson, Weissman, & Klerman, 1990).

Research on treatment outcome should also consider the impact of comorbidity. Brown & Barlow (1992) address this issue by commenting on the loss of information regarding the effects of treatment when patients are included into treatment studies on the basis of a given principal diagnosis without consideration of comorbid diagnosis. This may give the illusion of sample homogeneity when the sample is in fact heterogeneous. They call for future research on the effects of comorbid disorder or symptoms on long and short term response to treatment, and on the type of adjustments which can be made to enhance treatment efficacy when comorbidity is present.

Rates of Comorbidity

DSM-111-R, encourages multiple diagnoses and a general finding has been high rates of comorbidity between anxiety disorders, and with mood disorders. Barlow, Di Nardo, Vermilyea, Vermilyea, & Blanchard (1986) studied 126 patients of which 108 (86%) were assigned a diagnosis within the anxiety-affective spectrum. Of these 108 patients, 65% were given at least one comorbid diagnosis. This result, which has been supported from other studies, demonstrates the importance of research in this area in order to determine the degree to which the overlap reflects a true underlying relationship, and to determine the nature of this relationship. Clarity here, would help in separating fundamental relationships from those which exist because of definitional artifact, and in finding new and improved methods of organizing and classifying psychopathology. However, high rates of comorbidity may be indicative of poor discriminant validity among the diagnostic categories. As Blashfield (1990) states 'the diagnostic system may be artifactually (sic.) distinguishing phenomena that would be more parsimonious if combined'.

Comorbidity and Anxiety Disorders

Most of the present interest in comorbidity is centered on the Anxiety Disorders which are one of the major problems requiring medical and mental health input (Boyd, 1986). It has also been estimated that 15% of the population will suffer from a DSM-111-R anxiety disorder at some time during their lives (Regier, Burke, & Burke, 1990). Studies on comorbidity have so far concentrated on identifying rates and patterns at both the syndrome and symptom level. Comorbidity between different mental disorders is frequent in both clinical (75%), and epidemiological (50%) populations. Both the New Haven survey (Weissman, Myers, & Harding, 1978) and the Epidemiological Catchment Area programme (ECA ; Regier et al. 1990) have reported that 30-80% of people with an anxiety disorder had at least one other

comorbid condition. In clinical samples the results are similar. Sanderson, Di Nardo, Rapee, & Barlow (1990) found that the overall rate of comorbidity was 70% which closely corresponds to that found by Barlow et al. (1986). Other studies have reported lower rates e.g. (Moras, Di Nardo, Brown, & Barlow, 1991) who found that 50% of patients with an anxiety disorder had at least one additional clinically significant anxiety or mood disorder.

Differences in reported rates of comorbidity reflect differences between samples that are studied e.g. Clinical versus Community, and the various diagnostic criteria used. Another factor which should be recognised, is the threshold used to determine the presence or absence of various disorders (Frances, Widiger, & Fyer, 1990). In addition to affecting overall comorbidity rates this also influences patterns. For example, Moras et al.(1991) reported that simple and social phobia were given as additional diagnosis much more often when thresholds for 'caseness' was lowered whereas other diagnosis e.g. obsessive compulsive disorder remained unaffected.

Patterns of Comorbidity

In addition to looking at rates of comorbid diagnosis, studies have also revealed consistent patterns of comorbidity. Moras et al. (1991) reported that the principal diagnosis of generalized anxiety disorder and panic disorder with severe agoraphobia were the categories associated with the highest comorbidity rates, and simple phobia was associated with the lowest. They also found that GAD was the most frequently assigned additional diagnosis (23%), followed by social phobia (14%). de Ruiter, Rijken, Garssen, Van Schaik, & Kraaimaat (1989) reported that simple phobia was the most common additional diagnosis followed by social phobia and dysthymia. When major depression and dysthymia were considered together as a mood disorder, then this became the most frequently assigned additional diagnosis in both the Barlow et al. (1986) and the de Ruiter et al.(1989) studies.

Indeed, studies looking at comorbidity between mood and anxiety disorders have cited rates between 19% (Fawcett, & Kravitz, 1983) and 91% (Bowen, & Kohout, 1979) with a mean between 30-40% (Hecht, Von Zerssen, Krieg, Possel, & Wittchen, 1989). This variability is again reflected in recent studies. Sanderson et al. (1990) assigned dysthymia or major depression as an additional diagnosis in 33% of his sample whereas Barlow et al. (1986) followed at 29% and di Nardo & Barlow (1990) assigned this additional diagnosis at 16%. This variability may be a reflection of factors commented on previously.

Symptom Comorbidity

The high pattern of syndrome comorbidity in these studies has led to questions about the discriminant validity among the diagnostic categories, and the development of an interest in symptom comorbidity (Brown & Barlow, 1992). Key features found in the various anxiety disorders e.g. panic attacks, worry, intrusive thoughts and social fears, seem to exist in most patients presenting with a principal diagnosis of almost any anxiety disorder. Whereas DSM-111-R is a categorical system which follows the medical tradition, (GAD is diagnosed or not), an alternative approach would be to determine and rate the underlying dimensions that may best account for the surface correlation among clinical categories. For example, the DSM-111-R categories of panic disorder with agoraphobia, panic disorder without agoraphobia and agoraphobia with panic may be more adequately described using a dimensional rather than a categorical system (Frances et al. 1990). It may be, that these categories are different manifestations of a common underlying pathology. A dimensional system that provided ratings of two or three key features e.g. avoidance behaviour and panic attacks which are found to cut across these three categories would be an alternative way to address the comorbidity question. Proponents for this approach to examining the impact of comorbidity e.g. Sanderson et al. (1990) suggest that it may be more accurate in describing the overall pattern of psychopathology. Brown & Barlow (1992) hoped that it would help identify symptoms, or

symptom patterns which predict response to treatment or the course of the disorder, and finally help to identify symptoms which are responsive or resilient to psychological and pharmacological treatments. Researchers have recently proposed alternative classification systems that simply measure the presence of various symptoms on a continuum and use this data in a dimensional analysis e.g. Blashfield (1990).

Panic Disorder and Comorbidity

Many of the studies on comorbidity have been conducted on a general level e.g. looking at rates and patterns between/within the anxiety and mood disorders. While this is a topic worthy of exploration it is also useful to look in more depth at specific disorders. The majority of studies at this level have focused on panic disorder. This condition was first described as a separate anxiety state in DSM-111 and now occupies a central position among the anxiety disorders in DSM-111-R. A further step has been taken by ICD-10, where panic disorder is now recognised as a disorder in its own right, in addition to its coexistence with agoraphobia. Between 35-93% of patients with panic disorder have a comorbid diagnosis (Stein et al. 1989), while 69% of patients with panic disorder and agoraphobia received at least one additional diagnosis (Sanderson et al. 1990). The most frequently assigned additional diagnosis for patients with a principal panic disorder has been GAD followed closely by mood disorder (Brown & Barlow, 1992) or simple phobia followed by mood disorder (Sanderson et al. 1990). Other studies have found that the overall comorbidity rate for panic disorder with agoraphobia was significantly higher than that for panic disorder alone. Moreover, panic patients with agoraphobia were more likely to receive multiple diagnoses (up to eight) than were patients with panic disorder alone (Noyes, Reich, Christiansen, Suelzer, Pfohl & Coryell, 1990). The high rate of mood disorder found in patients with a principal panic disorder has been commented on previously. Clark (1989) concluded from a review of studies that the mean percentage of patients with past and present major depression was 67%. One

longitudinal study of patients with panic disorder who were non-depressed at entry into the study and who were followed up for two years, found that 24% experienced a major depressive episode during the two years (Ball, Otto, Pollack, & Rosenbaum, 1994). Epidemiological studies (e.g. Wittchen, Essau & Kreig, 1991b) have found that 32-50% of all subjects with panic disorder also fulfilled criteria for major depression. The majority of these patients had depression clearly after the occurrence of panic disorder and thus it could be regarded as secondary.

Future directions for research

The recent interest in Comorbidity at both the general and more specific syndrome level has concentrated mainly on rates of occurrence and patterns of overlap of both the anxiety and mood disorders (Barlow et al. 1986 ; Sanderson et al. 1990). This theme of interest has been stimulated in an attempt to improve classification and determine the fundamental relationship between the various disorders. Indeed, ICD-10 has included a new category labelled mixed anxiety-depression. Dimensional theorists have also addressed this issue in relation to the presence or absence of key symptoms, but have not related their findings to the present categorical system. There is a need for more studies which link the two parallel research directions, perhaps by looking at symptoms within a categorical system. Other studies have looked in more depth at the relationship between specific DSM-111-R conditions (Stein et al. 1989). They reported that panic patients with a comorbid condition i.e. social phobia were more likely to have an additional co-existing depression. However, there is little information available on the variables which predict comorbidity. Future research which examines predictors of comorbidity within a specific DSM-111-R diagnosis would help identify those individuals more at risk, and help target treatment on the predictor variables associated with comorbidity.

Reference List

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders* (1V ed.), Washington DC.

American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., revised), Washington DC.

American Psychiatric Association (1980). *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.), Washington DC.

Ball, S.G., Otto, M.W., Pollack, M.H., Rosenbaum, J.F. (1994). Predicting prospective Episodes of Depression in patients with Panic Disorder: A longitudinal Study. *Journal of Consulting and Clinical Psychology*. Vol - 62, No 2. 359-365.

Barlow, D.H., Di Nardo, P.A., Vermilyea, B.B., Vermilyea, J.A., & Blanchard, E.B. (1986). Comorbidity and depression among the anxiety disorders: Issues in classification and diagnosis. *Journal of Nervous and Mental Disease*, 174, 63-72.

Blashfield,R.K. (1990). Comorbidity and Classification in J.D. Maser & C.R. Cloniger (Eds.) *Comorbidity of Mood and Anxiety disorders* (pp. 61-82) Washington DC: American Psychiatric Press.

Bowen, R.C., and Kohout, J. (1979). The relationship between agoraphobia and primary affective disorders. *Can. J. Psychiatry*, 24, 317-322.

Boyd, J.H., Burke, J.D., Gruenberg, E., Holzer, C.E., Rue, D., George, L.K., Karno, M., Stoltzman, T., McEvoy, L., & Nestadt, G. (1984). Exclusion criteria of DSM-III: A study of co-occurrence of hierarchy free syndromes. *Archives of General Psychiatry*, 41, 983-989.

Boyd, J.H. (1986). Use of Mental Health Services for the treatment of Panic Disorder. *American Journal of Psychiatry*; 143, 1569-1574.

Brown T.A., and Barlow D.H. (1992). Comorbidity among Anxiety Disorders: Implications for treatment and DSM-IV. *Journal of Consulting and Clinical Psychology*. Vol 60. No.6. 835-844.

Clark, L.A.(1989). The Anxiety and Depressive disorders: Descriptive psychopathology and differential diagnosis. In P. Kendall & D. Watson (Eds), *Anxiety and Depression: Distinctive and overlapping features* (pp. 83-129). San Diego, CA: Academic Press.

Coryell, W., Endicott, J., Andreasen, N.C., Keller, M.B., Clayton,P.J., Hirschfeld, R.M.A., Scheftner, W.A., & Winokur, G. (1988). Depression and Panic Attacks: The significance of overlap as reflected in follow up and family study data. *American Journal of Psychiatry*, 145, (3), 293-300.

de Ruiter, C., Rijken, H., Garssen, B., Van Schaik, A., & Kraaimaat, F. (1989). Comorbidity among the anxiety disorders. *Journal of Anxiety Disorders*, 3, 57-68.

Di Nardo, P.A., & Barlow, D.H. (1990). Syndrome and symptom comorbidity in the anxiety disorders. In J.D. Maser & C.R. Cloniger (Eds.), *Comorbidity in anxiety and mood disorders* (pp, 205-230). Washington, D C: American Psychiatric Press.

Fawcett, J., and Kravitz, H.M. (1983). Anxiety syndromes and their relationship to depressive illness. *Journal of Clinical Psychiatry*, 44, 8-11.

Foa, E.B., Grayson, J.B., & Steketee, G. (1987). Depression, habituation and treatment outcome in obsessive-compulsives. In J.C. Boulougouris (Ed.), *Practical applications of learning theories in psychiatry* (pp. 129-142). New York: Wiley.

Feinstein, A.R. (1970). The Pre-therapeutic classification of comorbidity in chronic disease. *Journal of Chronic Diseases*, 23, 455-468.

Frances, A., Widiger, T., Fyer, M.R. (1990). The Influence of classification methods on comorbidity. In J.D. Maser & C.R. Cloniger (Eds.) *Comorbidity of Mood and Anxiety disorders*. (pp. 41-59).

Hecht, H., Von Zerssen, D., Krieg, C., Posselt, J., and Wittchen, H. (1989). Anxiety and Depression: comorbidity, psychopathology and social functioning. *Compreh. Psychiatry*, 30, 420-433.

Johnson, J., Weissman, M., & Klerman, G.L. (1990). Panic Disorder, comorbidity, and suicide. *Archives of General Psychiatry*, 47, 805-808.

Klerman, G., (1990). Approaches to the phenomena of comorbidity. In J.D. Maser, & C.R. Cloniger (Eds.) *Comorbidity of mood and Anxiety disorders* (pp 13-37).

Lechman, J.F., Weissman, M., Merikangas, K., Pauls, A., & Prusoff, B. (1983). Panic Disorder and Major Depression. *Archives of General Psychiatry*, 40, 1055-1060.

Moras, K., Di Nardo, P.A., Brown, T.A., & Barlow, D.H. (1991). Comorbidity and depression among the DSM-111-R anxiety disorders.

Moras, K., & Barlow, D.H., (1992). Dimensional approaches to diagnosis and the problem of anxiety and depression. In W Fiegenbaum (Eds.) *Perspectives and Promises of Clinical Psychology*. (pp. 23-37). New York, Plenum Press.

Noyes, R., Reich, J., Christiansen, J., Suelzer, M., Pfohl, B & Coryell, W.A. (1990). Outcome of Panic Disorder: Relationship to diagnostic subtypes and comorbidity. *Archives of general Psychiatry*, 47, 809-818.

Regier, D.A., Burke, J.D., & Burke, K.C. (1990). Comorbidity of affective and anxiety disorders in the NIMH Epidemiologic Catchment Area Programme. In J.D. Maser & C.R. Cloniger (Eds.) *Comorbidity of mood and anxiety disorders* (pp. 113-122). Washington DC: American Psychiatric Press.

Sanderson, W.C., Di Nardo, P.A., Rapee, R.M., & Barlow, D.H. (1990). Syndrome comorbidity in patients diagnosed with a DSM-111-R anxiety disorder. *Journal of Abnormal Psychology*, 99, 308-312.

Spitzer, R.L., and Williams, J.B.W. (1983). Proposed revisions in the DSM-111 classification of anxiety disorders based on research and clinical experience. Paper presented at the conference, DSM: An interim appraisal, sponsored by the *American Psychiatric Association*, Washington D.C.

Stein, M.B., Shea, C.A., & Uhde, T.W. (1989). Social phobic syndromes in patients with Panic Disorder: Practical and theoretical implications. *American Journal of Psychiatry*, 146, 235-238.

Szasz, T.S. (1974). *The myth of mental illness: Foundations of a theory of personal conduct*, rev. ed. New York: Harper & Row.

Weissman, M.M., Myers, J.K., & Harding, P.S. (1978). Psychiatric disorders in a US urban community. *American Journal of Psychiatry*, 135, 459-462.

Wittchen, H.U., Essau, C.A., Krieg, C.J. (1991b). Comorbidity: Similarities and differences in treated and untreated groups. *British Journal of Psychiatry*, 159, 23-33.

World Health Organisation (1992). *International Classification of Disease*. 10 th Edition.

Panic Disorder : The strength and influence of selected variables as predictors of Comorbidity

Research Proposal

Therese Carney

Department of Psychological Medicine

University of Glasgow

Applicant: Therese Carney, Department of Psychological Medicine, Glasgow University.

Title: Panic Disorder: The Strength and Influence of Selected Variables as Predictors of Comorbidity.

Ethical Approval : Granted by Lanarkshire Health Board.

Introduction

Panic disorder affects 2% of the population (Weissman & Merikangas, 1986) and patients with this disorder are the most frequent users of outpatient mental health services (Boyd, 1986). Moreover, it is associated with depression, suicide and substance abuse (Weissman, Klerman, Markowitz, & Ouellette, 1989). Many develop agoraphobia or other comorbid disorders. However, research on comorbidity is relatively new. Prior to DSM-III-R these disorders were subsumed under disorders considered more significant (e.g. psychotic and depressive disorders). Present research in this area mainly explores rates and patterns of comorbidity (Sanderson, Di Nardo, Rapee, & Barlow, 1990). Between 35-93% of patients with Panic Disorder have a comorbid condition (Stein, Shea & Uhde, 1989), (Brown & Barlow, 1992). Other studies have looked at various symptoms which appear to be key or common features (Frances, Widiger & Fyer, 1990). What is less well known are the features which predict the likelihood of having a comorbid diagnosis. Why do some patients present with panic disorder alone while others present with panic and agoraphobia or panic with mood disorder? If predictor variables do exist, then identifying and examining them may help in the management and treatment of panic disorder. At present there is little data relating to adjustment of treatment in light of comorbid symptoms. More effective treatment techniques could possibly reduce relapse rates.

Summary

This study has been designed to complement previous research on comorbidity within panic disorder. It was also hoped that additional new information will be obtained on the impact of specific predictor variables. The term 'predictor variable' refers to those variables which are associated with a comorbid diagnosis. Measures describing this association should help in the prediction of a comorbid diagnosis. These predictor variables, or associations may be related to comorbidity in such a way that an increase in one may be accompanied by an increase in the other, or conversely an increase in one may be accompanied by a decrease in the other. No attempt will be made to measure or quantify this relationship which should therefore be seen as an association rather than a correlation.

The term comorbidity was coined by Feinstein (1970) and has its basis in general medicine. It refers to the co-occurrence of at least two different disorders in the same individual. The symptoms are concurrent with those of the principal disorder but are not a typical feature of that disorder (Barlow, 1988). This is distinct from associated symptoms in which the symptoms are aspects of the primary disorder and thus do not warrant a separate diagnosis. In this study, the principal diagnosis will be the one judged to be associated with the greatest distress or life interference at time of interview and the reason why treatment was sought. No attempt will be made to use a chronological or causal distinction which assumes that the principal diagnosis comes first temporally, or causes the comorbid symptoms (Klerman, 1990).

The study will be undertaken at Hairmyres Hospital, with patients referred from East Kilbride/Hamilton Primary Care Services.

Aims and Hypothesis

The Principal aim of the study is to address the following question:

Within Panic Disorder, can comorbidity be predicted by specific variables ?

The variables to be included in this study have been chosen as they are common features of panic disorder (Frances et al. 1990) or have been associated with additional psychological distress (Coryell, Endicott, Andreasen, Keller, Clayton, Hirschfeld, Scheftner & Winokur, 1988). They can be divided into three general categories.

1. *Subject variables* Epidemiological studies have shown that the risk of developing panic disorder varies with personal and environmental factors (Wittchen, Essau, Von Zerssen, Kreig & Zaudig, 1992). In order to look at comorbid status in panic disorder as related to subject and social variables the following will be included: age, sex, marital status, employment status, medication and dependants.
2. *Symptomatic and Clinical Variables* Studying symptoms and symptom patterns of panic disorder and comorbidity has given valuable information in the past (Blashfield, 1990). The clinical picture of panic disorder although consistent in some aspects (e.g. diagnostic criteria) will vary along some dimensions. In order to look at associations between these symptoms (and their variability), and comorbid status of the individuals, the following variables will be looked at : Severity of principal disorder (the severity rating of the ADIS-R and duration of symptoms), autonomic arousal, catastrophic ideation, relative contribution of somatic, affective, cognitive and behavioural components of panic and general anxiety and depressive status.

3. *Adjustment variables* The concept of adjustment usually refers to psychological well-being or adaptive mental functioning. Models of coping (Lazarus & Folkman, 1984), and Locus of Control (Wallston & Wallston, 1978) are just two variables which could explain differences in adjustment found in people who experience panic disorder and may be predictive of comorbidity. For these reasons they are included as potential predictor variables. These variables will be assessed by using standardised questionnaires and a diagnostic interview guided by DSM-111-R criteria.

In addition to the principal aim of the study, the following questions will be addressed:

Are anxiety symptoms less severe and more diffuse with comorbidity or does symptom severity increase with comorbidity?

What are the relative contributions of somatic, behavioural, cognitive and affective components in both groups ?

Are there any differences in the coping styles between the two groups ?

To what extent do subjects in both groups believe that their health is determined by their behaviour ?

Subjects

Subjects will be recruited from the waiting list of a local primary care psychological service. There has traditionally been good liaison between local GPs and Psychologists in the area of anxiety disorders, as there are strong clinical and research interests on this subject. All referrals used in the study will be referred from local GPs (the waiting list stands at 8

months). Only those referred with panic disorder (with or without a comorbid diagnosis) will be considered. Subjects with a history of psychosis, serious physical illness and current alcohol abuse will be excluded. The maximum time waiting before receiving an appointment will be eight months and this should gradually reduce to a few weeks only as the study progresses. It is hoped that a minimum sample size of 50 patients will be included but if possible the sample size will be increased.

Measures

A. Structured Clinical Interview

Anxiety Disorders Interview Schedule - Revised (ADIS-R ; Di Nardo and Barlow, 1988). This is a structured interview designed to assess in detail all anxiety and mood disorders and includes a screen for psychotic disorders, substance abuse and somatoform disorders. Additional background information including social and medical history and current medication are also assessed. Psychometric studies indicate that adequate reliabilities can be obtained from this measure for DSM-111-R depressive and anxiety disorders (Di Nardo, Moras, Barlow, Rapee and Brown, 1993)

B. Questionnaires

1. *Four Systems Anxiety Questionnaire (FSAQ ; Koksal and Power, 1990).* This is a 60 item self-report measure. It assesses anxiety on four components: somatic, cognitive, behavioural and affective. It has been shown to be reliable and valid in identifying different patterns of symptoms in different anxiety diagnostic groups and is sensitive to change following psychological treatment.

2. *Ways of Coping Checklist* (Folkman and Lazarus, 1985). This scale was designed to assess categories of coping posited by Lazarus and Folkman's transactional model of stress. It consists of 72 items and subjects are asked to indicate the extent to which they use each strategy to cope with a problem they choose as being most stressful to them at the time - in this study this was panic disorder. It categorises eight coping styles i.e. problem solving, cognitive restructuring, emotional expression, social support, passive avoidance, wishful thinking, self criticism and social withdrawal.

3. *Body Sensations Questionnaire* (BSQ ; Chambless, Caputo, Bright & Gallagher, 1984). This 17 item scale comprises items concerning sensations associated with autonomic arousal. Each item is rated on a 5 point scale ranging from not frightened or worried by this sensation (1), to extremely frightened by this sensation (5), which indicates how anxiety provoking the patient found each sensation.

4. *Agoraphobic Cognitions Questionnaire* (ACQ ; Chambless et al. 1984). Despite its name this 14 item scale comprises catastrophic ideation typically noted during exposure to anxiety provoking experiences including panic attacks. Each item is rated on a five point scale, ranging from thought never occurs (1) to thought always occurs (5), of the frequency which this thought occurred during a panic attack. Both scales have been found reliable and valid.

5. *Multidimensional Health Locus of Control Scale* (Wallston and Wallston, 1978). This scale measures three dimensions of perceived control over health, one of which assesses internal locus of control and two of which assess perceived control of health from external sources e.g. powerful others and chance.

6. *Hospital Anxiety and Depression Scale* (HAD ; Zigmond and Snaith, 1983). This self report scale has been designed to measure anxiety and depression in both out patient and in patient populations.

Research Method and Design

This will be a cross-sectional study, and no control group will be used. On the basis of the structured interview subjects will be divided into two independent groups, viz. those presenting with panic disorder alone and those presenting with panic disorder and comorbid diagnosis. A comparative analysis between these two groups will be carried out in order to identify predictor variables which may be present.

Making a reliable diagnosis in terms of principal and comorbid disorder will be of primary concern to this study as it is the basis on which subsequent information will rest. Therefore, certain criteria will have to be adopted. Firstly, all interviews will follow a standard format by using ‘The Anxiety Disorders Interview Schedule - Revised’ (ADIS-R). This semi-structured interview schedule has been designed to assess in detail all anxiety and mood disorders following DSM-III-R criteria. Psychometric studies indicate that it is reliable in measuring DSM-III-R anxiety and depressive disorders (Di Nardo & Barlow, 1988 ; Di Nardo et al. 1993). The interviewer will be trained in the use of the ADIS-R by an experienced clinician and practice will be undertaken before the study commences. Secondly, all interviews will be conducted by the same individual and for every subject included in the study, diagnosis will be confirmed by the senior clinician who is to commence follow-up treatment. Thirdly, caseness ratings will be based on levels of impairment or distress. The ADIS-R rates severity on a scale of 0 - 8. A rating of 4 or more indicates definite caseness while a rating of 3 indicates probable caseness. Only those with a score of 4 or over for both principal and comorbid diagnosis are to be included in the study. This rationale has been employed in order to strengthen the reliability of diagnosis.

Procedure

Subjects will be given an appointment date and time by post as is usual in the department. On initial appointment, the structured interview of the ADIS-R will be carried out to ensure standardisation of diagnosis. All interviews will be conducted by the author to reduce interpersonal bias. If a diagnosis of panic disorder is made, the subject will then be told of the study and given an explanation leaflet to read. If willing to participate they will then get the choice of completing the questionnaires in another room before leaving the department, or taking them away to complete at home and return by post. The information gathered from interview will then be written up in psychological case notes and passed onto the qualified clinical psychologist who is willing to see them subsequently for treatment. This initial contact should take between 60 and 90 minutes. All returned information will be matched via initials. Subjects will be seen on a Friday between March and November 1994, with an average of four appointments per day.

Settings and Equipment

All patients will be seen at the local Psychology department. Other than a consulting room and questionnaires used in the study no extra equipment will be required.

Data Analysis

All means of identification on questionnaires and interview schedules will be coded. This will also apply to computer data ensuring confidentiality. Data will only be assessed by qualified psychologists involved in follow-up treatment or interested in the study. The data will be analysed using SPSS.

Purpose

It is hoped that this study will help in the early identification of patients with panic disorder who are more at risk of comorbidity. Treatment may then be adjusted in order to improve outcome and possibly reduce relapse rates.

Reference List

American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed. Rev), Washington DC.

Barlow, D.H. (1988). *Anxiety and its disorders: The Nature and Treatment of Anxiety and Panic*. New York: Guilford Press.

Blashfield, R.K. (1990). Comorbidity and Classification in J.D. Maser & C.R. Cloninger (Eds.) *Comorbidity of Mood and Anxiety disorders* (pp. 61-82) Washington DC: American Psychiatric Press.

Boyd, J.H. (1986). Use of Mental Health Services for the treatment of Panic Disorder. *American Journal of Psychiatry*; 143, 1569-1574.

Brown, T.A., and Barlow, D.H. (1992). Comorbidity among Anxiety Disorders: Implications for treatment and DSM-IV. *Journal of Consulting and Clinical Psychology*. Vol 60, No 6. 835-844.

Chambless, D.L., Caputo, G.C., Bright, P., & Gallagher, R. Assessment of Fear of Fear in Agoraphobics: The Body Sensations Questionnaire and the Agoraphobic Cognitions Questionnaire. *Journal of Cons. and Clinical Psychology*. (1984) Vol 52, No6. 1090-1097.

Coryell, W., Endicott, J., Andreasen, N.C., Keller, M.B., Clayton, P.J., Hirschfeld, R.M.A., Scheftner, W.A., & Winokur, G. (1988). Depression and Panic Attacks: The significance of overlap as reflected in follow-up and family study data. *American Journal of*

Psychiatry, 145, 293-300.

Di Nardo, P.A., & Barlow, D.H. (1988). *Anxiety Disorders Interview Schedule - Revised (ADIS-R)*. Albany, NY : Phobia and Anxiety Disorders Clinic, State University of New York.

Di Nardo, P.A., Moras, K., Barlow, D.H., Rapee, R.M., Brown, T.A. (1993) Reliability of DSM-111-R anxiety disorder categories using the Anxiety Disorders Interview Schedule-Revised (ADIS-R). *Archives of General Psychiatry* ; 50:251-256.

Feinstein, A.R. (1970). The Pre-therapeutic classification of comorbidity in chronic disease. *Journal of Chronic Diseases*, 23, 455-468.

Frances, A., Widiger, T., Fyer, M.R. (1990). The Influence of classification methods on comorbidity. In J.D.Maser & C.R. Cloniger (Eds.) *Comorbidity of Mood and Anxiety disorders*. (pp. 41-59).

Klerman, G., (1990). Approaches to the phenomena of comorbidity. In J.D.Maser, & C.R. Cloniger (Eds.) *Comorbidity of mood and Anxiety disorders* (pp 13-37).

Koksal, F., & Power, K.G., (1990) Four Systems Anxiety Questionnaire (FSAQ): A Self-Report Measure of Somatic, Cognitive, Behavioural, and Feeling components. *Journal of Personality Assessment*, 54, 534-544.

Folkman, S., & Lazarus, R.S. (1985). If it changes it must be a process: Study of emotion and coping during three stages of a college examination. *Journal of Personality and Social Psychology*, 48, 150-170.

Lazarus,R.N., & Folkman, S., *Stress,Appraisal and Coping*. Springer, New York (1984).

Moras, K., & Barlow, D.H., (1992). Dimensional approaches to diagnosis and the problem of anxiety and depression. In W Fiegenbaum (Eds.) *Perspectives and Promises of Clinical Psychology*. (pp. 23-37). New York, Plenum Press.

Sanderson, W.C., Di Nardo, P.A., Rapee, R.M., & Barlow, D.H. (1990). Syndrome comorbidity in patients diagnosed with a DSM-111-R anxiety disorder. *Journal of Abnormal Psychology*, 99, 308-312.

Stein, M.B., Shea, C.A., & Uhde, T.W. (1989). Social phobic syndromes in patients with Panic Disorder: Practical and Theoretical implications. *American Journal of Psychiatry*, 146, 235-238.

Wallston,K.A., & Wallston,B.S.(1978). Development of the Multidimensional Health Locus of Control (MHLC) Scales. *Health Education Monographs* Vol 6 No 2.

Weissman, M., Klerman,G.L.,Markowitz, J.S., &Ouellette, R. (1989). Suicidal ideation and suicide attempts in panic disorder and attacks. *New England Journal of Medicine*, 321, 1209-1214.

Weissman, M.,Merikangas,K.R., (1986) The Epidemiology of anxiety and panic disorder. *Journal of Clinical psychiatry* (suppl.) 46, 11-17.

Wittchen, H.U., Essau,C.A., Von Zerssen, D., Krieg, C.J., & Zaudig, M., (1992). Lifetime and six-month prevalence of mental disorders in the Munich Follow-up Study. *European Archives of Psychiatry and Neurosciences*, 241, 247-258.

Zigmond, A.S., & Snaith, R.P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatr. Scand.* 67, 361-370.

**Panic Disorder : The strength and influence of selected variables as predictors of
Comorbidity**

Therese Carney

Department of Psychological Medicine

University of Glasgow

Running Head : Panic Disorder : Predictors of Comorbidity

Abstract

This study examines predictors of comorbidity amongst 42 panic disorder patients referred to a Primary Care service. A cross-sectional comparative analysis between 20 patients with panic disorder alone and 22 patients with panic disorder and comorbid conditions was undertaken. The comorbid group had significantly higher levels of psychopathology, symptom severity and avoidant type behaviour and cognition. They were more likely to engage in wishful thinking and self-criticism as coping strategies. In contrast, the panic only group were more likely to seek social support and engage in active problem solving. The greater incidence of anti-depressant medication use in the comorbid group was the only significant subject variable.

Introduction

The development of Taxonomic systems to classify mental health have helped to define disorders and improve clinical communication. The Diagnostic and Statistical Manual of Mental Disorders (Third Edition Revised, DSM-111-R; American Psychiatric Association 1987) has recently been updated to DSM-1V (1994) and has continued to inform on and define disorders on the basis of research studies and clinical expertise. One of the consequences of the development of this and other classification systems has been the interest generated in the area of comorbidity. The broad coverage of both psychological and psychiatric disorders and withdrawal of previous exclusionary hierarchy have increased reported rates and types of comorbidity (Brown & Barlow, 1992). This has been particularly noticeable for anxiety disorders as prior to DSM-111-R most of these disorders were excluded when presenting with major depression as this was considered to be of greater severity.

In the field of mental health, a comorbid condition is a symptom cluster which is concurrent with the symptoms of another disorder, but is not a typical feature of that disorder (Spitzer & Williams, 1983). Employing DSM-111-R criteria often results in assigning at least one comorbid disorder and in some cases several. In this situation it is necessary to assign a principal diagnosis for the purpose of treatment planning. This assignment can be made in any of the three ways outlined by Klerman (1990) in the literature review (Carney, 1995). The principal disorder as referred to in this paper is the condition which causes greatest life interference and prompts referral for treatment.

Comorbidity has important implications for research and clinical practice. The clinical course for a patient with a comorbid diagnosis may be quite different from that of a patient with a principal disorder alone e.g. (Stein, Shea & Uhde, 1989). Research on treatment outcome should account for the effects of comorbid disorders or symptoms on long and short term response to treatment. In addition, the type of adjustments which can be made to

enhance treatment efficacy should not overlook the contribution of comorbidity (Brown & Barlow, 1992). More detail on this issue has been reported in the literature review (Carney, 1995).

Much of the present interest on comorbidity is centered on the Anxiety Disorders. It has been estimated that 15% of the population will suffer from a DSM-111-R anxiety disorder at some time during their lives (Regier, Burke & Burke, 1990), and they are one of the major problems requiring medical and mental health input (Boyd, 1986). Comorbidity between different mental disorders is frequent in both clinical and epidemiological populations. Both the New Haven survey (Weissman, Myers & Harding, 1978), and the Epidemiological Catchment Area Programme (ECA) (Regier et al. 1990) reported that 30-80% of people with a principal anxiety disorder have at least one comorbid diagnosis. In clinical samples the results are similar. Sanderson, Di Nardo, Rapee & Barlow (1990) found that the overall rate of comorbidity was 70% which closely corresponds to that found by de Ruiter, Rijken, Garssen, Van Schaik & Kraaimaat (1989). High rates of comorbidity have also been reported between anxiety and mood disorders. A study by Barlow, Di Nardo, Vermilyea, Vermilyea & Blanchard (1986) reported that 65% of patients with a principal diagnosis within the anxiety-affective spectrum were given at least one comorbid diagnosis.

In addition to looking at rates of comorbid diagnosis, studies have also revealed consistent patterns of comorbidity. Moras, Di Nardo, Brown & Barlow (1991) reported that the principal diagnosis of generalized anxiety disorder and panic disorder were the categories associated with the highest comorbidity rates and simple phobia was associated with the lowest. When major depression and dysthymia were considered together as a mood disorder, this became the most frequently assigned additional diagnosis (Barlow et al. 1986; de Ruiter et al. 1989). Rates and patterns of comorbidity associated with individual anxiety disorders have also been examined and in particular with panic disorder. Between 35-93% of patients with panic disorder had a comorbid diagnosis (Stein et al. 1989; Brown

& Barlow, 1992). The large variation may be a reflection of the thresholds set to determine the presence of a disorder. A review of studies by Clark (1989) revealed that the mean percentage of patients with a principal diagnosis of panic disorder having past and present major depression was 67%. Despite the recent interest in comorbidity at both the general and more specific diagnostic level very little has been documented on issues other than reported rates and patterns. Less well known are the factors which predict the likelihood of having a comorbid diagnosis. Furthermore, why a particular patient is more at risk of having one or more comorbid diagnoses has not been systematically addressed.

This study selected panic disorder to ascertain which (if any) from a preselected set of variables were associated with comorbid conditions. The aim was to enhance present knowledge on the rates and patterns of comorbidity and in addition, to look at the indicators associated with comorbid conditions. This specific anxiety disorder was selected as it is associated with the highest comorbidity rates and examining one specific disorder has the advantage of increased practicality and the potential of leading to more meaningful conclusions. The variables included were selected as they have been found to be common features of panic disorder (Frances, Widiger & Fyer, 1990), or have been associated with additional psychological distress (Coryell, Endicott, Andreasen, Keller, Clayton, Hirschfeld, Scheftner & Winokur, 1988). As 'coping' is thought to be associated with mental disorders (Woodruff, Goodwin & Guze, 1974) and adaptation to stressful life events (Lazarus & Folkman, 1984), and 'locus of control' examines the degree of perceived control over health, these adjustment variables were included to investigate their role in predicting comorbidity. Knowledge of predictor variables could help identify those patients who are more at risk of comorbid disorders, and help target treatment more specifically to those variables associated with comorbid conditions. Treatment outcome studies could also comment on the impact of these variables on response to treatment. Patients referred to a Primary Care Psychological service with a principal diagnosis of panic disorder were allocated into one of two groups on the basis of presence or absence of comorbid conditions. A comparative analysis of potential predictor variables was made.

Method

Subjects

Subjects were 10 males and 32 females, recruited from the waiting list of a primary care psychological service. Only those whose principal diagnosis met the Diagnostic and Statistical Manual for Mental Disorders (3rd ed., rev; DSM-111-R ; American Psychiatric Association 1987) criteria for panic disorder and who were willing to participate were considered. Patients were excluded if they had a history of psychosis, current substance abuse or serious physical illness. They were then assigned to one of two groups : panic disorder alone or panic disorder with comorbid diagnosis. Twenty subjects (5 male and 15 female) presented with panic disorder alone and twenty two subjects (5 male and 17 females) presented with additional diagnoses. The demographics characteristics of the two samples are presented in **Table 1**.

Inspection of **Table 1** reveals that an individual with panic disorder and additional comorbid diagnosis is more likely to be married, a housekeeper with two children and on antidepressant medication. On the other hand, an individual with panic disorder alone is more likely to be unmarried, a non-housekeeper, with no dependants and on Benzodiazepine medication.

Table 1 Demographic characteristics

	Panic Disorder	Panic Disorder & Comorbid condition
Sex	Male 25%	Male 23%
	Female 75%	Female 77%
Mean Age	35.7 years ($\sigma=9.81$)	35.1 years ($\sigma=9.34$)
Marital Status	Single 30%	Single 18%
	Married 40%	Married 68%
	Divorced 25%	Divorced 14%
	Widowed 5%	Widowed 0%
Employment Status	Student 15%	Student 4%
	Employed 30%	Employed 23%
	P/T employed 10%	P/T employed 14%
	Unemployed 25%	Unemployed 18%
	Housekeeper 15%	Housekeeper 41%
	Retired 5%	Retired 0%
Dependants	No children 55%	No children 27%
	One child 15%	One child 14%
	Two children 25%	Two children 55%
	Three children 5%	Three children 4%
Antidepressant medication	Yes 0%	Yes 27%
Benzodiazepine medication	Yes 40%	Yes 27%

Measures

A. Structured Clinical Interview

- *Anxiety Disorders Interview Schedule - Revised (ADIS-R; Di Nardo and Barlow, 1988)*. This is a structured interview designed to assess in detail all anxiety and mood disorders. Psychometric studies indicate that adequate reliabilities can be obtained from this measure for DSM-111-R depressive and anxiety disorders (Di Nardo, Moras, Barlow, Rapee and Brown, 1993).

B. Questionnaires

- *Four Systems Anxiety Questionnaire (FSAQ; Koksal and Power, 1990)*. This is a 60 item self-report measure. It assesses anxiety on four components: somatic, cognitive, behavioural and affective.
- *Ways of Coping Checklist (Folkman and Lazarus, 1985)*. This scale was designed to assess categories of coping posited by Lazarus and Folkman's transactional model of stress. It categorises eight coping styles i.e. problem solving, cognitive restructuring, emotional expression, social support, passive avoidance, wishful thinking, self criticism and social withdrawal.
- *Body Sensations Questionnaire (BSQ; Chambless, Caputo, Bright & Gallagher, 1984)*. This 17 item scale comprises items concerning sensations associated with autonomic arousal.
- *Agoraphobic Cognitions Questionnaire (ACQ; Chambless et al. 1984)*. This 14 item scale comprises catastrophic ideation typically noted during exposure to anxiety provoking experiences including panic attacks.
- *Multidimensional Health Locus of Control Scale (Wallston and Wallston, 1978)*. This scale measures three dimensions of perceived control over health, one of which assesses internal locus of control and two of which assess perceived control of health from external sources e.g. powerful others and chance.

- *Hospital Anxiety and Depression Scale* (HAD; Zigmond and Snaith, 1983). This self report scale has been designed to measure anxiety and depression in both out patient and in patient populations.

Procedure

On initial interview all subjects underwent a Structured Clinical Interview for DSM-111-R criteria (AIDS-R; Di Nardo & Barlow, 1988) to establish diagnosis and to detail history. The interviewer was trained in the use of the ADIS-R by an experienced clinician and practise was gained before the study commenced. All interviews were conducted by the author and diagnosis was confirmed for each subject by the senior clinician who commenced treatment. Only those subjects with a caseness severity rating of 4 or over on the ADIS-R for both principal and comorbid diagnosis were included. If a principal diagnosis of Panic Disorder was given, the patients were given information on the study and asked if willing to participate. If agreeable, they were given the included questionnaires to complete in the department or to take them home and return by post. This contact took between 60-90 minutes. Relevant information was then written up in the casenotes and patients were given appointments to see a clinical psychologist for follow-up. In all no-one refused to take part in the study and 85% were willing to complete the questionnaires before leaving the department. Subjects were seen one day per week over a period of nine months with an average of four appointments per day. Eighty two appointments were made of which 12% cancelled and 10% had to be rescheduled. 15% failed to attend, 17% had a diagnosis other than panic disorder, 10% failed to reach severity criteria and 5% failed to return questionnaires. Of the 123 hours spent on initial interview 51% of this time was useful for the purposes of the study.

Results

Descriptive Analysis

An analysis of the rates of comorbidity revealed that 52% of patients with a principal diagnosis of panic disorder received at least one additional Axis 1 diagnosis. Of the comorbid group 95% had an additional anxiety disorder, 50% had an additional mood disorder and 45% had both an additional anxiety and mood disorder. Only 5% had a comorbid mood disorder alone but 32% had a comorbid anxiety disorder alone. The most common comorbid anxiety disorder in the sample was agoraphobia (77%), followed by social phobia (18%), generalised anxiety disorder (18%) and post traumatic stress disorder (9%). **Appendix 2.2** presents further information on the number and percentage of Panic Disorder patients to whom additional diagnoses were assigned.

Inferential Analyses

As a first stage in the analyses, difference testing between groups was conducted, on subject, symptomatic and adjustment measures. Other than taking antidepressant medication ($\chi^2 = 6.36, p < 0.03$), there were no significant differences found between the two groups on subject variables (see **Appendix 2.3**). For the symptom and adjustment variables the Mann-Whitney U Test was employed as its use is appropriate where the variables are measured only at the ordinal level, and on small samples where it could not be assumed that the underlying population was normally distributed. It was predicted that HAD anxiety and depression scores would be higher for the comorbid group (Coryell et al. 1988) so a one-tailed test was carried out on these variables. Otherwise two-tailed tests were conducted.

Table 2 shows those predictor variables which were tested significant. The Hospital Anxiety and Depression Scale, Bodily Sensations Questionnaire and all components of the Four Systems Anxiety Questionnaire proved to be strong discriminators of comorbidity i.e.

they were all significant at 1%. However, seeking social support was the only coping mechanism which attained the same level of confidence. The rest were significant at 5%. Those variables which tested insignificant are included in the complete data set in **Appendix 2.4**. These include the three dimensions of perceived control over health as measured by the Multidimensional Health Locus of Control Scale and the coping mechanisms of expressed emotion, passive avoidance and cognitive reappraisal.

Table 2 Results of between-groups difference testing on symptomatic and adjustment measures using the Mann-Whitney procedure

Variable	Comorbid Group Mean (SD) (n=22)	Panic Group Mean (SD) (n=28)	Mann-Whitney U	z ¹
Symptom Duration	1.87 (1.45)	0.93 (0.56)	133.5	-2.22 *
Severity Rating	5.26 (1.05)	4.53 (0.80)	137.0	-2.25 *
HAD Anxiety	16.54 (2.19)	13.85 (2.36)	87.5	-3.36 ****
HAD Dep	9.90 (4.12)	5.70 (2.02)	86.0	-3.41 ****
BSQ	3.03 (0.73)	2.34 (0.87)	98.5	-3.07 **
ACQ	2.94 (0.68)	2.29 (0.81)	118.5	-2.56 *
FSAQ_COG	68.79 (10.09)	43.2 (21.19)	68.0	-3.83 **
FSAQ-BEH	58.68 (16.36)	21.20 (12.29)	22.0	-4.99 **
FSAQ_FEE	63.38 (12.30)	41.00 (16.74)	62.5	-3.97 **
FSAQ_SOM	58.09 (12.48)	46.8 (11.85)	112.0	-2.72 **
WOC_SS	8.00 (3.4)	12.90 (5.00)	94.5	-3.17 **
WOC_PS	8.04 (3.00)	10.40 (4.00)	142.5	-1.96 *
WOC_WT	20.22 (3.14)	16.25 (5.70)	128.5	-3.32 *
WOC_SW	17.09 (6.20)	12.5 (5.24)	120.5	-2.51 *
WOC_SC	15.40 (7.20)	10.75 (7.80)	140.0	-2.02 *

* - $p < 0.05$; 2 tail ** $p < 0.01$; 2-tail **** $p < 0.01$; 1-tail

¹ Calculated from the U score adjusted for tied ranks.

A descriptive analysis of the ways in which both groups cope confirms the results (see **Appendix 2.5**). The panic only group resort to seeking social support and active problem solving, whereas the comorbid group are more disposed to engage in social withdrawal, wishful thinking and self criticism. The differential contributions of cognitive, affective, behavioural and somatic components can be seen in **Appendix 2.6**.

A Correlation Matrix was derived to investigate the degree of association between the variables. A high incidence of multicollinearity was found to be present (see **Appendix 2.8**) implying that many of the predictor variables are not independent of one another. This is not surprising given that many symptoms do not exist in isolation from each other. Accordingly, any measures designed to quantify the severity of any one symptom will necessarily be correlated with those which occur simultaneously.

The next stage in the inferential analysis was to determine more rigorously which variables account for the greatest variance between the two clinical populations. Regression Analysis cannot be utilised here, as the dependent variable is Categorical, not Continuous. Therefore, a Discriminant Analysis was performed to see if was possible to distinguish between comorbid and panic only subjects on the basis of the variables achieving significance (see **Table 2**). This techniques is used to classify subjects into distinct groups without knowing, in advance, the group to which they belong. The technique generates a discriminant function based on a set of subject attributes of known group membership. The same attributes of the unknown subjects are then combined with the discriminant function, and probability of group membership predicted. The quality of the function as a predictor is tested when the actual group memberships of the subjects is eventually ascertained.

The Mann-Whitney U significant variables were used to estimate the discriminant function, but was permitted to exclude those variables the discriminant routine deemed to be insignificant (see **Table 3**).²

² According to the calculated Wilks Lambda.

Table 3 U-significant variables remaining in analysis

Variable	Wilks' λ
FSAQ_BEH	0.36912
WOC_SS	0.29626
WOC_PS	0.27429
FSAQ_FEE	0.25153
WOC_SC	0.22977
ACQ	0.21982

On the basis of the discriminant function generated using the variables in **Table 3**, it was possible to clearly distinguish between the two clinical populations. In the first instance, 97% of the subjects used in generating the function had their group membership correctly 'forecast' (see **Table 4**). A Case-by-Case classification is given in **Appendix 2.7**.

Table 4 Classification Table (selected cases only)

		Predicted	
Actual		Comorbid	Panic
Comorbid	19	18 (94.7%)	1 (5.3%)
Panic	17	0 (0%)	17 (100%)

Percent of cases correctly classified : 97.22 %

In order to test the model more rigorously, 6 subjects (3 comorbid, 3 panic only) were excluded from the model construction phase. These were subsequently all classified correctly by the discriminant function (**Table 5**)

**Table 5 Classification Table for subjects
withheld from model construction**

	Predicted	
Actual	Comorbid	Panic
Comorbid	3 (100%)	0 (0%)
Panic	0 (0%)	3 (100%)

Discussion

This study examined a sample of primary care patients presenting with panic disorder (with and without comorbid diagnosis). In answer to the principal aim of the study, specific variables were found to predict comorbidity. These variables, and the answers to further questions raised were addressed below. Symptom severity, as measured by severity rating on ADIS-R and symptom duration, was significantly higher for the comorbid group than for the panic only group. There was evidence of greater psychopathology with comorbidity, as higher general levels of anxiety and depression were measured by the HAD. In addition, as anxiety is composed of the relatively independent cognitive, behavioural, affective and somatic components (Lang, 1971; Rachman & Hodgson, 1974; Zajonc, 1980) the differential contributions of these components for both groups were examined. The comorbid group had significantly higher scores on all components of the FSAQ. The higher cognitive component was also reflected by higher catastrophic ideation found on the ACQ. In addition, this group were more likely to engage in wishful thinking and self-criticism in coping with their condition(s). The higher somatic component on the FSAQ for the comorbid group was associated with high anxiety provoking autonomic arousal as found on the BSQ.

Avoidance behaviours and cognitions which are specific components of panic disorder were found to be significantly associated with comorbidity in this study. Avoidant type behaviour as measured by the FSAQ was higher in the comorbid group, and coping strategies included social withdrawal. By contrast, the panic disorder group were significantly more likely to seek social support and engage in active problem solving. There were no differences between the two groups in terms of most subject variables, locus of control and the use of some coping strategies such as, cognitive reappraisal, expressed emotion and passive avoidance.

There has been some suggestion in the literature that agoraphobic avoidance is a secondary result of more severe panic disorder (Barlow, 1988) and that patients with panic disorder suffer from a less severe condition (Thyer, Himle, Curtis, Cameron & Nesse, 1985). This study would seem to support this finding. The panic only group did have less severe symptomology and 77% of the comorbid group had an additional diagnosis of agoraphobia. Avoidance behaviour was only a minor determinant of depression within panic disorder in a recent study (Ball, Otto, Pollack & Rosenbaum, 1994), so the relationship between this comorbid condition and avoidance behaviour is less clear cut.

An additional mood disorder was found in 50% of the comorbid group, a result which has also been supported by previous research (Clark, 1989). It has been hypothesised that the co-occurrence of panic disorder and depression is the result of a demoralization response to panic attacks. The unexpected and uncontrollable characteristics of panic attacks may predispose to helplessness and depression (Stein et al. 1990). In addition, ruminative coping strategies have been implicated in the maintenance of depressed mood (Nolen-Hoeksema, 1991) and the comorbid group were significantly more likely to engage in wishful thinking and self criticism.

All four response modalities as measured by the FSAQ were significantly higher for the comorbid group and there was more agreement between the modalities (see **Appendix 2.6**). In contrast, the panic group had a noticeable lack of agreement between the modalities with a much lower behavioural component than somatic or cognitive. It is difficult to determine from a cross-sectional study how and when the change occurs, but a possible explanation can be drawn from what is already known. The cognitive model of panic (Clark, 1986a) states that panic occurs because of catastrophic misinterpretation of bodily sensations. This is the first stage in panic disorder and does not appear to change much with comorbidity, as the somatic response modality has the closest mean scores for both groups. However, in the presence of comorbid conditions, avoidance behaviour and cognitive distortions may increase (as in this study), increasing the discrepancy between the two groups in behaviour and cognitive response modalities.

It has been suggested that the presence of maladaptive coping strategies might determine whether, when and how severely a psychiatric disorder is expressed in vulnerable individuals (Roy-Byrne, Vitaliano, Cowley, Luciano, Zheng, & Dunner, 1992). In this study the panic only group were more likely to use adaptive coping strategies e.g. problem solving and seeking social support, whereas the comorbid group were more likely to use maladaptive strategies e.g. self-criticism, wishful thinking and social withdrawal.(see **Appendix 2.5**). The practice of seeking social support with panic disorder has been found previously. Borden, Clum, Broyles & Watkins (1988) reported that patients with panic were more likely to seek professional help and talk to another person. Coping is viewed as a process that changes according to the degree of distress associated with the stressor (Folkman and Lazarus, 1980). Perhaps the change from active adaptive coping as seen with the panic only group to maladaptive coping in the comorbid group was a result of greater severity of symptoms and psychopathology. However, a longitudinal study demonstrated a causal relationship between emotion focused coping strategies e.g. wishful thinking and an increase in symptoms of anxiety and depression (Bolger, 1990).

The variables identified as predictors of comorbidity help to determine individuals with primary panic disorder who are more likely to have an additional disorder. However, the variables employed were self-selected thus raising the possibility that better predictors (should they exist) may have been excluded. In addition, the predictor variables are not in themselves primary explanations of why someone is more likely to develop a comorbid diagnosis. It is implicit in this study that these variables are associated with comorbidity and no causal relationship is implied. The variables employed here are essentially 'static', in the sense that they were non-repeated measures taken at one point in each subject's life. A clearer picture may be gained by examining the dynamic behaviour of these variables over time by employing a longitudinal research design. The small sample size did not permit meaningful analysis of the effects of individual comorbid conditions. Future research could gain additional information by identifying predictors for specific comorbid conditions within panic disorder and not just comorbidity in general.

References

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders* (1V ed.), Washington DC.

American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders* Third Edition Revised, Washington DC.

Ball, S.G., Otto, M.W., Pollack, M.H., Rosenbaum, J.F. (1994). Predicting prospective Episodes of Depression in patients with Panic Disorder: A longitudinal Study. *Journal of Consulting and Clinical Psychology*. Vol - 62, No 2. 359-365.

Barlow, D.H. (1988). *Anxiety and its disorders*. New York: Guildford Press.

Barlow, D.H., Di Nardo, P.A., Vermilyea, B.B., Vermilyea, J.A., & Blanchard, E.B. (1986). Comorbidity and depression among the anxiety disorders: Issues in classification and diagnosis. *Journal of Nervous and Mental Disease*, 174, 63-72.

Bolger, N. (1990). Coping as a personality process: A prospective study. *Journal of Personality and Social Psychology*, 59, 525-537.

Borden, J.W., Clum, G.A., Broyles, S.E., & Watkins, P.L. (1988). Coping strategies and panic. *Journal of Anxiety Disorders*, 2, 339-352.

Boyd, J.H. (1986). Use of Mental Health Services for the treatment of Panic Disorder. *American Journal of Psychiatry*; 143, 1569-1574.

Brown T.A., and Barlow D.H. (1992). Comorbidity among Anxiety Disorders: Implications for treatment and DSM-1V. *Journal of Consulting and Clinical Psychology*. Vol 60. No.6. 835-844.

Chambless, D.L., Caputo, G.C., Bright, P., & Gallagher, R. Assessment of Fear of Fear in Agoraphobics: The Body Sensations Questionnaire and the Agoraphobic Cognitions Questionnaire. *Journal of Cons. and Clinical Psychology*. (1984) Vol 52, No6. 1090-1097.

Clark, D.M. (1986a). A cognitive approach to panic. *Behaviour Research and Therapy* 24, 461-70.

Clark, L.A.(1989). The Anxiety and Depressive disorders: Descriptive psychopathology and differential diagnosis. In P. Kendall & D. Watson (Eds), *Anxiety and Depression: Distinctive and overlapping features* (pp. 83-129). San Diego, CA: Academic Press.

Coryell, W., Endicott, J., Andreasen, N.C., Keller, M.B., Clayton, P.J., Hirschfeld, R.M.A., Scheftner, W.A., & Winokur, G. (1988). Depression and Panic Attacks: The significance of overlap as reflected in follow up and family study data. *American Journal of Psychiatry*, 145, (3), 293-300.

de Ruiter, C., Rijken, H., Garssen, B., Van Schaik, A., & Kraaimaat, F. (1989). Comorbidity among the anxiety disorders. *Journal of Anxiety Disorders*, 3, 57-68.

Di Nardo, P.A., & Barlow, D.H. (1988). *Anxiety Disorders Interview Schedule - Revised* (ADIS-R). Albany, NY : Phobia and Anxiety Disorders Clinic, State University of New York.

Di Nardo, P.A., Moras, K., Barlow, D.H., Rapee, R.M., & Brown, T.A. (1993). Reliability of DSM-111-R anxiety disorder categories using the Anxiety Disorders Interview Schedule- Revised (ADIS-R). *Archives of General Psychiatry*; 50: 251-256.

Folkman, S., & Lazarus, R.S. (1985). If it changes it must be a process: Study of emotion and coping during three stages of a college examination. *Journal of Personality and Social Psychology*, 48, 150-170.

Folkman, S., & Lazarus, R.S. (1980). An analysis of coping in a middle-aged community sample. *Journal of Health and Social Behaviour*, 21, 219-239.

Frances, A., Widiger, T., Fyer, M.R. (1990). The Influence of classification methods on comorbidity. In J.D.Maser & C.R. Cloninger (Eds.) *Comorbidity of Mood and Anxiety disorders*. (pp. 41-59).

Klerman, G., (1990). Approaches to the phenomena of comorbidity. In J.D.Maser, & C.R. Cloninger (Eds.) *Comorbidity of mood and Anxiety disorders* (pp 13-37).

Koxsal, F., & Power, K.G. (1990). Four Systems Anxiety Questionnaire (FASQ): A Self-Report Measure of Somatic, Cognitive, Behavioural and Feeling components. *Journal of Personality Assessment*, 54, 534-544.

Lang, P.J. (1971). The application of psychophysiological methods. In Garfield, S.L. & Bergin, A.E. (Eds), *Handbook of psychotherapy and behaviour change*. New York: Wiley.

Lazarus, R.N., & Folkman, S. (1984). *Stress, Appraisal and Coping*. Springer, New York.

Moras, K., Di Nardo, P.A., Brown, T.A., & Barlow, D.H. (1991). *Comorbidity and depression among the DSM-111-R anxiety disorders.*

Nolen-Hoeksema, S.K. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, 100, 569-582.

Rachman, S. & Hodgson, R (1974). Synchrony and desynchrony in fear and avoidance. *Behaviour Research and Therapy*, 17, 311-318.

Regier, D.A., Burke, J.D., & Burke, K.C. (1990). Comorbidity of affective and anxiety disorders in the NIMH Epidemiologic Catchment Area Programme. In J.D. Maser & C.R. Cloniger (Eds.) *Comorbidity of mood and anxiety disorders* (pp. 113-122). Washington DC: American Psychiatric Press.

Roy-Byrne, P.P., Vitaliano, P.P., Cowley, D.S., Luciano, G., Zheng, Y., & Dunner, D.L. (1992). Coping in Panic and Major Depressive Disorder - Relative Effects of Symptom Severity and Diagnostic Comorbidity. *The Journal of Nervous and Mental Disease*, Vol 180, No 3, 179-183.

Sanderson, W.C., Di Nardo, P.A., Rapee, R.M., & Barlow, D.H. (1990). Syndrome comorbidity in patients diagnosed with a DSM-111-R anxiety disorder. *Journal of Abnormal Psychology*, 99, 308-312.

Spitzer, R.L., and Williams, J.B.W. (1983). Proposed revisions in the DSM-111 classification of anxiety disorders based on research and clinical experience. Paper presented at the conference, DSM: An interim appraisal, sponsored by the American Psychiatric Association, Washington D.C.

Stein, M.B., Shea, C.A., & Uhde, T.W. (1989). Social phobic syndromes in patients with Panic Disorder: Practical and theoretical implications. *American Journal of Psychiatry*, 146, 235-238.

Thyer, B., Himle, J., Curtis, G., Cameron, O. & Nesse, R. (1985). A comparison of panic disorder and agoraphobia with panic attacks. *Comprehensive Psychiatry*, 26, 208-214.

Wallston, K.A., & Wallston, B.S. (1978). Development of the Multidimensional Health Locus of Control (MHLC) Scales. *Health Education Monographs* Vol 6 No 2.

Weissman, M.M., Myers, J.K., & Harding, P.S. (1978). Psychiatric disorders in a US urban community. *American Journal of Psychiatry*, 135, 459-462.

Woodruff, R.A., Goodwin, D.W., & Guze, S.B. (1974). *Psychiatric Diagnosis*. New York: Oxford University Press.

World Health Organisation (1992). *International Classification of Disease*. 10th Edition.

Zigmond, A.S., & Snaith, R.P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatr. Scand.* 67, 361-370.

Zajonc, R.B. (1980). Feeling and thinking: Preferences need no inferences. *American Psychologist*, 35, 151-175.

**Improving Health in Scotland guided by "Framework for Action". An evaluation of
Psychologists health attitudes, behaviour and role.**

**Therese Carney, B.Sc. (Hons)
Trainee Clinical Psychologist
University of Glasgow**

Abstract

Improving Health in Scotland is one of the main goals of the "Framework for Action" document (1). As the role of the Clinical Psychologist in fulfilling this goal is potentially a key one, this evaluation project targeted fifty qualified and trainee clinical psychologists to examine their health behaviours, attitudes and role in Health Promotion. The results reveal that eighty per cent of this group were unfamiliar with the Framework for Action document and its goals, but were nevertheless involved in health promotion. Communication and education about current policies will need to target key workers more effectively if their potential as facilitators of Health Promotion is to be utilized.

Introduction

There have been many changes in the National Health Service (NHS) in the past few years not least of which has been the emphasis placed on health promotion. The World Health Organisation (WHO) has taken a leading role in promoting health in the past decade and stated at the 30th World Health Assembly (2) that :

'the main social target of Governments and WHO in the coming decades should be the attainment of all citizens of the World by the year 2000 of a level of health that will permit them to lead a socially and economically productive life'

This was the beginning of what has become known as 'The health for all movement' from which Regional Strategy on this issue evolved. In September 1991 the NHS in Scotland produced the document 'Framework for Action' (1), in which three goals for provision of service were identified. They included :

- Improving health in Scotland
- Improving care for patients
- Empowering staff

- and were accompanied by a programme of action for putting them into practice. A survey was conducted by the NHS (3), to obtain feedback on progress and the following information was obtained. 69 per cent of NHS staff expected things to change as a result of Framework for Action and 56 per cent thought that it would help staff be clearer about their tasks. In addition, the highest priority among staff for improving health in Scotland was to adopt a healthier lifestyle personally. It is with this goal 'improving health in Scotland' that this evaluation project is concerned. As Psychologists working within this organisation it is important to be familiar with current literature and practice in order to participate in health promotion.

Health means different things to different people. Being healthy may just mean not being ill but there are more positive ways in which the general public think of health. For example, Williams (4) found that elderly Scottish people saw three major dimensions of health; the absence of illness and disease, a dimension of strength- weakness, and being fit to do the jobs expected. Attitudes towards health can be linked with people's social and cultural situations and can be passed on through generations, Calnan (5). To health professionals, health may be viewed more objectively as the WHO (6) propose: ‘.. a conception of health as the extent to which an individual or group is able, on the one hand, to realise aspirations and satisfy needs; and, on the other hand, to change or cope with the environment. Health is, therefore, seen as a resource for everyday life, not the objective of living; it is a positive concept emphasising social and personal resources, as well as physical capacities’.

Health in Scotland is improving annually for a number of reasons. The overall standard of living has improved with greater economic prosperity and better social and public health measures. The provision of drinking water, more housing and safe sewerage systems have gone a long way towards reducing overcrowding, spread of infection and related illness. Information on diet and nutrition and the effects of overindulgence in alcohol and tobacco have raised awareness of personal health. Immunisation has also played a major part with declining rates of polio and rubella which added to infant mortality and morbidity rates. Improved medical and surgical treatment techniques and the introduction of antibiotics have all contributed to improved National Health.

Despite the improvements many people still die before the age of 65 years and the quality of life in later years may be impaired by poor health. Compared with other European countries Scottish men and women have the worst death rates from coronary heart disease; Scottish women have the worse record for deaths from lung cancer and Scottish men have the shortest life expectancy, HMSO (1). For this reason national targets were set which are to be achieved by the year 2000 as recommended by the WHO. They include:

- 30 per cent cut in deaths from heart disease in the under 65's
- 15 per cent cut in Cancer deaths in the under 65's
- 30 per cent cut in smoking in the 12-14 year old and 20 per cent in the 25-65 year group
- 20 per cent cut in drinking above recommended limits

In reaching these targets it is important that everyone in the NHS plays a role. Each Board/Trust has its own policies regarding how these targets are reached thus achieving better health for the people of their district. Most set local targets and run programmes to achieve them and have flexibility in determining their own health priorities according to local needs. They may decide to concentrate on improving cardiac rehabilitation, for example, or whatever problem presents local health concerns. It is hoped, however, that they reflect national priorities. As stated in Framework for Action those people involved in primary care have a key role to play as they are in regular contact with the community.

The unique profile of skills which a clinical psychologist possesses, Management Advisory Service (7), should ensure that they play a key role in improving health in Scotland. This can be seen in more detail by taking a closer look at their potential role within the Health Board in putting objectives outlined in Framework for Action into practice.

Assessing local health. This could involve primary care psychologists who are in regular contact with the community and other primary care workers. As such they will have access to local health concerns and be familiar with local health issues.

Ensuring health promotion. This requires good health educators. Psychologists can promote positive psychological well-being on a preventative, treatment or intervention level. They are also potentially good communicators, with effective presentation skills, which could help in establishing rapport and allow the easy flow of health information to target groups or individuals. They may also act as consultants in passing on these skills to other health care workers.

Monitoring and Review. Framework for Action states 'Boards need to become discriminating fathers and processors of data. They need to keep checking the effectiveness of their systems - and of the information that results'. Psychologists as 'scientist-practitioners' are familiar with clinical audit, i.e. evaluating an aspect of a service using measurable indicators of quality, then making changes to the service and conducting a follow-up, Dixon (8). They are also familiar with research and data analysis and are thus in a position to become involved in monitoring and review on an individual or consultancy role.

The above issues refer to aspects of improving health in Scotland which Psychologists can achieve within their work should they choose to do so. Framework for Action, however, takes one step further and states that 'everyone in the NHS can set a good example in the pursuit of good health. Everyone, wherever they work, has a role to play in health promotion, whether formally as part of their job, or informally in their social, business or domestic contacts'. Everyone should be seen to promote good health personally by eating and drinking sensibly, not smoking and taking regular exercise. The individual should also encourage friends, family and colleagues to lead a healthy life.

As Clinical Psychologists work within the NHS, and as previously identified they could be key players in the promotion of good health, it is necessary for them to be familiar with the goals and standards set in Framework for Action (in Scotland). Following on from the finding that the highest priority among staff for improving health in Scotland was to adopt a healthier lifestyle on a personal basis, it would be of interest to identify the health behaviour and attitudes of psychologists as a group by using a cross-sectional approach. It would also be of benefit to assess the role they play in setting a personal example of good health and of actively encouraging others to adopt a healthy lifestyle.

Method

Subjects

A sample of qualified psychologists from the West of Scotland were invited to participate. Subjects from Greater Glasgow, Argyll and Clyde, Lanarkshire and Ayrshire and Arran were included. A cross section of specialities were included e.g. adult mental health, child health and learning disability. All current Trainees on the Glasgow University Postgraduate Clinical Psychology course were also invited to participate. Sixty five subjects were included in the study and fifty (77 per cent) returned completed questionnaires. Of these twenty five were clinical psychology trainees, 15 were qualified and 10 declined to disclose their designation

Materials

A Questionnaire, (see **Appendix 3.2**), together with an accompanying information leaflet and stamped self-addressed envelope were posted to all qualified psychologists or placed in the department of Psychological Medicine in a sealed envelope for all trainees. The questionnaire consisted of 14 closed questions closely reflecting the goals stated in the Framework for action document. Each participant was invited to tick the appropriate box or add additional information when necessary. This style of questionnaire can invite passive participation so to check for this the relevant questions asked respondents to display their knowledge of the question, e.g. question 14. Respondents were aware that their questionnaires were anonymous and that replies would be treated in confidence.

Design and Procedure

All completed questionnaires were coded and entered onto a spread sheet and used to produce summary statistics. A spreadsheet was used rather than a statistical package as analysis involved calculating percentage respondents for each category only. Each separate category response was identified by using a unique number code. Different combinations of response were also identified using this method.

Results

The results were reported in three sections, namely : Health attitudes, Health behaviours and Health promotion. The summery statistics are presented in the following tables and discussed in the final discussion section.

Health attitudes

What is good health? ¹	86% Manageable stress levels 86% Good quality of life 80% Physical Fitness 78% Healthy Diet 36% Following Recommended Guidelines
How often think about health ?	44% Weekly 28% Monthly 26% Daily 2% Not at all
Influences on attitudes about health ²	88% How you feel 68% Medical opinion 58% Literature / video 54% TV 36% Family History 30% Parents/family 22% General advertising 6% additional information

¹ Other ideas on good health included, adequate income and accomodation, positive psychological wellbeing, ability to choose, medical and physical status, absence of poor health.

² Other identified influences on health included; analysis of relationships between poverty and health, through own academic work, research, noticing others around especially those with less fortunate lifestyles.

Health Behaviours

Present lifestyle	4% Very healthy 80% Moderately healthy 16% unhealthy 0% don't know
Regular health Checks	68% Yes 32% No
Smokers	90% No 10% Yes
Alcohol intake	64% at or under recommended level 36% over recommended level
Exercise Rates	16% over three times per week 44% up to three times per week 40% less than three times per week
Eating Habits	10% always healthy 84% moderately healthy 6% unhealthy

Health Promotion

Familiar with Framework for Action	80% No 20% Yes
Familiar with NHS policies (one respondent pointed out that there was no stress policy within the Trust)	Smoking 90% Yes Alcohol 74% Yes Food 50% Yes Stress 24% Yes
Role to play in health promotion	88% Yes (70% formally at work) (13.6 % formally & socially) (6.8% formal & domestic) (9% formally & domestic & social) 12% No
Actively encourage others	66% Yes (64% family only) (6% friends only) (0% colleagues only) (15% family & friends) (6% family & colleagues) (9% all) 34% No
Present an example of good health	70% Yes (20% at work only) (0% socially only) (18% at home only) (20% at work & home) (9% at work & socially) (34% at work, socially & at home) 30% No

Discussion

The results of the evaluation project reveal some interesting outcomes. There are a number of dimensions to the concept of health of which the most obvious is physical health. However, health also involves mental health, emotional health, social health and spiritual health. The two most popular aspects of good health identified by this group of psychologists refer to both mental and emotional health and not one of the more obvious physical aspects. This may be a reflection of the priorities of their work. Seedhouse (9), in emphasising the holistic view of health, stated that working for health is linked closely to improving people's quality of life which was again recognised as an important aspect in good health. It has been proposed that health is taken for granted and only considered when illness or health problems are interfering with people's lives, Ewles & Simnett (10). However, this group who considered themselves moderately healthy, thought about their health on a regular basis, with close to half considering their health on a weekly basis.

Many factors can influence personal health beliefs, and the single most important identified influence on health attitudes in this population was how a person feels in themselves. Interestingly, the second most important influence was medical opinion, as there has been an increasing awareness in the past decade that medicine, as a professional practice, has had little effect on the nation's health, McKeown (11). It has also been argued that control over health and illness has been taken away from people themselves who then become dependent on external sources for treatment. Perhaps this result reflects the fact that all psychologists involved in this evaluation project worked within the health service, which is still a treatment and medical care system (in the main) for people who are ill, and not primarily the means by which health is promoted. Therefore, medical opinion would be reinforced almost as a daily influence on health.

Looking at the health behaviours of this group it can be seen that the majority (80 per cent) of respondents felt that their life was moderately healthy. However, some aspects of health behaviour fell short of the recommended guidelines. Over one third of this sample drink above the safe levels, don't get enough exercise and don't have regular health checks. Eating habits were considered to be healthy in the main and the majority were non smokers. Therefore, some respondents who felt they were leading a healthy life do not in fact carry this out in practice. Choosing health behaviour does not automatically lead to practising it. Changes such as taking more exercise or stopping smoking are hard work and may in themselves be stressful. Therefore, guidelines alone may not be enough to make the change. People need support and facilitation for active decision-making which involves committing time and effort to understanding the factors which influence health choices and behaviour. This in turn leads to making considered decisions and actions, which have more chance of success than behaviour which develops without conscious decision-making, Tones (12).

The health promotion section produced the most surprising result which identified a large number, (80 per cent), of psychologists who were not familiar with the framework for action document. One could look at this positively, when despite this, 88 per cent think that they have a role to play in health promotion, and 70 per cent think that they should set an example of good health. Perhaps to psychologists, this document has put into words what the majority are practising professionally. However, it has major implications for health promotion, when such a large proportion of key health professionals are unaware or unfamiliar with NHS policies on this issue. Firstly, psychologists need to know what the standards are in order to meet them both personally and professionally and secondly, to influence policy on health promotion it would be useful to have a working knowledge of current codes of practice. From the Health Board point of view, it is financially and clinically disadvantageous to have potential key players in health promotion (by means referred to previously) unfamiliar and therefore redundant as regards this issue.

Health promotion in the workplace can result in considerable improvements in health. The NHS has taken on a broad concept of health at work including giving priority to issues such as smoking, alcohol, food and stress. Policies regarding these issues have been based on WHO guidance, WHO (13). The majority of psychologists are familiar with NHS policies on smoking and alcohol, but only half are familiar with policies on food and less than a quarter on stress policies. Indeed as one person remarked, there were no policies on stress in one local trust. Peoples health choices are usually made in the context of their environment, which if conducive to a healthier lifestyle will encourage people to choose the healthier alternatives.

This evaluation study has identified one group of professionals who are unfamiliar with the current standards of health promotion within the NHS in Scotland. Their influence and involvement on a personal and professional level falls short of what could be achieved. Communication and education about health promotion and current policies may need to improve before participation becomes a reality. It would seem also that the gaps in the development of some policies within local trusts need to be redressed and certainly in making them familiar to staff. Some professionals may have too narrow a concept of health and what is meant by health promotion, leading to individual behaviour change only and a failure to encourage others. In addition, resource constraints may hinder professionals from achieving their potential in health promotion. There is an acknowledged shortage of clinical psychologists and workloads may not be conducive to active involvement in health promotion.

References

1. Framework for Action. HMSO (1991).
2. World Health Organisation (WHO) resolution WHO30.43, quoted in : WHO Regional Office for Europe .Targets for Health for All. Copenhagen, Denmark : WHO, p1, (1977).
3. Framework for action Survey. HMSO (1993).
4. Williams, R. Concepts of health: an analysis of lay logic. *Sociology* (1983): 17, 185-204.
5. Calnan, M. Health and Illness - The Lay Perspective. London: Tavistock Publications (1987).
6. World Health Organisation. Health Promotion: a WHO Discussion Document on the Concepts and Principles. Reprinted in: *Journal of the Institute of Health Education*, 23(1), (1985).
7. Management Advisory Services to the NHS. Review of Clinical Psychology Services, (1989).
8. Dixon, N. Medical audit assistant manual. Hampshire: Health Care Quality Quest, (1991).
9. Seedhouse, D. Health: The Foundations for Achievement. Chichester : Wiley ,(1986).
10. Ewles T, & Simnett S. Promoting Health, A Practical Guide. Scutari Press. London, (1992).

11. McKeown, T. *The Role of Medicine: Dream, Mirage or Nemesis*. Oxford; Blackwell, (1979).

12. Tones K, Tilford, S., Robinson, Y. *Health Education - Effective and Efficiency*. London: Chapman and Hall. Ch 3, (1990).

13. World Health Organization. *Health Promotion for working populations. Report of a WHO Expert Committee; Technical Report Series No 765*. Geneva : WHO, (1988).

Group Management of Chronic Pain

An Outcome Study

Therese Carney

Trainee Clinical Psychologist

Glasgow University

Running Head: Group Management of Chronic Pain

Abstract.

Responding to recent research which suggests that multidisciplinary pain management programmes are a successful way to manage benign chronic pain, a small pilot study was undertaken at a District General Hospital. Outcome measures included depression, anxiety, activity level, coping skills, pain related cognition, impact of pain on daily living and patient satisfaction with the programme. Five measures showed statistically significant improvements, with all other measures highlighting a trend towards improvement. Patient satisfaction with the programme was also high. In conclusion, this small pilot study gives reason for optimism for developing this mode of intervention and paves the way for further study.

Introduction

The management of chronic pain has always presented challenges mainly due to changing views on causal models e.g. psychological or organic, linear or multi-dimensional explanations. The present challenge however, is how best to manage chronic pain given the growing numbers of referrals with this condition. Rigge (1990) surveyed more than 1,000 adults in Britain and found that 11.5% had chronic pain, of whom 55% were unable to work or lead a normal life because of pain. In addition, 17% had retired, 6% were housebound by pain and 70% remained in pain despite taking analgesics. A further study by the Department of Health (1994) found that of 6,000 adults, 40% had experienced back pain in the past year of which 10% were unable to lead a normal life. In addition 3% had spent at least one day in the previous four weeks resting or lying down because of back pain.

The cost to industry is also noteworthy. Frank (1993) found that back pain was the biggest single cause of sickness absence from work, with 52.6 million days lost in 1988-9 throughout Britain. This accounted for 12.5% of the total, and the estimated cost was £2,000 million. Furthermore, back pain accounted for 2 million general practitioner consultations annually, 300,000 hospital outpatient consultations and 100,000 hospital inpatient episodes. From these figures it has been estimated that in the average health district of 250,000 population there will be up to 1,000 people moderately to severely affected by back pain.

In the district served by this health trust, 119 new patients with chronic pain problems were seen in the first six months of 1994 at the pain relief clinic. They were all seen on an individual basis by a pain consultant and referred on when appropriate to psychology and physiotherapy. This type of intervention obviously means commitment in terms of cost

and professional time. It also relies heavily on a medical model of chronic pain which has now been replaced in favour of a psychosocial model.

In America, Fordyce et al., (1968) were the first to apply the behavioural model to pain, on which subsequent operant pain management programmes were based. This changed the focus away from the traditional medical models of pain and opened the door for psychological intervention. These multidisciplinary programmes however, were often targeted towards in-patient populations with settings often on physical rehabilitation or psychiatric wards (Pither and Nicholas, 1991). The linear causal model on which these groups were based ignored variables in the patients real life setting in which pain is embedded. Subsequently outpatient pain management programmes were also established partly on the grounds of cost, but also with the knowledge that gains were maintained and programmes were also effective (Vasudevan et al., 1985).

Melzack and Wall (1965) inspired cognitive approaches to the treatment of pain with their gate-control theory which established a role for cognitive-evaluative processes in the modulation of pain. Programmes based on cognitive interventions to control pain aim to increase patient's self-efficacy (Nicholas et al., 1991) and are modelled on research which shows that a patient's appraisal of a difficult situation and beliefs about his or her ability to cope with it influence the experience of stress (Bandura, 1977). At present, programmes generally combine cognitive and behavioural approaches to help patients restructure the way they think about their pain as well as to increase their day to day activity.

Multidisciplinary programmes are usually run in group format. This is more efficient in terms of number of patients seen in a limited time and is more suitable to the integration of multidisciplinary treatments that take place within one session. It also facilitates encouragement and support from other patients and reduces feelings of isolation. The aim

of pain management programmes is not to cure the pain, and it is misleading to use this as a measure of success. They do aim to restore normal function despite the pain, and gains have been made. Published outcome studies derive in the main from America and suggest that 60-70% of patients will make beneficial changes. A meta-analysis of 65 studies of 3089 patients with back pain supported the conclusion that pain management programmes are efficacious (Flor et al.,1992). Multidisciplinary approaches were superior to no treatment, conventional medical therapy and physical therapy, although the majority of studies were conducted without controls. Even when controls were used they were frequently inappropriate.

A few such programmes have been developed in Britain. Skinner et al., (1990) reported on a cognitive-behavioural approach run in a district general hospital. It took place over one afternoon per week for seven weeks and showed significant improvements in mood, coping skills, physical disability and analgesic consumption despite less clear cut improvement in pain intensity. Wells and Miles (1991) reported that their programme reduced drug intake by 50%, doubled the activity level of more than half the patients and returned 29% to employment despite time off work of 2.7 years before attendance.

The current climate within the local NHS trust encourages provision of high quality services based on current research, and the opportunity to use innovative methods to deal effectively with increasing referral rates. This current pilot group was run to assess effectiveness of the group format and to encourage funding for a larger scale pilot study on multidisciplinary group management of chronic pain.

Method

Subjects - Patients were selected from a population of chronic benign pain sufferers referred to the outpatient pain relief clinic of a local general hospital. Selection criteria included age between 18 and 70 years, with major continuing disability from chronic benign pain, despite having had appropriate investigations and treatment. They also had to be fluent in English, able to make their own way to the hospital, without past or present serious mental illness and not actively involved in litigation. Ten patients were originally selected to commence the programme - two however, cancelled before starting. One subject was returning to work and did not have the time to commit and the second had to decline due to a personal bereavement. Eight patients commenced the programme and seven completed it. Attendance rates for this core group ran at nearly 100%. All suffered from back pain and seven had a definite diagnosis. The eighth patient could trace pain origins to pregnancy and epidural analgesia received at delivery. **Table 1** provides a summary of the group's characteristics.

Table 1 Demographics

Sex	Male	43%
	Female	57%
Age-Mean (σ)		44.5 (11.1)
Employment Status	Employed	0%
	Unemployed	100%
Disability Benefit		43%
Marital Status (Married)		100%
Diagnosis	Back Pain	100%
Duration of Pain - mean/sd		5/4.7
Previous Therapies	Medication	100%
	TNS	71%
	Epidural	43%
	Physiotherapy	43%
	Hydrotherapy	29%
	Acupuncture	14%
Previous Specialists	Corset/Plasters	14%
	Pain Consultant	100%
	Orthopaedic Surgeon	43%
	Physiotherapist	43%
	Psychologist	14%

Measures

All participants were interviewed by the pain consultant and clinical psychologist before commencing the group. A range of measures was used to evaluate the multidimensional nature of pain.

1. The Hospital Anxiety and Depression Scale (HAD ; Zigmond and Snaith 1983).

Included to obtain a measure of anxiety and depression in this outpatient population. Such emotional disorders often occur in patients with chronic pain.

2. General Health Questionnaire (GHQ ; Goldberg 1978). Aimed at detecting psychiatric disorders in general medical outpatients among others. The questionnaire looks at four main areas - depression, anxiety, hypochondriasis and behaviour.

3. West Haven Yale Multidimensional Pain Inventory (MPI ; Kerns, Turk And Rudy 1985). This questionnaire is specifically designed to assess chronic pain from a cognitive-behavioural perspective. The MPI contains 52 items and has 13 empirically derived scales arranged in three parts. These include observing the impact of pain on patient's lives, the responses of others to the patients communications of pain, and the extent to which patients participate in general daily activities.

4. Pain related Self-Statements Scale (PRSS ; Flor 1992) Assesses situation specific aspects of patients cognitive coping with pain. It consists of two sub-scales labelled catastrophizing and coping.

5. Pain related Control Scale (PRCS ; Flor 1992). This scale measures general attitudes towards pain. It consists of two sub-scales labelled helplessness and resourcefulness.

This was a single group repeated measurement study. Measurements were taken before the programme, immediately post programme and have still to be taken 3 months post programme.

Procedure

The multidisciplinary team consisted of a clinical psychology trainee, a physiotherapist and a pain consultant. The group met once a week for two hours and the programme ran for nine weeks. The aims of the group were to:

- Improve understanding of chronic pain
- Improve management of pain
- Help patients function as best they can despite the persisting pain
- Increase confidence in ability to function and cope
- Learn and practice relaxation therapy
- Identify unhelpful thoughts concerning pain. Challenge these thoughts with a more realistic thinking style
- Increase physical fitness in terms of power, confidence and flexibility
- Increase the range and level of daily activities
- Reduce the level of pain behaviours
- Improve sleep

Each session was highly structured with input from the anaesthetist on the first and last session. Psychology input ran over seven weeks for one hour followed by physiotherapy for 45 minutes. Topics covered by psychology included cognitive therapy, goal setting, reducing pain behaviours, recognising psychological and physical tension, increasing activity levels, introduction of various coping techniques and finally reviewing progress

and reinforcing achievements. Physiotherapy input included relaxation therapy and the introduction of a physical exercise programme. An explanation of chronic pain and related medical aspects were provided by the anaesthetist.

The hourly sessions followed a set agenda. The first 10 minutes were spent reviewing homework from the previous session and giving feedback. This was followed by introducing the general topic for discussion. A problem solving style was adopted throughout with the expectation of active participation from the group. Techniques introduced during the session were viewed as skills to be acquired by practice in their home environment through homework assignments. Subjects were not seen individually and discussing personal problems not relevant to the group was discouraged. A handout with relevant summary of the covered topic was given at the end of the session in order to enhance retention of information (Ley, 1977).

Results

Significant improvements between preprogramme and postprogramme measurements were made on five measures (**Table 2**)

Situation specific self-statements which promote active coping increased significantly as did convictions of resourcefulness. Given that solicitous responses have decreased significantly (**Figure 1**) may suggest that the subjects were actively doing more for themselves post programme and not dependent on reassurance from others to the same degree. Patients who believe solicitous responses from others are appropriate demonstrate lower levels of well-being than those who do not express such an attitude (Jensen & Karoly, 1992).

Table 2 Outcome measures

		t	Raw Scores	
			Before	After
HAD	- anxiety	1.32	65	46
	- depression	1.46	75	51
GHQ		4.92*	51	6
MPI	- interference	1.83	332	272
	- support	2.10	116	102
	- pain intensity	1.52	123	105
	- self control	2.17	57	42
	- negative mood	2.40*	109	82
	- punishing responses	1.88	68	55
	- solicitous responses	3.58*	118	91
	- distancing responses	0.00	75	75
	- household chores	0.48	120	129
	- outdoor work	1.08	48	52
	- activities away from home	1.73	59	66
	- social activities	1.16	77	83
	prss	- catastrophising	1.22	129
- coping		3.77*	121	189
prcs-	- helplessness	0.48	88	73
	- resourcefulness	3.24*	182	217

* - significant at $p < .05$.

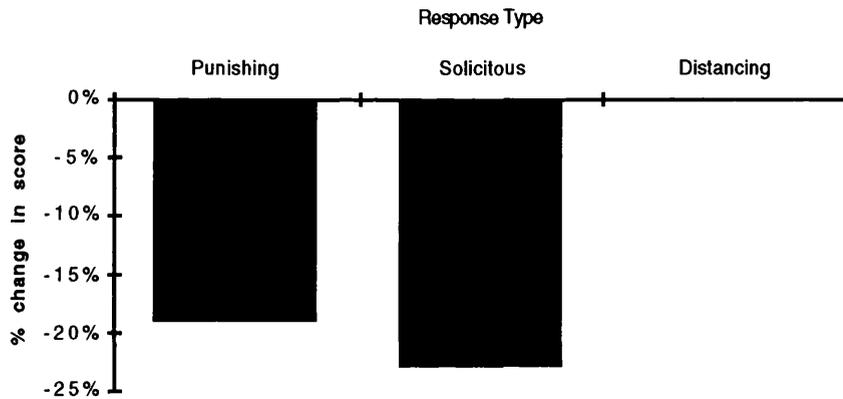


Figure 1 Partner's responses

Negative mood as measured by the GHQ and the MPI decreased significantly and this trend was also noticeable on the HAD (Figures 2(a), (b)).

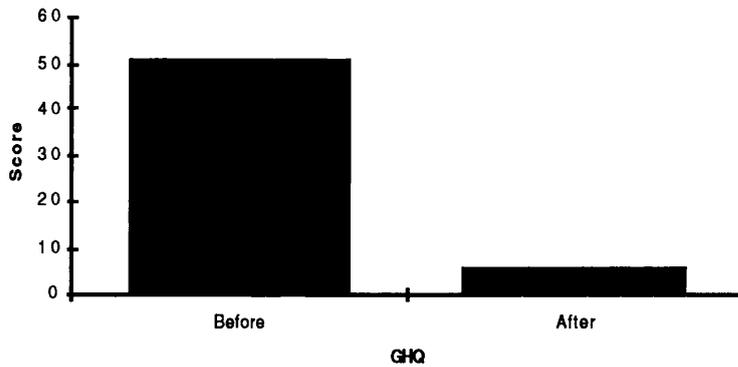


Figure 2(a) General Health questionnaire

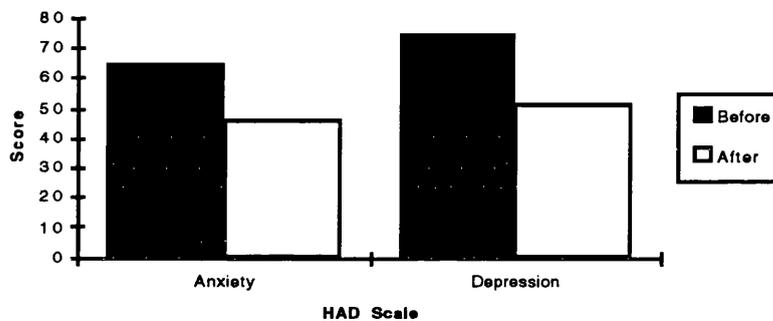


Figure 2(b) Hospital Anxiety & Depression scale

The course evaluation questionnaire reviewed each of the aims of the programme (**Appendix 4.2**). Everyone found the programme helpful on most areas with improvements in sleep rated by fewer subjects. Improving understanding of chronic pain was rated as the most helpful aspect of the programme with less satisfaction over mastery of relaxation.

Discussion

The significant decrease in catastrophizing in this group may be linked to the significant improvement in mood and reduced feelings of helplessness. Catastrophizing is a cognitive process characterised by a lack of confidence and control and the expectation of negative

outcomes (Chaves & Brown, 1987). It has been used to explain the variation in pain and depression in chronic pain and is considered to be a maladaptive coping strategy which intensifies the experience of pain and depression (Keefe et al., 1989). The significant decrease in solicitous responses is also encouraging. In a study by Flor et al., (1992) the degree to which spouses engaged in solicitous, distracting or punishing responses was assessed. Patients whose spouse ignored or responded negatively to their pain behaviour had higher activity levels. In addition, those whose spouses were overly solicitous had higher levels of pain and lower levels of activity.

This pain management group was run as a small pilot to assess one alternative to current clinical practice. It differed in two following ways. Firstly, it used group rather than individual format. Secondly, a multidisciplinary approach was adopted i.e. three disciplines worked simultaneously rather than following a single mode of treatment and only referring on once other approaches have been ruled out. Other than the obvious advantages in terms of time and more effective use of available resources there were other important implications for service.

The underlying principle of multidisciplinary pain management programmes, whether run in group or individual format, is that psychological factors contribute to the onset and maintenance of chronic pain. It follows that appropriate treatment will include psychological components. At present all patients referred to this Out-patient Pain Clinic are seen initially by a medical Doctor, who then decides what patients are to be referred to psychology. This current practice would seem to be flawed in three ways :

1. As psychological variables appear to be contributory to chronic pain in all instances it follows that psychological intervention would be beneficial in all situations.

2. Referral to a pain consultant from other medical specialists e.g. GPs or Orthopaedic Surgeons may reinforce the medical model of illness in both the patient and the professional. Further referral to a psychologist may then be viewed as a last resort to helping the individual by the consultant and as a rejection by the patient. The efficacy of psychological treatment may then be undermined as the patient may have difficulty reconceptualizing their problem in psychological terms and continue to seek medical cure. Multidisciplinary pain management programmes ensure that all patients receive appropriate psychological intervention and that the contribution of psychological variables to chronic pain is viewed in conjunction with physiological variables.

3. Many patients referred to outpatient pain clinics with chronic benign pain will not respond to medical intervention. Continuing contact with the medical profession including initial assessment at a pain relief clinic perpetuates the myth of a cure for chronic pain.

In addition to addressing shortcomings in present practice and introducing a mode of treatment which is based on recent theory and research findings, this initial small group programme helped to highlight practical and methodological shortcomings in establishing such a group. From a practical viewpoint the need for close cooperation between all disciplines involved is essential, with preferred regular meetings to review progress. All members of the team should be aware of the psychological principles of the approach being used and familiar with the nature of input from the other disciplines. Some methodological shortcomings were also evident. The sample size was small and there was no control group (other than the preprogramme condition). All data were subjective and the use of an independent assessor was not possible. These subjects could be seen to represent a motivated subgroup as they completed the programme and no follow up data was available from the person who dropped out. Follow up assessment has not yet been

undertaken so it is not possible to look at follow up data. Partners and family are usually included in pain management programmes because of the social context in which pain occurs. However, they were not actively involved in this study and there is only subjective data on responses from family. It is evident that the ability to generalise from these findings is restricted but there is room for optimism as patients had improved in all measures of outcome and they were better able to manage their pain at the end of the programme.

References

Bandura, A.(1977). Self- efficacy: towards a unifying theory of behavioural change, *psychol. Rev.*, 84 191-215.

Chaves, J.F., & Brown, J.M., (1987) Spontaneous cognitive strategies for the control of clinical pain and stress. *Journal of Behavioural Medicine*, 10, 263-276.

Department of Health, OPCS. (1994). *The Prevalence of Back Pain in Great Britain*. An omnibus data survey produced on behalf of the Department of Health.

Flor, H., Behle, D.J., Birbaumer, N. (1992). Assessment of Pain -Related Cognitions in chronic Pain Patients. *Behav. Res. Therapy* 31: 63-73.

Flor, H., Fydrick, T., & Turk, D.C.(1992). Efficacy of multidisciplinary pain treatment centers: a meta-analytic review. *Pain*. 49 : 221-230.

Fordyce, W.E.,Fowler, R.S.,Lehmann, J.F.,& De Lateur, B.J. (1968) Some implications of learning in problems of chronic pain, *Journal Chronic Disability.*, 21 179-190.

Frank, A. (1993). Low Back Pain. *British Medical Journal* 306: 901-909.

Goldberg, D. (1978). *The General Health Questionnaire*. NFER-NELSON Publishing Company Ltd., Darville House, 2 Oxford Road House East, Windsor, Berks. SL4 1DF.

Jensen, M.P., & Karoly, P. (1992). Pain -Specific Beliefs, Perceived Symptom Severity, and Adjustment to Chronic Pain *The Clinical Journal of Pain* 8: 123-130.

Keefe, F.J., Dunsmore, J., Burnett, R. (1992). Behavioural and Cognitive-Behavioural Approaches to Chronic Pain: Recent Advances and Future Directions *Journal of Consulting and Clinical Psychology*. Vol 60, No. 4, 528-536.

Kerns, R., Turk, D.C., Rudy, T.F. (1985). The West Haven-Yale Multidimensional Pain Inventory. *Pain*. 23: 345-356.

Ley, P. (1977). Psychological Research on Doctor-Patient Communication in S.J. Rachman (Ed) *Advances in Medical Psychology*. Pergamon

Melzack, R., Wall, P.D. (1965). Pain Mechanisms: a new theory, *Science*, 150, 971-979.

Nicholas, M.K., Wilson, P.H., & Goyen, J. (1991). Operant - behavioural and cognitive- behavioural treatment for chronic low back pain, *Behav. Res. Ther.*, 29 225-238.

Pither, C.E.,& Nicholas, M.K.(1991). Psychological approaches in chronic pain management, *British Medical Bulletin*. Vol47, No 3, pp 743 - 761.

Rigge, M. (1990) WHICH ?. *Way to Health*

Skinner, J.B., Erskine, A., Pearce, S. (1990). The Evaluation of a cognitive behavioural treatment programme in outpatients with chronic pain. *J. Psychosom. Res.* 34: 13-19.

Vasudevan, S.V., Lynch, T., Grunert, B.K. (1985). Outpatient management of chronic Pain: Long term results. Wisconsin Medical Journal. 84 : 7-9.

Wells, J.C.D., & Miles, J.B. (1991). Pain clinics and pain clinic treatments. British Med. Bull. 47 : 762-785

Zigmond, A.S., & Snaith, R.P., The Hospital Anxiety and Depression Scale. Acta Psychiatr. Scand. (1983) ; 67: 361-370.

Specific Language Impairment

A Single Case Report

Therese Carney

Department of Psychological Medicine

Glasgow University

Running Head: Specific Language Impairment

Abstract

A diagnosis of Specific Language Impairment (SLI) may not always be beneficial, as it is based on exclusionary criteria and theoretical models. This single case study documents the assessment and subsequent diagnosis of a three year old male with this condition. The relevance of making this diagnosis here is addressed and in conclusion it is surmised that there are advantages for future management.

Introduction

Making a diagnosis of specific language impairment (SLI) raises a number of issues. Terminology involving language and speech disorders presenting in childhood has evolved in the past few years leading to some confusion. Diagnostic and Statistical Manual of Mental Disorders (Third Edition - Revised; American Psychiatric Association, 1987) lists under its language and speech disorders 'Developmental Expressive language Disorder', 'Developmental Receptive language disorder' and 'Developmental Articulation Disorder'. These terms however, are not often referred to in the literature and would seem to fall under the more general terms 'specific developmental language disorder' (Bishop, 1987) and more recently 'specific language impairment' (Bishop, 1991). Other terms which are still in use include 'developmental dysphasia' and 'developmental aphasia' (Rapin, 1987) but they may be misleading and imply a condition with a known neurological basis. A diagnosis of SLI includes the child who understands very little spoken language and who produces only short and unintelligible utterances, the child who talks easily but seldom responds appropriately to adult attempts to initiate conversation, the child who seems to understand most of what is said but is limited in his ability to form any but the simplest utterances and finally the child who can talk clearly in single words but has problems as the length of the utterances increase.

At present, diagnosis of SLI is based on exclusion criteria. A neurological basis for this condition has not been identified and there is seldom any evidence on history of acquired brain damage. Those children with a history of acquired brain damage usually present with global delay affecting motor, social and non-verbal skills alongside language development (Stewart, 1984). Developmental delay in other areas prevents a diagnosis of SLI being made. Studies using investigative techniques are at best contradictory. Harcherik et al. (1985) used blind assessment of CT scans and quantitative analysis to compare children with developmental language disorder to a control group and found no difference. They concluded that

developmental language disorder is seldom associated with CT abnormalities unless the child has additional neurological problems. Lou, Henriksen, and Bruhn (1984) measured cerebral blood flow in eight language disordered children and reported hypoperfusion of certain brain areas. All but one of the children however, had additional problems which may have accounted for the blood flow abnormality. In addition to the above exclusion criteria, hearing impairment, emotional disorder or environmental deprivation must be ruled out before a diagnosis of SLI may be made.

The cause of SLI is unknown, and the failure of normal language development is explained by various psychological models in terms of impairment in a particular aspect of cognitive functioning. Eisenson (1972) viewed SLI as an impairment of auditory perception whereas Cromer (1978) argued that some children may have a deficiency in the specialized linguistic mechanisms that have evolved to handle language processing. Other models emphasise impairments in conceptual development (Johnston, Ellis and Weismer, 1983) or learning strategies (Kamhi, Catts, Koeing and Lewis, 1984). Proponents of each model advance evidence to support their viewpoints but the different explanations can add to the confusion.

What are the implications of making this diagnosis given that there is no known cause other than theoretical models, that explanatory conditions are excluded, and the range of linguistic manifestations subsumed under this diagnostic label is extensive. The present case study looks at this issue. The child in question was referred by the Area Clinical Medical Officer following a 39 month medical. The referral letter stated that his general development, and in particular his language skills, were delayed and that he had a tendency to do “his own thing” (sic). There were no obvious medical problems.

Clinical Presentation

This three-and-a-half year old boy lived with his parents and sister (7 years). His family moved from England six months before assessment due to his father's job being reallocated. Previously there had been no house moves in his lifetime. His mother was the main caregiver and looked after him full-time, except when he attended playgroup two mornings per week. She had made the choice of giving up work as a clerk when the first child was born. From reports and observation, this appeared to be a stable interactive family, spending quality time together whenever possible. He was born by Caesarian Section for transverse lie with no complications. His Apgar scores were good and there were no reported medical problems. He walked at just over one year and was fully toilet trained when seen.

This child had a history of delayed language development. His mother reported that he was 11 months old when he made his first attempt to speak and had made very little progress at 18 months. After his move to Scotland, he was referred to a Speech Therapist by his GP, but no action was taken following the initial interview. At his 39 months medical it was felt that there was a general developmental delay, particularly with language. He subsequently had a hearing test which proved negative and there had been no history of otitis media. A family history revealed that this child's maternal uncle had speech therapy as a child for difficulty pronouncing words and that his mother had received help with reading at age 6-7 years. His sister had no known problems. He was becoming increasingly difficult to manage at home due to frequent tantrums and oppositional behaviour.

Procedure

The principal aim of assessment was to determine if there was a developmental delay and if so, the nature of this impairment. This assessment included gaining information from his family,

the staff at his playgroup, the speech therapist involved initially and the CMO, all with his parent's consent. Formal psychometric assessment of his developmental status was included to get an objective picture of his current level of functioning. Finally observation of the child with his family and throughout the assessment gave valuable information.

He was seen on five occasions in all, the first being conducted at home together with his family, and further assessments taking place in the department. Physically he was small and slight for his age. He interacted well with his family and myself on the whole, but got frustrated easily. Most contact was initiated by his parents and they responded to his non-verbal communication. He played well with his sister on the first visit and was keen to share his toys. During the formal psychometric assessment, his behaviour varied from being very co-operative to being non-compliant and disruptive. Throughout, he had very definite ideas of preference and concentrated well when given a task he enjoyed. During this time he responded well to questions and there was evidence of precision, structure and creativity in his approach. In the main, his approach to assessment tasks was solitary but on occasion, invited participation from his mother and myself. When requested to participate in a task which was unappealing to him (perhaps due to lack of understanding), he was immediately non-compliant and reacted strongly sometimes by throwing items of the assessment across the room. At times, assessment had to be terminated due to this lack of co-operation. His actions throughout were accompanied by appropriate words or sounds.

Measures

As the assessment by the CMO for general developmental status relied on verbal instruction, understanding and expression a true picture of the child's abilities may not have been gained. For this reason, formal assessment employed non-verbal assessment wherever possible e.g. the Symbolic Play Test (Lowe and Costello, 1975), and subscales of the Griffiths Test

(Griffiths, 1984) which did not rely on expressive speech. The Symbolic Play Test is designed to assess the language potential of children who fail to develop receptive or expressive speech. It does so by evaluating concept formation and symbolization which develop alongside verbal language. It is used in conjunction with language tests e.g. The Reynell Development Language Scale (Reynell, 1977) and allows comparison of functioning in different areas. The Griffiths Mental Development Scale assesses the current developmental functioning of infants. It looks at motor development, personal-social skills, and cognition, and can diagnose developmental delay. The formal assessments employed were the following:

1. Reynell Developmental Language Scales
2. The Symbolic Play Test
3. Griffiths Mental Development Scales (non verbal sections)

Results

The Reynell Scale was used to get an objective and reliable estimate of language skills. The results of this test placed him at an equivalent age of 2 years 7 months for verbal comprehension and 1 year 11 months for expressive language.

In addition to having a picture of actual language status it was also thought necessary to get a profile of his language potential. On the Symbolic Play Test, his equivalent age was 36+ months.

To reduce bias and get a true picture of developmental status, the non-verbal subtests of the Griffiths Mental Development Scales were completed. The results of this assessment were as follows:

Performance Skills - Year III - 6 out of 6 tasks successfully completed
Year IV - 6 out of 6 tasks successfully completed

Eye and Hand - Year III - 6 out of 6 tasks successfully completed
Co-ordination Year IV - 2 out of 6 tasks successfully completed

In addition to formal assessment, family history taking and observation, further information was gained from staff at his playgroup. They reported that he was a self-contained boy who joined in group activities on occasion, but in the main played singularly. He joined in with other children's games and was open to letting them join with him but rarely invited companionship in play which he had initiated. He spoke little and was successful at getting his message across without verbalizing. He was reported to get frustrated very easily and could be oppositional and destructive when 'the mood takes him' [sic]. On closer questioning this would appear to mean when he was tired, when asked to do something, or when he was given something to play with which no longer held his attention.

The speech therapist whom he had seen on one occasion was of the impression that his speech was slow but that this was normal for children coming second in the family, and he would catch up at his own pace without intervention. Formal assessment was not completed.

Discussion

From the results it would appear that there was a definite language delay. This was more in evidence as an expressive problem, but his verbal comprehension was also below average. However, his language potential, as assessed by his use of concepts and symbols, was normal for his chronological age. This points to a gap between his actual and potential language development. Non-verbal performance ability is also normal for his age which again points to

a discrepancy between his verbal and his performance skills at this stage. His eye and hand coordination, although less well developed, were not outside the normal range for his age. There was no direct evidence of a general developmental delay (other than his small physical frame).

This boy was part of a close-knit family. He appeared securely attached to both parents and had ample play and interactive opportunities. Exposure to language of sufficient quality and quantity was the norm, and his sister sharing the same environment had no such difficulties. Therefore, environmental deprivation did not appear to be a factor. Hearing impairment had been ruled out and there was no history of otitis media which can sometimes be linked with verbal deficiencies (Paradise, 1981). This excluded deficient auditory stimulation as a potential cause. As the referral letter disclosed, he did do 'his own thing' but there was no evidence of autism. He played on his own, but was not abnormally self-contained and enjoyed contact. His ability to use symbols and form concepts was normal for his chronological age and there was no evidence of repetitive play or resistance to change. He had good non-verbal ability and there was no indication of any emotional or medical disorder. Acquired brain damage was also excluded, with an unremarkable prenatal or perinatal picture and birth being less traumatic than the normal vertex delivery for the child.

Having excluded the former possible reasons for language delay, a specific language impairment could now be diagnosed. As is usual for this disorder, no obvious cause for this condition was identified (Bishop, 1987). Genetic influences have sometimes been cited as a potential cause. Investigations of language-disordered children have found an increased incidence of chromosomal disorders, but these only account for a small proportion of cases (Friedrich, Dalby, Staehelin-Jensen and Bruun-Petersen, 1982). However, specific language disorder tend to run in families and this case was no exception. The theory that children with specific language delay follow normal developmental progression, but at a slower rate, may be

appropriate here. His vocabulary was limited for his stage of development and his sentence structure telegraphic. He was slow at every stage of language development to this point. Leonard (1982) found that there were many similarities between the language skills of language-disturbed children and those of younger normally developing children. If this boy was to follow this course, the prognosis should be optimistic. Four year olds presenting with specific language delay were found to be indistinguishable from normal children on language tests by the age of 5 yrs 6 months (Bishop & Edmundson, 1988).

Despite problems inherent in making a diagnosis of SLI it is a useful classification here because of its implications. This child's 'bad' behaviour was observed mainly during verbal interactive scenarios. Research has shown that children with low language structure scores at 3 years (social class and sex controlled for), had significantly higher rates of neurotic deviance than children with high structure scores (Stevenson, Richman and Graham, 1985). The rate and amount of interaction initiated by a pre-school child is correlated to the rate and amount of interaction directed towards him by other children (Leiter, 1977). Also, linguistic and social interactions have been found to be influenced by the amount and complexity of the speech of the partner (Masur, 1978). Having a specific language disorder placed him at a potential disadvantage at nursery and on other occasions when he mixed with his peers. Therefore, making a diagnosis of SLI should guide management in helping with language development, in understanding potential influence on behaviour and in ruling out other causes of language delay.

References

American Psychiatric Association, (1987). *Diagnostic and Statistical Manual of Mental Disorders (Third Edition - Revised)*. Washington, DC.

Bishop, D., (1987) The causes of specific development language disorder (“Developmental Dysphasia”), *J. Clinical Psychol. Psychiat.*, 28, 1, 1-8

Bishop, D. & Edmundson, A, (1988) Specific language impairment as a maturational lag : evidence from longitudinal data on language and motor development, *Developmental Medicine & Child Neurology* ,29, 251-255.

Bishop, D. (1991) The Biological basis of specific language impairment. In P. Fletcher (ed), *Specific speech and language disorders in children*. London: Whurr.

Cromer R.F. (1978). The basis of childhood dysphasia: a linguistic approach. In M. Wyke (Ed.), *Developmental dysphasia* (pp.85-134). London: Academic Press.

Eisenson, J. (1972). *Aphasia in children*. New York : Harper & Rowe.

Friedrich, U, Dalby, M , Staehelin-Jensen, T & Bruun-Petersen, G (1982) Chromosomal studies of children with developmental language retardation, *Developmental Medicine & Child Neurology*, 24, 645-652

Griffiths, S (1955) Griffiths Mental Development scales (v1984), *Test Agency*

Harcherik,D.F., Cohen, D.J., Ort, S., Paul, R., Shaywitz, B.A. & Lechman, J.F. (1985). Computed Tomographic brain Scanning in four neuropsychiatric disorders of childhood. *American Journal of Psychiatry* 142, 731 - 734.

Johnston,J.R. & Ellis Weismer, S. (1983). Mental rotationabilities in language-disordered children. *Journal of Speech and Hearing Research*, 26, 397-403.

Kamhi, A.G., Catts,H.W.,Koeing, L.A., Lewis,B.A. (1984). Hypothesis-testing and non-linguistic symbolic abilities in language-impaired children. *Journal of Speech and Hearing Disorders*, 49, 169-176.

Leiter, M (1977) A study of reciprocity in pre-school school groups, *J. Child Development*, 48, 1353-1361

Leonard, L (1982) Phonological deficits in children with developmental language impairment, *J. Brain & Language* 16, 73-86

Lou, H.C., Henriksen, L. & Bruhn, P. (1984) Focal cerebral hypofusion in children with dysphasia and /or attention deficit disorder. *Archives of Neurology* 41, 825-829.

Lowe, M, & Costello, A, J , (1975) The symbolic play test , *NFER-Nelson*

Masur, E (1978) Pre-school boys' speech modifications : the effect of listeners' linguistic levels and conversational responsiveness, *J. Child Development*, 49, 924-927

Rapin, I. (1987). Developmental dysphasia and autism in pre-school children: characteristics and subtypes. In *Proceedings of the first international symposium on specific speech and language disorders in children* (pp.20-35). London: AFASIC.

Paradise, J (1981) Otitis media during early life : how hazardous to development ? A critical review of the evidence, *J Paediatrics* 68, 869-873

Reynell, J (1977) Reynell developmental language scales, *NFER_Nelson*.

Stevenson, J , Richman, N & Graham, P (1985) Behavioural problems and language abilities at three years and behavioural deviance at eight years, *J. Clinical Psychol. Psychiat.*, 26, 2, 215-230

Stewart, A., (1984) Severe perinatal hazards. In M.Rutter (Ed). *Developmental Neuropsychiatry* (pp. 15-31) Edinburgh : Churchill Livingstone.

Adrenoleukodystrophy - A Psychological Profile

Case study of a thirteen year old Male

Therese Carney

Department of Psychological Medicine

Glasgow University

Running Head: Adrenoleukodystrophy - A Psychological Profile

Abstract

This case study describes the psychological profile of a thirteen year old male presenting with Adrenoleukodystrophy and Controlled Epilepsy. An MRI scan demonstrated extensive posterior demyelination and CT scan showed some suggestion of possible abnormality in the (L) hippocampal area, with general atrophy involving the cerebrum and cerebellum. Assessment and history reveal dementia and learning difficulties. Cognitive, affective and social difficulties are described.

Introduction

Adrenoleukodystrophy (ADL) is a rare endocrinal disease of childhood which causes neurological and neuropsychological deficits. The latter have rarely been described in the literature. It is a progressive hereditary disorder which affects the adrenal gland and the white matter of the nervous system. It was first described in 1923, and in 1976 it was first recognised that ADL was one of the lipid storage diseases (Mosser, Douar, Sarde, Moser & Poustka, 1993). This means that there is progressive abnormal accumulation of certain fats, particularly in the brain white matter and in the adrenal gland, with resulting central nervous system demyelination. ADL is one of approximately 80 sex-linked disorders which affect males only and is transmitted by a female carrier (Close, Sinnott & Nolen, 1993). Boys with ADL usually develop normally until they are 4-10 years of age. Then they may experience behaviour and cognitive changes such as loss of memory, intellectual or emotional instability and varying degrees of difficulty with vision, hearing, walking and motor-function (Harris, 1985). The course of the disease is variable, and may demonstrate rapid or gradual decline with plateaus and periodic minimal or mild restitution of function over many months. The patient usually dies within one to ten years after diagnosis. ADL which begins in infancy ordinarily comes to medical attention because of developmental delay. When it starts later the patient usually presents with changes in behaviour at home and at school. For example, one case study of ADL (Shiga, Saito, Mochizuki, Chida & Tsuburaya, 1992) presented initially by school refusal and difficulty adapting to new school mates. Adrenomyeloneuropathy (AMN) is a milder form of the disease, with onset at 15 - 30 years of age and a more progressive course. Prevalence rates for ADL range from 1 in 20,000 to 1 in 50,000 male births (Mosser et al. 1993; Sereni, Paturneau & Aubourg, 1993).

Despite the wealth of literature on ADL in terms of neurology and endocrinology, for example, Sargent, Coupland & Wilson (1994) very little has been documented on the cognitive, social

and psychological deterioration which accompanies neurological change. Several interesting single case studies, for example, Zammarchi, Donati, Tucci & Fonda (1994) cite detailed biochemistry and radiography findings but only allude to the behavioural expression of the underlying pathology. This case study is therefore presented in order to describe the psychological status of one thirteen year old boy with recently diagnosed ADL.

Clinical Presentation

At the age of 4 years this boy had a right sided focal seizure and was admitted to hospital. The feeling at that time was that it might have been caused by a mild viral encephalitis from which he recovered completely. He was symptom free until the age of eleven, when he had a complex partial seizure in November 1992. At that time his parents separated and there were reports that his mood and behaviour changed and he became moody and withdrawn. Teachers at his school had noted a lack of concentration. In August 1993 he had another seizure and was referred to Neurology where he was diagnosed as having Epilepsy and was commenced on Carbamazepine and subsequently on Phenytoin.

In March 1994 he took an overdose of Phenytoin and was admitted to hospital for gastric levage and observation. It was reported that his sister had dared him to take the tablets during a quarrel and he had followed it through. He said that he had felt left out at school and was feeling down about it. He also spoke about hating his father, and of his mother saying he was just like him, his father having taken overdoses in the past. In hospital during this admission his behaviour was thought to be depressive and he was reviewed by a psychiatrist who felt that 'some elements of depression were present'. An EEG was carried out which was thought to be abnormal indicating the possibility of left temporal pathology. A CT scan followed which showed no definite lesion. He was discharged home and followed up on one occasion by the department of child and family psychiatry.

Since that period there have been ongoing concerns regarding his behaviour and his performance at school. In particular he had been noted to have difficulties understanding his reading, and with constructional work at school. There were concerns regarding his speech which seemed slightly dysarthric and he seemed to have a degree of receptive dysphasia. He was therefore referred to the Neurology team in July 1994 for neurological examination, at which time he was first seen by psychology. He reported that he had been having problems at school. He described his difficulty as 'being in a muddle sometimes'. He stated that he found it difficult to concentrate and lost track of conversation. He had then completed one year at secondary school. Reports from his primary school state that he was of average ability but did not apply himself. A CT scan was carried out which showed some suggestion of possible abnormality in the left hippocampal area and some general atrophy involving the cerebrum and the cerebellum. An MRI scan which followed found extensive posterior demyelination with an appearance typical of that found in ADL. Despite the obvious difficulties found on formal psychological assessment his mother did not want the school informed or Educational Psychology involved. He was followed up by the Neurology team, and in May 1995 he was seen for reassessment. A repeat MRI scan revealed no change in the demyelination process and following extensive endrocrinal analysis ADL had been diagnosed

Reports from his school confirm that they had become increasingly worried about his difficulties. They reported that his motor skills had deteriorated. In practical subjects all expressed concern that his movements were clumsy and he has to be monitored closely at all times. His handwriting had deteriorated, with difficulty writing along a line, forming his words properly or drawing a line with a ruler. In art classes, brushes had been damaged with his handling and at times his speech was indistinct and slurred. He was reported to have difficulty taking in instructions and following them without continued support. His work rate was slow and he was reported to be apart from the class. They described a tendency to drift into a dream like state and a difficulty organising himself - remembering to bring kit, equipment

or books. In casual conversation, they reported, it was difficult to establish eye contact or sustain a conversation. Often what he said did not connect with what he previously said. This was affecting his relationship with his peers as he seemed unable to relate to others, either one to one or in a group.

His mother reported that at home he had word finding difficulty and confused names of known people. He also got characters on television programmes mixed up and forgot verbal messages and phone calls. He had begun to repeat information to the extent that his mother got annoyed. She also noted that he had changed from being a popular sociable boy to having no close friends. He withdrew from contact with his peer group and spent most of his time at home with his family outside school. His mother was anxious as he was being bullied at school and had been caught shoplifting which was something she reported he would never have done in the past.

When seen in July 1994, he was relaxed and communicative. He spoke easily and with sensitivity about his family and his relationship with peers. His relationship with his father appeared ambivalent, on the one hand saying he hated him and on the other obviously upset that he had left to start a new life elsewhere. He had a slightly oppositional manner, sitting with his legs and arms crossed looking around the room and only maintaining eye contact fleetingly when he was talking. His affect was not depressed but he had easy access to emotionally laden material. His difficulty with some aspects of formal assessment was not easily gleaned from his overt behaviour. It was thought that this may have been due to a lack of awareness of problems or conversely his easy communication and relaxed manner may have distracted from or disguised his underlying difficulty. When seen for reassessment he covered for his difficulties by joking, changing the subject or confabulation. He was immature for his age based on his behaviour, and replies to questions which were sometimes not age appropriate.

Formal Assessment

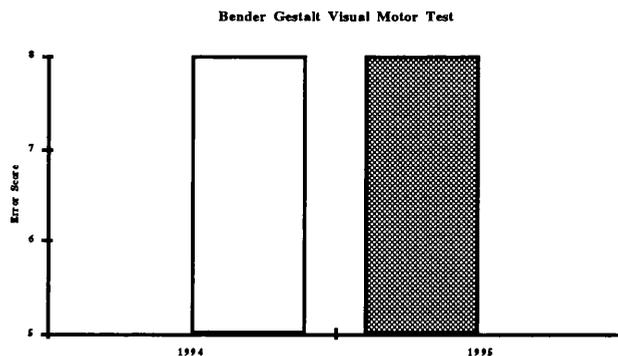
1. Wechsler Intelligence Scale for Children - Revised (Scottish) (WISC-RS)

This test was included to formally assess intellectual function.

	<u>Verbal Scaled Scores</u>		<u>Performance Scaled Scores</u>		
	1994	1995	1994	1995	
Information	7	6	Picture completion	4	4
Similarities	8	8	Picture Arrangement	8	6
Arithmetic	9	7	Block Design	4	3
Vocabulary	6	9	Object Assembly	4	3
Comprehension	6	5	Coding	3	3
Verbal IQ	84	82	Performance IQ	65	<65
Full Scale IQ	72	69			

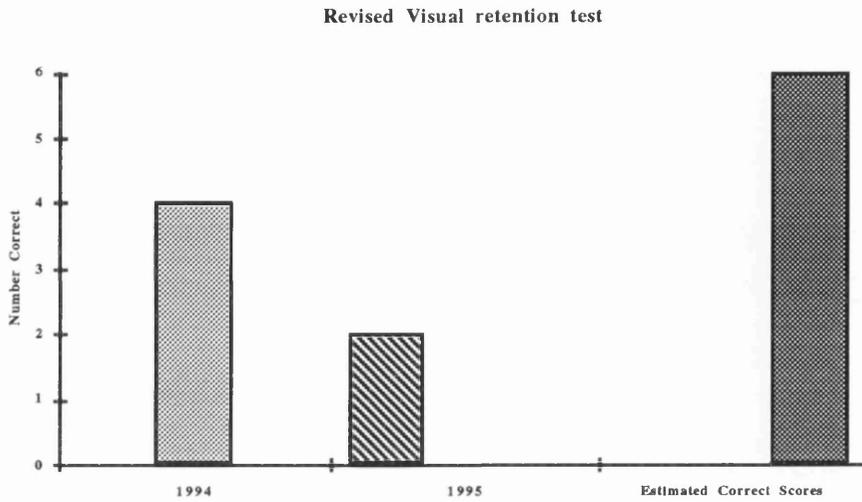
2. The Bender Visual Motor Gestalt Test

Included to assess visual perception and spatial awareness.



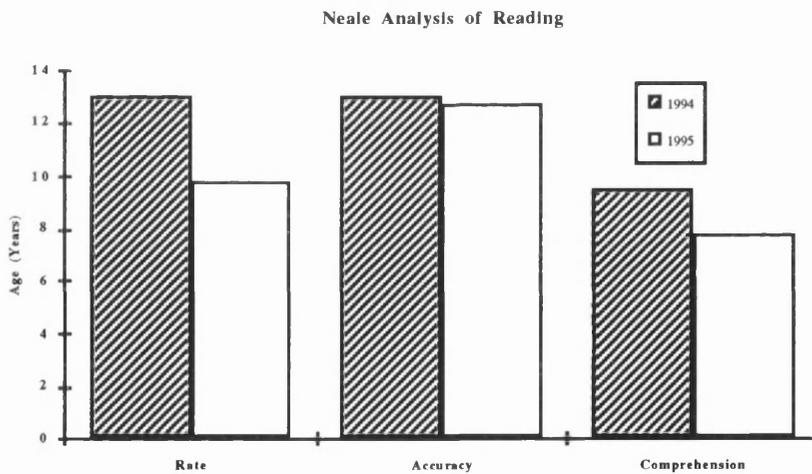
3. The Benton Visual Retention Test (BVRT)

This test was included to assess visual memory.



4. The Neale Analysis of Reading Ability - Revised British Edition

This test was included to assess reading skills and verbal memory.



Results

The results of the WISC-RS indicated that he had specific learning difficulties especially in performance related skills. His pre morbid history (conversing in sentences at age 2, riding a bike without stabilisers at age 3, reciting the alphabet forwards and backwards at age 3, and primary school reports - bright child) would not reflect this result. Although the reports are retrospective, it would appear that there has been a deterioration in functioning. The significant difference between his verbal and performance scores on both occasions may be a reflection of underlying psychopathology. His verbal skills fell within the low average range with little change on reassessment, and comprehension continued to be his weakest area of verbal functioning. However, he was found to be functioning within the disabled range for performance skills with a trend towards deterioration on reassessment.

The relatively poor comprehension of the passages on the Neale Analysis of Reading may be attributed partly to a poor verbal logical memory. There was evidence of confabulation from his replies, in that he responded to hints in the questions or gave logical but incorrect answers to compensate for his loss of memory for the passage. When a story was read to him, and asked to repeat it immediately afterwards his recall was patchy and sparse. On reassessment, his comprehension age had fallen by over one year. There was certainly evidence of a deficit in immediate visual memory on both occasions of assessment as seen by his performance on the BVRT. Performance declined with an increase in length or complexity of information. His ability to answer questions on mental arithmetic declined in relation to the length of the questions. In the Neale, his attention to reading the text interfered with his ability to remember the passage.

On both assessments there was evidence of visuoperceptual and visuoconstructional difficulties. His performance on the Block design, Object assembly and Coding subtests reflect

major difficulty in visuoconstructional functioning. This could be reflective of an additional underlying perceptual difficulty as qualitative analysis revealed. Three of the designs on the Bender Gestalt Test contained rotational errors on both occasions and on reassessment he attempted to assemble the car on the object assembly subtest upside down. Also on reassessment, there was evidence of complete reversals on the picture sequencing subtest on two occasions.

Discussion

For this boy, dementia and learning difficulties are the functional expression of the interference of pathology with normal development. Loss of intellectual function and poor memory for recent events were sufficiently severe to interfere with his social functioning. Abstract thought was also impaired with difficulty planning novel tasks and in defining concepts comparative to chronological age. There was also evidence of disturbance of higher cortical function. His speech was dysarthric and he had marked constructional difficulty. His personality had also changed from being an extraverted sociable boy with good social judgement to becoming withdrawn and inappropriate at times. The course of his dementia is dependent on underlying pathology. If his first presentation at age four with suspected viral encephalitis was indeed the initial onset of ADL (as has been noted in other case studies e.g. Zammarchi et al. (1994) then the course of the dementia until present has been slowly progressive.

A diagnosis of dementia could not have been made without considering the differential diagnosis of depression. This boy had attempted suicide in the past year and had some depressive symptomology when assessed by psychiatry. However, this diagnosis was ruled out for the following reasons. Although this boy took an overdose of tablets, and some depressive symptomology was present at that time, a major depressive episode was not diagnosed. Follow up consisted of one appointment only and his affect was judged to be

normal. This does not fit the clinical picture of childhood depression (American Psychiatric Association, 1987). Other reasons for the overdose also need to be considered. The act may have been reflective of impaired judgement or impulse control which are features of dementia. His history shows other examples of this e.g. shop lifting. When seen for assessment there was no evidence of depression. He denied feeling low or sad and appeared rather unconcerned. In addition, an underlying causative organic factor has been identified to support a diagnosis of dementia.

Although this boy was not depressed when seen for assessment his increased risk of psychiatric disorder was noted. The presence of brain dysfunction in childhood appears to be associated with a greater risk for the development of a psychiatric disorder (Brown, Chadwick, Shaffer, Rutter & Traub, 1981). Moreover, Rutter, Graham & Yule (1970) found that the rate of psychiatric disorder was over five times higher in their neuroepileptic group consisting of children ranging from 5 - 14 years of age. This risk was found to be greater for children with accompanying low IQ.

With epilepsy, cognitive and behavioural changes present as a result of seizure activity, psychological variables and medication. Complex partial seizures, which are present in this case, may be associated with personality attributes including deepened emotions, increased aggressiveness and alterations in social interactions (Bennett, 1987) some of which are evident here. As this boy has good seizure control, exhibits only one seizure type and has had the disorder for a relatively short period of time, the cognitive and behavioural changes associated with epilepsy are less likely to be present (Hermann & Whitman, 1986). However, his present drug Phenytoin, has been shown to have adverse effects on memory (Andrewes, Tomlinson, Elwes & Reynolds, 1984), psychomotor performance and problem solving (Thompson & Trimble, 1982).

Despite the inability to influence pathology here it is important to recognise that we can influence the ways in which this pathology can contribute nonspecifically to a lowered adaptive capacity and a greater likelihood of exposure to adverse experiences. Boll & Barth (1981) argued that brain dysfunction operates indirectly by creating the functional deficits that make successful adjustments more difficult for the child. Pathology here gives rise to dementia and learning difficulties which in turn render him more likely to encounter frustration and failure at home and at school. Subsequently he withdraws and becomes isolated. The complex interaction of ADL and epilepsy and the functional problems of learning difficulty and dementia all have an impact on motor, affective, cognitive and social development.

References

American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed. Rev), Washington DC.

Andrewes, P.J., Tomlinson. L., Elwes, R.D., & Reynolds, E.H. (1984). The influence of carbamazepine and phenytoin on memory and other aspects of cognitive function in new referrals with epilepsy. *Acta Neurologica Scandinivica*, Suppl. 99. 23-30.

Bennett, T.L. (1987). Neurological aspects of complex partial seizures : Diagnostic and treatment issues. *International Journal of Clinical Neuropsychology*, 9, 37-45.

Bender, L. (1938). A visual motor gestalt test and its clinical use. American Orthopsychiatric Association Research Monographs, No 3. (Children's Scoring Booklet, Largos,C.J. (1973) - Western Psychological Services.

Benton, A.L. (1974). Revised Visual Retention Test : San Antonio, TX : Psychological Corporation.

Boll, T., & Barth, J. (1981). Neuropsychology of brain damage in children. In S.B. Filskov & T.J. Boll (Eds.), *Handbook of clinical neuropsychology* (pp. 418-452). New York: Wiley.

Brown, G., Chadwick, O., Shaffer, D., Rutter, M., & Traub, M. (1981). A prospective study of children with head injuries. 111. Psychiatric sequela . *Psychological Medicine*, 11, 63-78.

Close, P.J., Sinnott, S.J., Nolen, K.T. (1993). Adrenoleukodystrophy. A case report

demonstrating unilateral abnormalities. *Pediatr - Radiol.* 23 (5): 400-1.

Harris, J.C. (1985). Living with Adrenoleukodystrophy. Paper presented at 'United Leukodystrophy Families Conference' Towson State University, Baltimore, Maryland.

Hermann, B.P., & Whitman, S. (1986). Psychopathology in epilepsy: A multi-etiological model. In S Whitman & B.P. Hermann (Eds), *Psychopathology in epilepsy: Social dimensions* (pp. 5-37). New York: Oxford University Press.

Moser, J., Douar, A.M., Sarde, C., Moser, L., Poustka, A. (1993). 'Putative X-linked ADL gene shares unexpected homology with ABC transporters'. *Nature* Feb 25, 361 (726-30).

Neale Analysis of Reading Ability - Revised British Edition. (1987). NSER Nelson.

Rutter, M., Graham, P., & Yule, W. (1970). *A neuropsychiatric study in childhood* (Clinics in Developmental Medicine Nos. 35-36). London: Spastics International Medical Publications. Heinemann Medical Books.

Sargent, J.R., Coupland, K., Wilson, R. (1994). Nervonic acid and demyelinating disease. *Med-Hypotheses.* Apr; 42(4) : 237-42.

Shiga, Y., Saito, L., Mochizuki, J., Chida, K., & Tsuburaya, K. (1992). A case of ADL having progressed from the frontal lobes. *Rinsho-Shinkeigaku* Jun; 32(6): 100-5.

Sereni, C., Paturneau, P.M., Aubourg, P. (1993). Adrenoleukodystrophy in France: an epidemiological study. *Neuroepidemiology*; 12(4) 229-33.

Thompson,P.J., & Trimble, M.R. (1982). Anticonvulsant drugs and cognitive functions. *Epilepsia*, 23, 531-544.

Wechsler Intelligence Scale for Children - Revised (Scottish). (1987). Psychological Corporation.

Zammarchi,E., Donati, M.A., Tucci,F., Fonda, C. (1994). Acute onset of X-linked adrenoleukodystrophy mimicking encephalitis. *Brain-Dev.* (1994). 16(3): 228-40.