

**AN INVESTIGATION OF THE RELATIONSHIP BETWEEN  
PERSONALITY-FAMILY FACTORS & RESPONSE TO TREATMENT  
IN YOUNG ADULT ANOREXIC AND BULIMIC PATIENTS**

**MOHAMMAD ALI BESHARAT DEHAQANI**

**Thesis Submitted to the University of London  
for the Degree of Doctor of Philosophy**

**1997**



ProQuest Number: 10018561

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 10018561

Published by ProQuest LLC(2016). 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

## **ABSTRACT**

This thesis reports on a study of family-, personality-, and treatment-related factors in eating disorders. The primary aim of the investigation is to examine the relationship between personality-family factors and eating disorders, and their prognostic value in relation to engagement-outcome in different types of psychological treatment.

A study was undertaken evaluating 78 consecutive outpatient referrals to the Maudsley Hospital Eating Disorder Clinic for eating disorders. The patients were all at or over the age of 18 years. An extensive evaluation for both pre- and post-treatment phases was made of the patients and their families, in which three types of outpatient psychological treatment (family therapy, individual focal psychoanalytic psychotherapy and cognitive analytic therapy) for young adults suffering from eating disorders were compared with a fourth control treatment, supportive therapy.

This thesis is divided into two parts. Forwarded by presenting the “introduction and outline of the thesis”, the first part comprises a review of the literature, which includes research on “expressed emotion”, “personality disorders”, “self-disclosure”, and “self- and other-blame”. The design and methodology of the study are presented in this part of the thesis, as well.

The second part presents the results and discussion of the study in five sections, family expressed emotion, personality disorders, patient’s response style to therapist and therapy, self- and other-blaming attitudes, and prognostic factors, respectively. First, it is shown that a critical attitude of the relatives is associated with specific interactional behaviours of the relatives, patients, and the

whole family. Therefore, a better understanding of both the nature of EE constructs and the family EE patterns can be gained when it is evaluated within the family relationships. Second, it is shown that both AN and BN patients who require outpatient treatment seem to equally display a personality pathology necessitating special attention in the treatment setting. Characterological differences between anorexic and bulimic patients are important aspects of the present investigation. Third, evidence is provided for reliability (and to a lesser degree for validity) of a new measure of assessing patient's response style to therapist and therapy, the Patient Response Style Scale (PRSS). Fourth, evidence is also provided for reliability (and also to a lesser degree for validity) of a new measure of assessing self- and other-blaming attitudes, the Self- and Other-Blame Scale (SOBS). A high level of feeling self-blame among eating disorder patients and their relatives is shown. The association of other-blaming attitudes with personality disorders and poor psychological well-being is confirmed. Fifth, It has become apparent that EE is sensitive to changes in the patient, and can predict the outcome of treatment. Personality disorders are found to be associated with poor response to treatment in eating disorders. The importance of prognostic values of the PRSS and SOBS variables are confirmed.

The clinical and theoretical implications of the study, as well as limitations of the study, are discussed in this part of the thesis. The thesis is closed with presenting the concluding statements drawn from the results of the study.



## **ACKNOWLEDGEMENTS**

I would like to express my sincere gratitude to the following persons:

Dr **IVAN EISLER**, my supervisor, for his consistent support, availability and constructive criticism and guidance throughout my Ph.D. project.

Dr. Christopher Dare for his encouragement, suggestions and comments on part of the thesis.

Elizabeth Dodge for rating treatment outcome.

Dr. Christine Vaughn for the Expressed Emotion (EE) training.

Professor Peter Tyrer for his comments on the Personality Assessment Schedule (PAS) and his group in St. Charles' Hospital for the PAS rating training.

Padmal de Silva for his advice on the Maudsley Obsessional-Compulsive Inventory (MOCI).

Professor Graham Dunn and Dr. Sophia Rabe-Sketh for their advice on the statistical analysis.

Eric Glover and Charles Sharp for their advice on matters computational.

Lastly my thanks to the many people who took part in this study and from whom I have learnt.

**TO MY FATHER, MOTHER, AND MY WIFE**

<b>CONTENT</b>	<b>Page</b>
<b>Title Page</b>	<b>1</b>
<b>ABSTRACT</b>	<b>2</b>
<b>Acknowledgements</b>	<b>4</b>
<b>Dedication</b>	<b>5</b>
<b>Contents</b>	<b>6</b>
<b>List of Tables</b>	<b>12</b>
<b>List of Figures</b>	<b>15</b>
<b>PART I</b>	<b>16</b>
<b>CHAPTER 1</b>	<b>16</b>
<b>INTRODUCTION AND OUTLINE OF THE THESIS</b>	<b>16</b>
<b>CHAPTER 2</b>	<b>20</b>
<b>A REVIEW OF THE LITERATURE</b>	<b>20</b>
<b>2.1. EATING DISORDERS</b>	<b>20</b>
<b>2.1.1. Anorexia Nervosa: historical development of the concept</b>	<b>20</b>
<b>2.1.2. Bulimia Nervosa: a new syndrome</b>	<b>22</b>
<b>2.1.3. Diagnostic Criteria and Classification of Eating Disorders</b>	<b>24</b>
<b>2.1.4. Aetiology</b>	<b>31</b>
<b>2.1.4.1. Psychodynamic and Developmental Models of Eating Disorders</b>	<b>31</b>
<b>2.1.4.2. Family Models of Eating Disorders</b>	<b>34</b>
<b>2.1.4.3. Family System Models of Eating disorders</b>	<b>35</b>
<b>2.1.4.4. Sociocultural Models of Eating disorders</b>	<b>39</b>
<b>2.2. EXPRESSED EMOTION</b>	<b>41</b>
<b>2.2.1. The Concept of Expressed Emotion</b>	<b>41</b>

2.2.2. Why Expressed Emotion Is Useful for this Study	41
2.2.3. Measures of Expressed Emotion	42
2.2.4. The Nature of Expressed Emotion	43
2.2.5. Family Expressed Emotion in Eating Disorder Patients	45
2.3. PERSONALITY DISORDERS	47
2.3.1. Personality Disorders in Eating Disorder Patients	49
2.4. PATIENTS' RESPONSE STYLE TO THERAPIST AND THERAPY	53
2.4.1. Self-Disclosure: conceptual background	54
2.4.2. Definition and Parameters of Self-Disclosure	56
2.4.3. Self-Disclosure and Personality	57
2.4.4. Self-Disclosure and outcome of therapy	59
2.4.5. Emotional Engagement	60
2.4.6. Response Style to Therapist and Therapy in Eating Disorder Patients	60
2.5. SELF- AND OTHER-BLAME	61
2.5.1. Characterological and Behavioural Self-Blame	62
2.5.2. Self- and Other-Blame in Eating Disorder Patients	64
CHAPTER 3	68
3.A. DESIGN AND METHODOLOGY OF THE STUDY	68
3.A.1. PURPOSE OF THE RESEARCH	68
3.A.2. PRESENTATION OF THE RESEARCH HYPOTHESES	69
3.A.3. METHOD	70
3.A.3.1. Sample	70
3.A.3.2. The Clinical Assessment Procedure	71
3.A.3.3. Instruments	75

<b>3.A.3.3.1. Observational Measures</b>	<b>75</b>
a) The Morgan-Russell Assessment Schedule	75
b) The Personality Assessment Schedule	76
c) The Standardised Clinical Family Interview	77
<b>3.A.3.3.2. Self Report measures</b>	<b>78</b>
a) The Eating Disorder Inventory	78
b) The Bulimic Inventory Test, Edinburgh	78
c) The Beck Depression Inventory	79
d) The Inventory of Interpersonal Problem	79
e) The Maudsley Obsessional- Compulsive Inventory	80
<b>3.A.3.4. Statistical Analysis</b>	<b>80</b>
<b>3.B. The Patient Response Style Scale (PRSS)</b>	<b>82</b>
<b>3.B.1. A Measure of Patient’s Response Style to Therapist and Therapy:</b>	
The Development of the Patient Response Style Scale (PRSS)	84
<b>3.B.2. The PRSS Scales</b>	<b>85</b>
<b>3.B.2.1. Self-Disclosure</b>	<b>85</b>
<b>3.B.2.1.1. Self-Disclosure Rating Criteria</b>	<b>85</b>
a) Simple Response	85
b) Elaboration	87
c) Self-Personal Information (Intimacy of Disclosure)	89
c.1) Family Personal Information	89
c.2.) Sexual Matters	91
d) Spontaneity	91
<b>3.B.2.2. Emotional Engagement</b>	<b>92</b>

<b>3.B.2.2.1. Emotional Engagement Rating Criteria</b>	<b>93</b>
<b>a) Positive Tone of Voice</b>	<b>93</b>
<b>b) Interest in the Therapist and Therapy</b>	<b>93</b>
<b>3.B.3. Allocation of Scores on the PRSS Scales</b>	<b>96</b>
<b>3.B.4. The PRSS Reliability</b>	<b>97</b>
<b>3.C. The Self- and Other-Blame Scale (SOBS)</b>	<b>99</b>
<b>3.C.1. A Measure of Self and Other-Blame: The Development of the Self- and Other-Blame Scale (SOBS)</b>	<b>100</b>
<b>3.C.2. The SOBS Rating Criteria</b>	<b>100</b>
<b>a) Causality</b>	<b>100</b>
<b>b) Responsibility</b>	<b>103</b>
<b>c) Negative Feeling</b>	<b>105</b>
<b>d) Negative Tone of Voice</b>	<b>106</b>
<b>3.C Allocation of Scores on the SOBS</b>	<b>107</b>
<b>3.C.4. The SOBS Reliability</b>	<b>108</b>
<b>PART II</b>	<b>110</b>
<b>CHAPTER 4</b>	<b>110</b>
<b>PRESENTATION OF THE RESULTS</b>	<b>110</b>
<b>4.A. EXPRESSED EMOTION</b>	<b>110</b>
<b>4.A.1. Clinical Features of the Diagnostic Groups</b>	<b>110</b>
<b>4.A.2. The Distribution of Family Expressed Emotion</b>	<b>112</b>
<b>4.A.3. The Course of Family Expressed Emotion</b>	<b>119</b>
<b>4.A.4. The Distribution of Treatment Outcome for AN and BN Patients</b>	<b>122</b>
<b>4.B. PERSONALITY DISORDERS</b>	<b>125</b>

<b>4.B.1. The Distribution of the PAS Personality Disorder Diagnoses</b>	<b>125</b>
<b>4.B.2. Clinical Features of the Patients and Family Factors Associated with Personality Disorders</b>	<b>129</b>
<b>4.C. PATIENT’S RESPONSE STYLE TO THERAPIST AND THERAPY</b>	<b>130</b>
<b>4.C.1. The Distribution of the PRSS</b>	<b>130</b>
<b>4.C.2. Clinical Features of the Patients and Family Factors Associated with the PRSS</b>	<b>134</b>
<b>4.D. SELF- AND OTHER-BLAME</b>	<b>137</b>
<b>4.D.1. The Distribution of the SOBS</b>	<b>137</b>
<b>4.D.2. Clinical Features of the Patients and Family Factors Associated with the SOBS</b>	<b>140</b>
<b>4.E. PRESENTATION OF PROGNOSTIC FACTORS</b>	<b>147</b>
<b>4.E.1. Prognostic Value of Family Expressed Emotion</b>	<b>147</b>
<b>4.E.2. Prognostic Value of Personality Disorders</b>	<b>151</b>
<b>4.E.3. Prognostic Value of the PRSS</b>	<b>152</b>
<b>4.E.4. Prognostic Value of the SOBS</b>	<b>159</b>
<b>4.E.5. Multivariate Evaluation of the Prognostic Factors</b>	<b>161</b>
<b>CHAPTER 5</b>	<b>164</b>
<b>DISCUSSION OF THE RESULTS</b>	<b>164</b>
<b>5.1. Summary of the Results</b>	<b>164</b>
<b>5.2. Limitations of the Study</b>	<b>168</b>
<b>5.3. Discussion of the Results</b>	<b>172</b>
<b>5.3.A. EXPRESSED EMOTION</b>	<b>172</b>
<b>5.3.A.1. The Distribution of Family Expressed Emotion</b>	<b>172</b>

5.3.A.2. Changes in Family Expressed Emotion during the Course of Therapy	174
5.3.A.3. Factors Associated with the Diagnostic Groups	175
5.3.A.4. The Differences in Treatment Outcome for AN and BN Patients	176
5.3.B. PERSONALITY DISORDERS	177
5.3.B.1. The Distribution of the PAS Personality Diagnoses	177
5.3.B.2. Clinical Features of the Patients and Family Factors Associated with Personality Disorders	179
5.3.C. PATIENTS' RESPONSE STYLE TO THERAPIST AND THERAPY	180
5.3.C.1. Reliability of the PRSS	180
5.3.C.2. The Distribution of the PRSS in Families with an Eating Disorder Patient	181
5.3.C.3. Clinical Features of the Patients and family factors Associated with the PRSS	182
5.3.D. SELF- AND OTHER-BLAME	184
5.3.D.1. Reliability of the SOBS	184
5.3.D.2. The Distribution of the SOBS among Families with an Eating Disorder Patient	184
5.3.D.3. Clinical Features of the Patients and Family Factors Associated with the SOBS	188
5.3.E. PROGNOSTIC FACTORS	192
5.3.E.1. Prognostic Value of Family Expressed Emotion	192
5.3.E.2. Prognostic Value of Personality Disorders	193
5.3.E.3. Prognostic Value of the PRSS	194
5.3.E.4. Prognostic Value of the SOBS	196
5.3.E.5. The Relationship between Prognostic Factors	199
CHAPTER 6	201
IMPLICATIONS OF THE STUDY AND CONCLUDING STATEMENTS	201
6.1. The Clinical Implications of the Study	201
6.2. The Theoretical Implications of the Study	204
6.3. Concluding Statements	210
REFERENCES	216
APPENDICES	249



## **LIST OF TABLES**

- 1. Table 2.1. Earlier Diagnostic Criteria for Anorexia Nervosa**
- 2. Table 2.2. DSM-IV Criteria for 307.1 Anorexia Nervosa (APA, 1994)**
- 3. Table 2.3. DSM-IV Criteria for 307.51 Bulimia Nervosa (APA, 1994)**
- 4. Table 3.A.1. Characteristics of 78 Female Eating Disorder Patients**
- 5. Table 3.B.1. Distribution of the PRSS reliabilities for two raters (A & B) in 30 ED patients**
- 6. Table 3.C.1. Distribution of the SOBS reliabilities for two raters (A & B) in 91 ED patients and their relatives**
- 7. Table 4.A.1. Clinical Features of AN and BN Patients**
- 8. Table 4.A.2. Distribution of Expressed Emotion among ED patients' relatives at Intake**
- 9. Table 4.A.3. Distribution of Expressed Emotion among ED patients' relatives at T1 and T2**
- 10. Table 4.A.4. Intercorrelations of family EE scales**
- 11. Table 4.A.5. Correlations of family EE scales**
- 12. Table 4.A.6. The course of family EE Levels over treatment period (T1-T2)**
- 13. Table 4.A.7. The course of family EE Scales over treatment period (T1-T2)**
- 14. Table 4.A.8. Distribution of ED Diagnostic Groups for General Outcome Categories**
- 15. Table 4.A.9. Distribution of ED Diagnostic Groups and Type of Treatment for GOC**
- 16. Table 4.B.1. Distribution of the PAS personality disorder diagnoses in eating disorder subtypes**
- 17. Table 4.B.2. Distribution of the PAS personality disorder clusters in ED subtypes**

18. **Table 4.B.3. Clinical Features of the patients with and without personality disorders**
19. **Table 4.B.4. Distribution of family EE and personality diagnoses in eating disorder patients**
20. **Table 4.C.1. Distribution of the PRSS in ED patients**
21. **Table 4.C.2. Spearman Correlations of the PRSS Scales for AN and BN patients**
22. **Table 4.C.3. Distribution of Clinical Features of ED patients for the PRSS Levels**
23. **Table 4.C.4. Distribution of the PRSS Scales for the PAS Personality Abnormality**
24. **Table 4.C.5. Spearman Correlations of the PRSS Scales with Family EE Scales**
25. **Table 4.C.6. Distribution of Family EE Scales for the PRSS Levels**
26. **Table 4.D.1. Descriptive statistics of the SOBS among ED patients and their relatives**
27. **Table 4.D.2. Intercorrelations of the SOBS scales**
28. **Table 4.D.3. Spearman correlations of parental SOBS scales**
29. **Table 4.D.4. Distribution of the SOBS scales in ED patients with and without personality disorders and their families**
30. **Table 4.D.5. Distribution of the SOBS Scales for the PRSS Levels**
31. **Table 4.D.6. Distribution of the SOBS scales for low and high EE families in ED patients**
32. **Table 4.E.1. Distribution of Family EE Scales at T1 for General Outcome Categories at T2**
33. **Table 4.E.2. Distribution of Family EE Levels at T1 for General Outcome Categories at T2**
34. **Table 4.E.3. Distribution of the PAS Personality Diagnoses for General Outcome Categories**

- 35. Table 4.E.4. Distribution of the PRSS Scales at T1 for General Outcome Categories at T2**
- 36. Table 4.E.5. Distribution of the PRSS Levels at T1 for General Outcome Categories at T2**
- 37. Table 4.E.6. Distribution of the PRSS Levels and Therapy Types for General Outcome Categories**
- 38. Table 4.E.7. Distribution of the SOBS Measures at T1 for General Outcome Categories at T2**
- 39. Table 4.E.8. Forward Logistic Regression Analysis on GOC at T2, Mothers' CC, and Patients' SD and EEn at T1**
- 40. Table 4.E.9. Forward Logistic Regression Analysis on GOC at T2, Mothers' CC and SB, and Patients' SD T1**

## **LIST OF FIGURES**

- 1. Figure 3.A.1. Research Design**
- 2. Figure 4.A.1. Distribution of Parents' CC and EOI at T1**
- 3. Figure 4.A.2. Distribution of Mothers' CC and EOI in AN and BN Patients**
- 4. Figure 4.A.3. Distribution of Parents' CC and EOI at T1 and T2**
- 5. Figure 4.A.4. Distribution of Outcome for AN and BN Patients**
- 6. Figure 4.B.1. Distribution of the PAS Personality Disorders in ED Patients**
- 7. Figure 4.B.2. Distribution of Anxious and Histrionic PDs in AN and BN Patients**
- 8. Figure 4.B.3. Distribution of Relatives' CC to NPD and PD Patients**
- 9. Figure 4.C.1 Distribution of the PRSS in Eating Disorder Patients**
- 10. Figure 4.D.1. Distribution of Parents' SOBS in Eating Disorder Patients**
- 11. Figure 4.D.2. Distribution of the SOBS for Eating Disorder Patients**
- 12. Figure 4.D.3. Distribution of the SOBS for NPD and PD Patients**
- 13. Figure 4.D.4. Distribution of the SOBS for the PRSS Levels (High vs Rest)**
- 14. Figure 4.D.5. Distribution of the SOBS for Low and High EE Parents**
- 15. Figure 4.E.1. Distribution of Parents' CC for Outcome Categories (Poor vs Rest)**
- 16. Figure 4.E.2. Distribution of the PAS Personality Abnormalities for Outcome Categories**
- 17. Figure 4.E.3. Distribution of the PRSS Variables for Outcome Categories**
- 18. Figure 4.E.4. Distribution of the PRSS Levels for Outcome Categories by Different Types of Therapy**
- 19. Figure 4.E.6. Distribution of Self-Blame for Outcome (Poor vs Rest)**

## **PART I**

### **CHAPTER 1**

#### **INTRODUCTION AND OUTLINE OF THE THESIS**

The relationship between family-individual factors and eating disorders is of both practical and theoretical importance. The family- and patient-related variables to be investigated in this study are organized into four areas: family expressed emotion; personality disorders; patient's response style to therapist and therapy; and self- and other-blaming attitudes.

A number of authors (Bruch, 1973; Dare, 1985; Lasegue, 1964; Minuchin, Rosman & Baker, 1978; Selvini-Palazzoli, 1974; Yager, 1981, 1982) have suggested that family factors have a crucial role in influencing the course of eating disorders. The empirical evidence in support of the particular form of family interaction in the families of eating disorder patients, however, is limited. Some family and personal factors are important for the course of eating disorders and response to treatment. The measure of expressed emotion has been extensively used for the investigation of family interaction in different clinical populations. Our understanding of the relationship between family EE and eating disorders, however, is in the early stages of development. Further research with eating disorder patients is needed to clarify the contribution of family factors in the course of eating disorders, as well as treatment outcome.

The importance of personality is widely acknowledged. Many patients presenting for treatment of eating disorders have concurrent personality disorders. A number of factors broadly related to personality have been suggested as predisposing individuals to eating disorders. However, it is difficult to draw meaningful conclusions about premorbid personality characteristics in a disorder

with such significant and wide-ranging physical and psychological consequences.

The relationship between eating disorders and personality disorder is also very interesting because of the implications for treatment. It has been emphasized by some that eating disorders rarely occur in the absence of personality disorders, but others have suggested that eating disorders may occur without primary personality disturbance. Further studies should pursue the prevalence and effect of personality disorders on the course and treatment outcome of eating disorders.

Self-disclosure, the communication of personal information about an individual's self to another, has received considerable attention with respect to the psychotherapeutic process and outcome in different clinical populations other than eating disorders. This is the first attempt to evaluate this important patient-related factor in a sample of eating disorder patients. For this purpose a new measure, the Patient Response Style Scale (PRSS), was developed.

Clinical observations recognize the psychological significance of blame and guilt attributions, in general. With regard to families containing an eating disorder patient, the descriptive data reinforce further the need to consider these feelings as risk or vulnerability factors of some importance. In an effort to assess this shared family and individual factor, a new measure, the Self-and Other-Blame Scale (SOBS), was developed.

This thesis is divided into two parts. **PART I:** Chapter 2 gives an overview in five sections of the many considerations and assumptions about family and personality of eating disordered patients, as well as the disorder itself. First, the psychopathology of AN and BN is discussed by describing the historical development of the concept of AN, BN as a new syndrome, diagnostic criteria and

classification of eating disorders, aetiology, psychodynamic and developmental models of eating disorder, family models of eating disorders, family system models of eating disorders, and Sociocultural models of eating disorders. Second, the concept of EE and the empirical evidence regarding family EE in eating disordered patients are provided. Third, the relationship between personality disorders and eating disorders is presented. Fourth, Self-disclosure and emotional engagement are discussed as part of a patient's response style to therapist and therapy. The fifth section of this chapter concerns self- and other-blame followed by "characterological" and "behavioural" self-blame. Each section will be closed by discussion about the need to study the contribution of family (e.g., criticism, emotional over-involvement, blaming) and personality (personality disorders and patient's response style to therapist and therapy) factors to the perpetuation of eating disorders, and their prognostic value in relation to engagement-outcome in different types of psychological treatments.

Beginning with the "purpose of the research," chapter 3 describes the design and methodology of this thesis. Then, formulation of the main hypotheses of the research will be presented followed by description of the sample, assessment procedure, instruments that were employed in this study including observational and self-report measures, and finally, methods of statistical analysis.

**PART II:** Chapter 4 presents the results of this investigation in five sections. Section A reports the results regarding the distribution of family Expressed Emotion (EE) at intake and at the termination of treatment, changes in family EE during the course of therapy, clinical features of the diagnostic groups, and the distribution of treatment outcome for AN and BN patients. Section B presents the results showing the frequency of personality disorders in this group of eating disordered patients. It also includes a comparison of personality disorders in AN and BN patients,

and the relationship between family EE and personality disorder. In sections C and D the distribution of the results for the Patient Response Style Scale (PRSS) and the Self- and Other-Blame Scale (SOBS) are presented, respectively. These results are discussed in association with clinical and family factors. Section E demonstrates the prognostic value of the family EE, personality disorders, self- and other-blaming attributes, and the PRSS variables. Chapter 5 presents the discussion of the results in five sections. Section A addresses three issues: 1) the distribution of family EE in eating disordered patients; 2) the changes of family EE during the course of therapy; 3) factors associated with the diagnostic groups. Section B presents two issues: 1) the distribution of personality disorder diagnoses in eating disordered patients; 2) clinical and family factors associated with personality disorders. Sections C and D discuss the results concerning the patient's response style to therapist and therapy, and the self- and other-blame, respectively. In section C, three issues are presented: 1) the reliability of the PRSS; 2) the distribution of the PRSS in families with an eating disorder patients; 3) clinical and family factors associated with the PRSS. Section D presents three issues in a similar way: 1) the reliability of the SOBS; 2) the distribution of the SOBS in families with an eating disorder patient; 3) clinical and family factors associated with the SOBS. Section E discusses the prognostic value of 1) family EE; 2) personality disorders; 3) the PRSS; and 4) the SOBS.

The clinical and theoretical implications of the research, as well as limitations of the study are discussed in chapter 6. The thesis is closed with presenting the concluding statements drawn from the results of the study.



## **CHAPTER 2**

### **A REVIEW OF THE LITERATURE**

This chapter reviews the literature of relevance to the subject of this thesis, under the following headings: 1) eating disorders; 2) expressed emotion; 3) personality disorders; 4) patient's response style to therapist and therapy; 5) self- and other-blame.

#### **2.1. EATING DISORDERS**

##### **2.1.1 ANOREXIA NERVOSA: Historical Development of the Concept**

The earliest clinical descriptions of anorexia nervosa (AN) were provided by Richard Morton (1694), Whytt (1764), Louis-Victor Marce (1860), Ernest Charles Lasègue (1873) and William Withey Gull (1874, 1888). Morton described two case histories of an illness that he referred to as “a Nervous Consumption”, caused by, “sadness, and anxious cares”. Whytt (1764) described a case of “a nervous atrophy” in a teenage boy, and made a similar diagnosis by exclusion. In 1860, Louis-Victor Marce saw a group of patients, all female, who presented with gastrointestinal complaints associated with weight loss. Their dyspepsia presented in the form of “absence of appetite or by the uneasiness caused by digestion”. Marce wisely concluded that his patients' food avoidance was psychological in character: “In one word, the gastric nervous disorder becomes cerebro-nervous”. He considered the illness to be “a hypochondriacal delirium”. He wrote that “the patients are no longer dyspeptics - they are insane” (Marce, 1860).

Gull (1874) and Lasègue (1873) formulated their clinical descriptions of “anorexia nervosa” and “l'anorexia hysterique” respectively. They both recognised a disorder associated with severe emaciation and amenorrhoea, inexplicable in terms of known causes of wasting. They were both

extremely cautious about the nature or origin of the mental disorder, while at the same time recognising that there was a morbid mental state (Gull), a mental perversity (Gull; Lasegue) or hysterical anorexia (Lasegue).

The clinical picture described by Gull and Lasegue is remarkably robust and has withstood the test of time. In recent years there has been a shift in emphasis on the nature of the central psychopathology of AN, with a greater stress on the patient's morbid preoccupation with her body weight and her dread of fatness. However, Russell (1985, 1995) has argued that this is best understood as a "pathoplastic" effect of socio-cultural influences which might not be crucial or necessary elements of the clinical entity. Developing and expanding on Russell's idea, Theander (1995) has reviewed the early writings on anorexia nervosa, those of Gull and Lasegue, and also of Marce, and identified those symptoms and signs which show "fidelity" (continuity over time): "the marked preponderance of females and young people among the patients; food refusal; the extreme, often life-threatening emaciation, but also the tendency to recovery; and the denial of illness".

The explanation previously proposed for this apparent oversight on the part of Gull and Lasegue is that the psychopathology of AN has changed between the 1870s and the 1960s (Russell, 1985; Russell & Treasure, 1989). There is, moreover, a strong argument for accepting the original descriptions by Gull and Lasegue as containing the essence of AN. The modifications made by modern observers are mainly to the psychopathological content of the illness. They merely represent changes during recent historical times in response to those influences which have favoured the expression of the patients' sensitivities to the size and shape of their own bodies. For instance, Bruch (1973) suggested that three areas of disordered function can be recognized in the

anorexic patients:

- a) a disturbance of delusional proportions in the body image and body concept.
- b) a disturbance in the accuracy of the perception or cognitive interpretation of stimuli arising in the body, with failure to recognize signs of nutritional need as the most pronounced deficiency.
- c) a paralysing sense of ineffectiveness which pervades all thinking and activities (pp. 251-4).

In his seminal report, “Bulimia Nervosa: An ominous variant of anorexia nervosa,” Russell (1979), described in detail this seemingly new illness and listed its diagnostic criteria.

### **2.1.2. BULIMIA NERVOSA: A New Syndrome**

It has been long recognized that bulimia occurs in some anorexic patients. Gull (1874) wrote: “occasionally for a day or two the appetite was voracious” (p. 133). The account of Ellen West by Binswanger (1959) is that of a young woman torn between the fear of fatness and constant preoccupations with food ingested in large amounts without achieving satiety.

The idea that bulimia might be a feature of a not-uncommon syndrome separable from AN, obesity, or other disorders, seems to have emerged during the 1970s. Different authors sought to describe a disorder occurring at normal weight in which episodic overeating with loss of control was associated with a variety of other features both psychological and behavioural. The definitions varied slightly as did the names which were coined for these ‘new’ disorders (Palmer, 1987, pp. 5-6).

Binge eating, as a distinct pattern of eating in a subset of the obese, was first recognized by Stunkard in 1959 and further described by him in 1976 (Stunkard, 1959, 1976). Hilde Bruch

(1973) reported bulimia, often followed by self-induced vomiting, in about 25% of her cases with primary AN. She also mentioned compulsive eating and eating spells in obese patients, and introduced a new label “thin fat people” for formerly overweight persons who succeeded in becoming thin but still have unsolved problems. Dorr-Zegers (1972) extensively discussed three cases of “hyperphagia followed by vomiting” in women, which he viewed as “a particular type of oral perversion in the female”.

Independently from one another, clinicians became more and more intrigued by a peculiar pattern of overeating they couldn't fit in the existing diagnostic classification. Wermuth, Davis, and Hollister (1977) did not hesitate to present the criteria of a new clinical syndrome, “binge eating syndrome”; Arthur Crisp (1979) elaborated some assertions concerning psychosexual development in obesity, anorexia nervosa, and the intermediate state of “abnormal-normal weight control”.

The term “dietary chaos syndrome” was proposed for a new syndrome in the borderlands of AN, although some subjects may never have been typically anorexic. Palmer (1979) gave three case descriptions (all women between 22 and 29 years of age) of the syndrome that is characterized by a grossly disordered pattern of eating (e.g., bulimia, self-induced vomiting, laxative abuse, eating in secret); a preoccupation with food and eating, and sometimes with weight (the impulse to eat is experienced as out of control and is paralleled by emotional instability); and body weight changes within or above the normal limits (amenorrhea may be present). “The state thus described may be long lasting, resistant to treatment, troublesome and even dangerous” (p. 188). According to Palmer the syndrome represents “an intermediate position between AN with bulimia and vomiting on the one hand and on the other those obese subjects with impulsive overeating”

(p. 190).

With Russell's description of bulimia nervosa in 1979, followed by the DSM-III diagnosis of bulimia, a 'new' eating syndrome found its official acceptance in the scientific world. As Palmer (1987) puts it, it is undoubtedly Russell's description of BN that has reached the status of landmark publication, and not just because of his fortunate choice of a new nosological term (Vandereycken, 1994). Russell's systematic description of the 30 BN patients he had seen between 1972 and 1978 echoes the clinical acuteness and plain clarity of Lasegue's classic paper on AN (Vandereycken & Van Deth, 1990). It took almost exactly one century before a "new" eating disorder was officially born.

The new syndrome of "bulimia nervosa" characterised by episodes of overeating, devices such as induced vomiting which mitigated the fattening effects of food, and a morbid dread of fatness identical with the psychopathology of AN which often preceded it (Russell, 1979).

### **2.1.3. DIAGNOSTIC CRITERIA AND CLASSIFICATION OF EATING DISORDERS**

**Anorexia Nervosa:** Various methods of diagnosis and classification have been suggested for AN.

Bruch (1961) proposed a psychological approach to diagnosis. She described three important features of the illness: body image disturbance, loss of awareness of interoceptive cues, and an overwhelming feeling of ineffectiveness.

In the late 1960s and early 1970s, the English psychiatrists Dally (1969) and Russell (1970) and the American Feighner and his colleagues (Feighner et al., 1972) sought to establish standardized diagnostic schedules for AN. They each proposed schemes by which the physical symptoms and the behavioural and psychological features were combined in various packages (Table 2.1.). All three sets of criteria required food refusal and a menstrual disturbance, but differed significantly in other respects and hence resulted in different diagnostic conclusions.

**Table 2.1. Earlier Diagnostic Criteria for Anorexia Nervosa**

Dally (1969)

- A. Refusal to eat enough to maintain normal weight and/or sustained efforts to prevent ingested food from being absorbed.
- B. Loss of at least 10% of previous body weight.
- C. Amenorrhea of at least 3 months - or if menstruation had been irregular, a period of amenorrhea of at least 6 months.
- D. Onset between age 12 and 39 years.
- E. No organic disease, serious affective disorder, or schizophrenia.

Russell (1970)

- A. Behaviours aimed at achieving weight loss - starvation, vomiting, laxative abuse.
- B. An endocrine disorder - amenorrhea in the female and loss of sexual interest in the male.
- C. A characteristic psychopathology, manifested by a morbid fear of becoming fat, is often accompanied by a distorted judgment by the patient of her own body size.
- D. A specific degree of weight loss is required - 20% of standard body weight.

Feighner et al. (1972)

- A. Onset prior to age 25 years.
- B. Anorexia with weight loss of at least 25% of original body weight.
- C. A distorted, implacable attitude towards eating, food or weight that overrides hunger, admonitions, reassurances, or threats. This could include: denial of the illness with a failure to recognize nutritional needs; apparent enjoyment of weight loss and food refusal; a desired body image of extreme thinness; unusual handling or hoarding food.
- D. No known medical illness.
- E. No other known psychiatric disorder.
- F. At least two of the following: amenorrhea, lanugo hair, bradycardia (persistent resting pulse of 60 or less).

Russell's criteria for AN are still embedded in the latest DSM criteria. Table 2.2. shows the DSM-IV diagnostic criteria for AN.

**Table 2.2. DSM-IV Criteria for 307.1 Anorexia Nervosa (APA, 1994).**

- A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).
- B. Intense fear of gaining weight or becoming fat, even though under-weight.
- C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
- D. In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration.)

*Specify type:*

**Restricting Type:** during the current episode of Anorexia Nervosa, the person has not regularly engaged in binge-eating or purging behaviour (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas).

**Binge-Eating/Purging Type:** during the current episode of Anorexia Nervosa, the person has regularly engaged in binge-eating or purging behaviour (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas).

Individuals with AN refuse to maintain a body weight that is above a minimally normal level for age and height (Criterion A). It suggests that the individual weighs less than 85% of that weight that is considered normal for that person's age and height. An alternative and somewhat stricter guideline (used in the ICD-10 Diagnostic Criteria for Research) requires that the individual have a body mass index (BMI) (calculated as weight in kilograms/height in meters<sup>2</sup>) equal to or below 17.5 Kg/m<sup>2</sup>. These cut-offs are provided only as suggested guidelines for the clinician, since it is unreasonable to specify a single standard for minimum normal weight that applies to all individuals of a given age and height.

The criterion of psychopathology characterized by a morbid fear of becoming fat in Russell's (1979) classification corresponds with the criteria B and C in DSM-IV

Intense fear of gaining weight or becoming fat (Criterion B) in AN individuals is usually not alleviated by the weight loss. In fact, concern about weight gain often increases even as actual weight continues to decrease. They also have a disturbance in the way that they experience their body weight or shape (Criterion C). This may include an undue influence of body weight or shape on their self-evaluation or a denial of the seriousness of the low weight.

An earlier version of criterion C for AN in DSM-III was described as a “body image disturbance”. This resulted in many studies of a narrow definition of body image related to visual self-perception. Since these studies show that many anorectics do not overestimate their sizes, and that over estimation is not unique to those with AN (Lindholm & Wilson, 1988; Touyz & Beumont, 1988) the criterion was reworded in DSM-III-R to focus on attitudinal and affective dimensions of body image. Because this newly worded criterion of “overconcern with body size and shape” had significant overlap between AN patients and the general female population, it was revised in DSM-IV to emphasize the central concern of weight and shape in the evaluation of the self in addition to a reference to the denial of the serious consequences of weight loss. These two additions to the body dissatisfaction criterion should reduce overlap with the general population. Criteria B and C specify the core psychological disturbance, sometimes referred to as the specific psychopathology of the disorder.

The inclusion of amenorrhea as a necessary condition is intended to pick out the “secondary endocrine disorder” (Garfinkel & Garner, 1982). The criterion of evidence of an endocrine disorder in Russell’s (1970) classification - in females, amenorrhea, and in males, loss of sexual potency and interest - corresponds with the criterion D in DSM-IV. There is no doubt that amenorrhea is a common feature in AN, and that in part it is based on loss of body weight and fat.



But the presence of amenorrhea is incompletely understood and can occur in a significant minority of women with AN before there is any real weight loss (Theander, 1970).

In response to numerous studies documenting reliable differences between anorectic patients who engage in bingeing/purging behaviour and those anorectic patients who merely restrict food intake (Casper et al., 1980; Garfinkel et al., 1980; Michalide & Andersen, 1985; Strober et al., 1982; Vandereycken & Pierloot, 1983; Yellowlees, 1985), the DSM-IV criteria have defined an AN restrictor type and AN binge/purger type:

**Restricting Type-** This subtype describes presentations in which weight loss is accomplished primarily through dieting, fasting, or excessive exercise. During the current episode, these individuals have not regularly engaged in binge eating or purging.

**Binge-Eating/Purging Type-** This subtype is used when the individual has regularly engaged in binge eating or purging (or both) during the current episode. Most individuals with AN who binge eat also purge through self-induced vomiting or the misuse of laxatives, diuretics, or enemas. Some individuals included in this subtype do not binge eat, but do regularly purge after the consumption of small amounts of food. It appears that most individuals with Binge-Eating/Purging Type engage in these behaviours at least weekly, but sufficient information is not available to justify the specification of a minimum frequency.

**Bulimia Nervosa:** Although there were earlier accounts of a few patients who might warrant a retrospective diagnosis of bulimia, and although bulimic episodes had been reported regularly in obese patients and in the purging form of AN, it was only in the late 1970s that attention was focused on persons at relatively normal weight whose most obvious problem was that their eating patterns were chaotic, with semistarvation alternating with uncontrolled overeating (Russell,

1979).

When Russell (1979) first proposed diagnostic criteria for BN he suggested three components:

1. a powerful and intractable urge to overeat, resulting in episodes of overeating
2. avoidance of “fattening” effects of food by inducing vomiting or abusing purgatives or both
3. a morbid fear of becoming fat

These criteria have undergone various modifications to the most recent, in DSM-IV (Table 2.3.).

**Table 2.3. DSM-IV Criteria for 307.51 Bulimia Nervosa (APA, 1994).**

- A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
- (1) eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
  - (2) a sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).
- B. Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.
- C. The binge eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for 3 months.
- D. Self-evaluation is unduly influenced by body shape and weight.
- E. The disturbance does not occur exclusively during episodes of Anorexia Nervosa.

*Specify type:*

**Purging Type:** during the current episode of Bulimia Nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.

**Nonpurging Type:** during the current episode of Bulimia Nervosa, the person has used other inappropriate compensatory behaviours, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.

Binge eating is clearly defined in DSM-IV as a necessary diagnostic criterion for BN: eating in a discrete period of time an amount of food that is definitely larger than most individuals would eat under similar circumstances (criterion A1).

Differentiating binge eating from continual 'snacking' throughout the day, a "discrete period of time" refers to a limited period, usually less than 2 hours.

The sense of losing control is a significant subjective aspect that occurs in binge eating (criterion A2). An individual may be in a frenzied state while binge eating, especially early in the course of the disorder.

Another essential feature of BN is the recurrent use of inappropriate compensatory behaviours to prevent weight gain (criterion B). Criterion C, the threshold criterion, is intended to exclude subclinical or partial cases.

Individuals with BN place an excessive emphasis on body shape and weight in their self-evaluation, and these factors are typically the important ones in determining self-esteem (criterion D). Individuals with this disorder may closely resemble those with AN in their fear of gaining weight, in their desire to lose weight, and in the level of dissatisfaction with their bodies. However, a diagnosis of BN should not be given when the disturbance occurs only during episodes of AN (criterion E).

The DSM-IV criteria have defined two subtypes to specify the presence or absence of regular use of purging methods as a means to compensate for the binge eating:

**Purging Type-** This subtype describes presentations in which the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas during the current episode.

**Nonpurging Type-** This subtype describes presentations in which the person has used other inappropriate compensatory behaviours, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas during the current episode.

#### **2.1.4. AETIOLOGY**

It is widely accepted that a combination of biological, psychological and social factors are of importance (Garfinkle & Garner, 1982; Katz, 1985). Currently there are several theories, organized along different conceptual levels, that attempt to explain this phenomenon. They are logically not mutually exclusive. While they overlap to some degree, their main thrust and conceptualization are distinct. The recent findings on psychodynamic, developmental, family and family systems, and sociocultural models of eating disorders will be reviewed.

##### **2.1.4.1. PSYCHODYNAMIC AND DEVELOPMENTAL MODELS OF EATING DISORDERS**

Older psychoanalytic concepts of anorexia nervosa have relied “heavily on theories about fixation at the oral level of psychosexual development, on regression in instinctual drives from the genital level of development, and on symptom formation around oral conflicts” (Ross, 1977, p. 424). Waller et al. (1940), for example, emphasized the role of symbolization of impregnation fantasies involving the gastrointestinal tract. These oral impregnation fantasies were presumed to be associated with marked guilt and the AN was thought to be a defence against these (Blitzer,

Rollins & Blackwell, 1961; Sandler & Dare, 1970). Moulton (1942), Lorand (1943), and Szyrynski (1973) have all described cases in which similar symbolic distortions were purported to produce the desire for weight loss. Benedek (1936) described the analysis of a patient with oral aggression which was aimed at the mother's breast. Thoma (1967) is representative of this drive-related psychoanalytic viewpoint. He theorized that AN derives from the patient's abandonment of the genital stage of development with a predominance of "oral ambivalence." He emphasized that the primary defence is against drive representatives: the sexual fears are displaced by concerns about body size.

More recent psychodynamic formulations have de-emphasized drive theory and have been focused on early object relations (Bruch, 1973; Ross, 1977; Selvini Palazzoli, 1974; Sours, 1974, 1980). These have been extremely useful observations, particularly when, as Kramer (1974) cautions, they don't overemphasize pathological mothering to the neglect of the child's role in the disturbed parent-child interaction.

Of major importance in this regard have been Bruch's hypotheses about AN. Bruch (1973) described AN as developing around three related ego disturbances - distortions of body image, internal perception, and a sense of ineffectiveness. Her central theme is that the search for self-mastery and autonomy is maladaptively pursued through control over one's body. Bruch's conception of this development involves familial and mother-child interactions as well as factors within the child herself. The mother-child interactions are significant from birth on. Bruch has suggested that anorexics have serious deficits in self-initiated behaviours. In part, these are related to a neglect of appropriate external responses to the child's inner states. Bruch (1973) has described the process of differentiating internal process thus:

“If confirmation and reinforcement of his own, initially rather undifferentiated needs and impulses have been absent, or have been contradictory or inaccurate, then a child will grow up perplexed when trying to differentiate between disturbances in his biological field and emotional and interpersonal experiences, and he will be apt to misinterpret deformities in his self body concept as externally induced. Thus he will become an individual deficient in his sense of separateness, with ‘diffuse ego boundaries’, and will feel helpless under the influence of external forces” (p. 56). Bruch feels that the anorexic’s fear of having no self-control can be related to these early experiences. “Such a child does not feel she is living her own life, but feels deprived of inner guide posts, helpless under the influence of internal urges and external demands, and like being the property of her parents” (1979, p. 107). In addition, Bruch feels that the preanorexic child’s own conceptual development does not advance appropriately and this provides a further contribution to the development of the disorder.

Selvini Palazzoli (1974) has independently evolved a similar formulation of the genesis of AN. Like Bruch, she avoids the emphasis on orality and focuses on the helplessness of the ego. According to this view, the individual perceives her body as not belonging to her; rather she concretely perceives her body as a threat which must somehow be controlled. Following Fairbairn’s model of object relationships, Selvini Palazzoli (1974) suggests that the anorexic experiences her body as “the maternal object, from which the ego wishes to separate itself at all costs” (p. 90). The anorexic incorporates the feared maternal object in order to control it. Central to this model is the concept of a mother who rewards compliance to her wishes, is overprotective, and is unable to allow separation in the child.

Recent research has begun to examine the relationship between abnormal eating attitudes and

childhood care patterns. Utilizing the Parental Bonding Instrument (PBI; Parker et al., 1979), several studies have shown that anorexics and bulimics recalled that their parents were low in care, but had normal levels of protection (Calam et al., 1990; Palmer, Oppenheimer & Marshall, 1988; Steiger et al., 1989).

#### **2.1.4.2. FAMILY MODELS OF EATING DISORDERS**

The principle of family involvement was outlined in detail by Lasègue (1873) more than a century ago. In his seminal 1873 account of AN, Lasègue wrote: “The relatives and friends begin to regard the case as desperate. It must not cause surprise to find me thus always placing in parallel the morbid condition of the hysterical subject and preoccupations of those who surround her. These two circumstances are intimately connected, and we should acquire an erroneous idea of the disease by confining ourselves to an examination of the patient ... The moral medium amidst which the patient lives exercises an influence which it would be equally regrettable to overlook or misunderstand” (p. 152).

Following Lasègue and Gull’s publications, the notion that family environment was fertile ground for the development of AN was accepted by many as an indisputable fact (Vandereycken & Van Deth, 1990).

By the same token, in a rarely cited paper published more than a decade before those of Lasègue and Gull, Louis Victor-Marce (1860) speculated that “hereditary antecedents” played a formative role in predisposing certain young people to a form of hypochondriacal insanity, the description of which is identical to the entity AN (Silverman, 1989). Still, Marce believed, as did Gull and Lasègue, that certain deviant elements of the family’s behaviour loomed large in perpetuating the illness and in impeding proper and effective treatment (Halmi, 1992).

Although this attitude implied, at least indirectly, the conception that parents played a pathogenic part in the eating disorder, this view was not explicitly elaborated. But, in the 19th century, the influence of familial factors and interactions on the development of mental illness was rarely explored (McPeak, 1975). The recognition of the importance of interpersonal dynamics within the family unit came only after the adoption and exploration of Freud's insights. Until world war II, psychoanalytic investigators dominated the literature and, hence, the familial role in the development of AN was narrowed to a one-sided analysis of the pathogenic mother-daughter interaction.

#### **2.1.4.3. FAMILY SYSTEM MODELS OF EATING DISORDERS**

The family system models from different family therapists (most notable of whom have been Mara Selvini Palazzoli and Salvador Minuchin) have postulated that certain family relationships are closely related to the development and maintenance of psychosomatic syndromes in children.

Selvini Palazzoli (1974) gradually shifted her focus from a model which emphasized mother-daughter relationships to that of the entire family's interactions. Her group from Milan evolved a systems approach in describing AN. In their clinical studies of families with an anorexic child, the Milan group have described certain predominant characteristics (Selvini Palazzoli, 1974, pp. 205-16):

- 1) Communication disorders in families in which the anorexic symptoms are complicated by other symptoms, like bulimia and violence.
- 2) The rejection of messages sent by others.
- 3) The rejection of personal leadership; precisely because every family member 'effaces' himself for the sake of the rest, no one member is really prepared to assume responsibility when



something goes wrong.

- 4) The central family issue relates to the formation of “covert coalitions”. Each member of the family is “married” to two persons; the mother to her husband and daughter, the father to his wife and daughter, and the daughter to both of her parents. This works fairly well for the parents but not for the daughter, who is expected to distribute her attention as equitably as possible. Consequently, she has no energy to build a life of her own, or to risk open rebellion. Selvini Palazzoli has referred to this triangle as “three-way matrimony” (1974, p. 211).
- 5) There is a spirit of “self-sacrifice.” All decisions are for the good of someone else, so decisions are never attributed to personal preferences.
- 6) The marital relationship is felt to be characterized by a facade of unity which generally conceals a profound underlying disillusionment. Each partner is thought to compete for a sense of moral superiority, i.e., for who has made the greater sacrifices for the sake of the family. Both this and the spirit of self-sacrifice have previously been emphasized by Bruch.

The structural family theory of Salvador Minuchin and colleagues (Minuchin et al., 1975, 1978) is the most comprehensive account of families of eating disordered patients. Minuchin and his co-workers have written extensively on the psychosomatic family (Minuchin, 1974; Minuchin et al., 1975, 1978). They advocated an open systems model for psychosomatic illness, AN included.

This system included parts such as extrafamilial stress, family organization and functioning, the vulnerable child, physiological and biochemical mediating mechanisms and the symptomatic child. The system could be activated at any point and the parts could affect each other. Nevertheless, these authors have emphasized almost exclusively the family pathology in this system and stated that “when significant family interaction patterns are changed, significant changes in the symptoms

of the psychosomatic illness also occur” (Minuchin et al., 1978, p. 21).

According to Minuchin et al. (1978), the traditional linear model links together a variety of factors which converge on the individual and the locus of pathology is thought to be in the individual (p. 83). The systems model, by contrast, emphasizes the interdependence and circular interactions of forces: “This model posits a circular movement of parts that affect each other. The system can be activated at any number of points. The activation and regulation of the system can be done by system members or by force outside the system” (Minuchin et al., 1978, p. 20).

According to this view, an individual’s behaviour is simultaneously caused and causation; a beginning and an end are defined only as arbitrary framing points. This model, the so-called “psychosomatic family”, has three factors:

“First, the child is physiologically vulnerable; Second, the child’s family has four transactional characteristics: enmeshment, overprotectiveness, rigidity and a lack of conflict resolution. Third, the sick child plays an important role in the family’s pattern of conflict avoidance; and this role is an important source of reinforcement for his symptoms” (Minuchin et al., 1975, p. 1033).

Enmeshment refers to an extreme form of proximity with weak boundaries between subsystems in the family/family subsystems. Family members often intrude on each other’s thoughts and feelings. There is excessive togetherness and sharing which result in a lack of privacy. Individuals tend to have a weak definition of self. In this framework, loyalty and protection take precedence over autonomy and self-realization.

Overprotectiveness is defined as a high degree of concern for other’s welfare in the family. “In such families, the parents’ overprotectiveness retards the children’s development of autonomy,

competence, and interests or activities outside the safety of the family. The children, in turn, particularly the psychosomatically ill child, feel great responsibility for protecting the family” (Minuchin et al., 1978, p. 31). Because parents are so preoccupied with the child’s behaviour, the child becomes more conscious of herself and other people’s expectations - she becomes a “parent watcher”. The denial of self for another’s benefit and indirect expression of one’s wishes are very similar to Selvini Palazzoli’s account of the family spirit of self-sacrifice and lack of leadership or personal responsibility for one’s actions.

Rigidity is a concept that bears upon the degree of adaptability of the family interaction. When external stress (e.g., changes in occupation) or internal family changes (e.g., children reaching adolescence) induce the need for a change of the usual transactional patterns, these families experience great difficulty and attempt to maintain a status quo.

Lack of conflict resolution refers to an absence of negotiation in the family, which can take either the form of avoidance of all conflicts because of the myth of family unity, or the form of continuous quarrelling without ever reaching a solution because of constant interruptions and subject changes. Selvini Palazzoli has also emphasized this point.

The child’s involvement in parental conflict, finally, is considered to be a key factor in the development and maintenance of psychosomatic symptoms. Minuchin et al., (1978) differentiate three patterns of involvement. In the first two patterns “triangulation” and “parent-child coalitions”, the spouse dyad is in open conflict. In triangulation, the child is put in such a position that she cannot express herself without siding with one parent against the other. In the parent-child coalition pattern, there exist a fixed coalition with one parent against the other. In the third

pattern, “detouring”, the parents submerge their conflicts in a posture of blaming or protecting their sick child. The child thus becomes an “avoidance circuit”, who is defined as their only problem but in fact has the role of keeping them together. The child’s involvement in the parental conflict was also observed by Selvini Palazzoli who saw the child as the arbitrator, mediator, or secret ally of mother or father.

Similar to Minuchin’s psychosomatic family model, Stierlin and his colleagues developed a theoretical framework for psychosomatic disorders in general (Stierlin, 1981, 1983; Winawer, 1983; Wirsching & Stierlin, 1985). Stierlin (1981) describes a model of three levels at which a child can be bound to parents. In the first, there is dependence on parents for gratification of the most primitive needs and no move toward separation. In the second level, the child is bound through cognitive confusion. She is told how much she is loved, and can not recognize or reconcile hostile, rejecting, or ambivalent behaviour by the parents. In the third level, the child’s loyalty to the family is exploited, and guilt is induced at transfer of affection away from the family to peers. Stierlin and Weber (1989) also confirm the lack of individual boundaries (similar to Minuchin’s concept of enmeshment), loyalty to family, and delegation (a tendency toward self-sacrifice) in anorexic families. These dynamic interaction features reoccur regularly in the family.

#### **2.1.4.4. SOCIOCULTURAL MODELS OF EATING DISORDERS**

Eating disorders are typically reported in young women from predominantly higher socio-economic classes in western industrialized society.

Since Gull’s and Lasegue’s initial descriptions, it has been repeatedly noted that AN occurs much more commonly in females, and usually develops during adolescence (Bemis, 1978; Beumont et al., 1972; Bruch, 1973; Crisp, 1970; Crisp & Toms, 1972; Jones et al., 1980; Kendell et al.,

1973). Over a century ago, Fenwick (1880) hinted at the role of social forces when he described AN as principally a disorder of the upper classes. Since then this over-representation in the upper social classes has frequently been observed (Bruch, 1973; Buhrich, 1981; Crisp, 1965; Crisp, Palmer & Kalucy, 1976; Jones et al., 1980; Miyai et al., 1975; Morgan & Russell, 1975).

As mentioned by Garfinkel and Garner (1982), these observations that AN occurs with a particular age, sex, and social class distribution suggest that sociocultural factors may be important determinants of the disorder in those who are otherwise vulnerable or predisposed to it. Furthermore, there is growing evidence that AN is increasing in frequency (Duddle, 1973; Halmi, 1974; Jones et al., 1980; Kendell et al., 1973; Sours, 1969; Szmukler et al., 1986; Thender, 1970; Willi & Grossmann, 1983).

Several interweaving lines of epidemiological evidence lend support to the importance of social factors in the origins of AN. From around 1960, an increase of cases alerted clinicians in many western countries to the possibility that societal changes might underpin the change (Theander, 1970). Later observations in other settings supported the role of societal pressure to be thin. Very few cases of anorexia presented to clinicians in non-western cultures where female ideals were not linked to thinness (DiNicola, 1990). In contrast, in some high-risk groups such as dancers, fashion models and athletes, thinness in young women was even more required than in society generally (Garner & Garfinkel, 1980).

In the last decades, the pronounced change in the morphology of eating disorders manifest in the emergence of bulimia nervosa has brought speculation that societal forces influence not only rates of eating disorders, but also their syndromal form.

## **2.2. EXPRESSED EMOTION**

### **2.2.1. THE CONCEPT OF EXPRESSED EMOTION**

The concept of Expressed Emotion (EE) has become increasingly well known in recent decades. Interest in the emotional attitudes of relatives living with psychiatric patients received impetus as a result of the observation by Brown et al. (1958) that chronic male schizophrenic patients who had returned from extended hospitalization to parents or wives relapsed significantly more often than those returning to other relatives or to lodgings. Given the confirmation of part of the original findings, Brown et al. turned their attention toward improving the ratings of emotion, establishing reliable techniques of measurement, and investigating issues of validity (Brown & Rutter, 1966; Rutter & Brown, 1966). The results was the development of the Camberwell Family Interview (CFI), the semi-structured interview in use today. EE attempts to capture the emotional attitude of a key relative towards the patient during the interview.

### **2.2.2. WHY EXPRESSED EMOTION IS USEFUL FOR THIS STUDY**

Problems in dealing with conflict is identified in families containing an anorexic (Bruch, 1973; Minuchin et al., 1975, 1978; Selvini Palazzoli, 1974) and bulimic (e.g., Schwartz, Barret, & Sabs, 1985) patient. The measure of expressed emotion has been extensively used for the investigation of family interaction in different clinical populations (e.g., Brown et al., 1962; Leff & Vaughn, 1985) and to shed light on the clinical descriptions of the families with an eating disorder patient (Dare et al., 1994; Eisler, Szumukler, & Dare, 1985). Work carried out over the last decades has also provided good evidence that the concept of EE is both valid and reliable.

EE would therefore be an appropriate instrument for investigating levels of family conflict and their relationship to treatment response.

In this study EE is used to investigate the contribution of family factors in the course of eating disorders. A brief review on EE measures and research, then are presented under the headings of “measures of EE” and “the nature of EE.”

### **2.2.3. MEASURES OF EXPRESSED EMOTION**

The EE measure consists of 5 independent variables rated from audiotape-recordings of interviews with key relatives. The 5 subscales are of two kinds: frequency counts and global scales. The frequency counts, Critical Comments (CC) and Positive Remarks (PR), correspond to the number of distinctive and clearly defined statements. The global scales, Hostility (H); Emotional Over-Involvement (EOI) and Warmth (W), are made up of an overall judgement based on the total interview. Of the five scales to be described, critical comments and emotional over-involvement have proved the most important for relapse of schizophrenia. These two scales, together with hostility (which is rarely found except in association with high criticism), comprise the key components of the index of EE used to predict outcome in several studies of schizophrenia.

High EE refers to a rating above cut-off point for one or combinations of the subscales CC, H and EOI. Only demonstrated feelings, not the interviewer’s interpretation, justify ratings. Traditionally, a family is defined as high EE even if only one member is so rated. The cut-off points are not standard. They have mainly arisen either as medians, or as the most predictive level for that sample. This has meant that cut-offs vary. This continues to cause problems of comparability across studies, although as Kavanagh (1992) points out: “the data do not support the contention that EE results are significantly affected by changing criteria” (p. 603).

More recently investigators into schizophrenia have used different criteria to dichotomize EE. A cut-off point of greater than or equal to 3 is now more commonly used for the EOI scale, for CC the cut-off point of greater than or equal to 6 is maintained (Leff et al., 1987, 1990; McCreadie & Phillips, 1988; Tarrier et al., 1988). For other disorders such as depression or eating disorders cut-off points of 2 or 3 CC have generally been used (e.g., Vaughn & Leff, 1976a).

#### **2.2.4. THE NATURE OF EXPRESSED EMOTION**

As operationally defined, expressed emotion includes measures of criticism, hostility, and emotional over-involvement. Do these three factors reflect a single underlying construct, or do they represent several constructs that have been arbitrarily brought together under the expressed emotion umbrella? The data suggest that hostility and criticism may represent degrees of the same tendency toward fault-finding, since hostility is never found without a high number of critical comments. Emotional over-involvement, however, does not appear as closely linked. It is never seen among spouses, and it occurs predominantly among mothers.

Examination of the Brown et al. (1972) data revealed that the relationship between EOI and relapse did not reach significance among the 55 parental families, of which 13 were high in EOI. Vaughn et al. (1984) reported significant correlations between EOI and measures of poor outcome but pointed out that this was almost entirely due to the high frequency of EOI among parents of patients who never remitted after discharge. In the study by Vaughn et al. (1984) the correlation between the two components was 0.30 for fathers, but only -0.03 for mothers. Miklowitz and colleagues (1983) have shown that high-EOI, but not critical comments, is associated with a history of poor premorbid psychosocial functioning. Thus it is possible that the relationship between high-EOI and relapse is a result of the fact that both correlate with



premorbid functioning.

Psychophysiological studies (Sturgeon et al., 1984; Tarrier & Barrowclough, 1987; Tarrier et al., 1979, 1988) failed to distinguish between the two components of EE from the patient's psychophysiological response to the presence of the relative. However, this does not mean that the variables are equivalent in other respects. Hooley (1985), on the other hand, has proposed an alternative hypothesis that criticism and EOI may both reflect a single underlying personality pattern in high-EE relatives, in which their mechanism for coping with chronic illness is an attempt to exert increased control over the patient.

The limited correlation between EOI and criticism has probably had little impact on the predictive studies. Criticism usually exerts more power over EE classification than does EOI, because of its greater frequency in most samples (e.g. Brown et al., 1972; McMillan et al., 1986; cf. Vaughn et al., 1984). There is also a substantial overlap between households that are above the cut-off points of EOI and criticism (Brown et al., 1972). A low correlation between the components as continuous variables is of little detriment to a dichotomous EE assessment.

Among the EE variables, criticism seems to make the greatest contribution to relapse in most of the studies that have examined the issue (e.g. Barrelet et al., 1990; Hogarty et al., 1986; McMillan et al., 1986). However, some studies have found hostility (Parker et al., 1988; Leff et al., 1990a) or EOI (Moline et al., 1985) to be more sensitive predictors. The relative effects of components may differ across samples according to their relative frequency and the sensitivity of the assessment to cultural differences in their expression.

### **2.2.5. FAMILY EXPRESSED EMOTION IN EATING DISORDER PATIENTS**

Research into the influence of family interaction, as measured by EE variables, in schizophrenia dates back to the works of Brown et al. (1958, 1962). Since then, several studies have investigated the influence of family EE in either schizophrenia (Barrelet et al., 1990; Brown, Birley & Wing, 1972; Karon et al., 1987; Leff et al., 1987; Leff & Vaughn, 1981; Moline et al., 1985; Nuechterlin et al., 1986; Parker G., Johnston P. & Hayward L., 1988; Tarrier et al., 1988; Vaughn et al., 1984; Vaughn & Leff, 1976a) or the other diagnostic populations including depressive neurosis (Hooley, Orley & Teasdale, 1986; Hooley & Teasdale, 1989; Vaughn & Leff, 1976a), bipolar illness (Miklowitz et al., 1986; Miklowitz et al., 1988; Priebe, Wildgrube & Muller-Oerlinghausen, 1989), post natal mental illness (Marks et al., 1992), elderly patients with senile dementia (Bledin et al., 1990; Gilhooley & Whittick, 1989; Orford, O'Reilly & Goonatilleke, 1987; Vitaliano et al, 1989), mental handicap (Greedharry, 1987), stroke patients (Weddell, 1987), obesity/weight reduction (Fischmann-Havstad & Marston, 1984; Flangan & Wagner, 1991; Havstad, 1979;), inflammatory bowel disease, sexual abuse of children, autism, Parkinson's disease (Vaughn, 1989), children with attention deficit hyperactivity disorder (ADHD; Marshall et al., 1990), and disturbed adolescents (Valone et al., 1983).

In eating disorders, a number of studies have used EE measures to investigate family interaction, prognostic value of EE, the role of EE in dropping out of treatment, and variability of EE over the course of therapy. Using an individual standard interview (CFI), several studies (Szmukler et al., 1985; Van Furth, 1991; Van Furth et al., 1996) reported that EE scales discriminate between families of anorexics and families of bulimics. Dare et al. (1994), however, failed to find significant differences between the two types of families on EE ratings as measured by the Standardized Clinical Family Interview (SCFI).

In the comparison between mothers' and fathers' EE scores of eating disordered patients, some findings show that mothers' CC (Szmukler et al., 1985) and EOI (Szmukler et al., 1985; Dare et al., 1994) are significantly more than those of fathers.

Little is known about the prognostic value of EE in eating disorders. It has been shown that the levels of family CC at the beginning of therapy can predict the treatment outcome (Le Grange et al., 1992b; Van Furth et al., 1996) and treatment dropout (Dare et al., 1990; Szmukler & Dare, 1991; Szmukler et al., 1985). Using the same instrument, however, Van Furth (1991), failed to find any association between EE variables and dropping out of treatment.

More consistently, intervention studies (Le Grange et al, 1992b, 1992c; Van Furth, 1991; Van Furth et al., 1996) have shown that the level of EE variables is changed during the course of therapy.

Studies (Berkowitz, 1987; Le Grange, 1992a; Szmukler et al., 1987) have shown a correspondence between EE measured in an individual and a family setting. Szmukler et al. (1987) found that, of the EE scales used, Critical Comments (CC) were highly correlated between the two settings, Emotional Over-Involvement (EOI) showed a modest correlation, Warmth (W) was highly correlated for mothers only, and the number of Positive Remarks (PR) was unrelated across the individual and family interviews. Although these findings suggest validity of the EE ratings in the family settings, more research is required to establish the reliabilities of these ratings.

Our understanding of the relationship between family EE and eating disorders is in the early stages of development. Further research with eating disordered patients is needed to clarify the family

EE patterns, and to establish whether the levels of EE and particularly CC are different in families containing an anorexic or bulimic patient. Further studies are also required to be directed toward understanding the value of EE in prediction of treatment outcome, and the variability of the levels of EE in relatives of eating disordered patients during the course of therapy.

It will be investigated to what extent treatment outcome can be predicted from family EE at intake (T1), and for what kind of therapy family EE could be the best predictor of outcome. It will be also examined whether changes in family EE over the treatment period may be predicted by changes in the patients' symptomatology.

### **2.3. PERSONALITY DISORDERS**

The importance of personality is widely acknowledged. The relationship between personality and symptoms (Kendell & Discipio, 1968), prognosis (Gittleson, 1966; Rosen, 1957), and response to treatment (Tyrer et al., 1983) is recognized and in the multi-axial classification system of DSM-III, formal recognition is given by including personality as the second axis.

The relationship between personality and psychopathological conditions is intriguing and complex. Akiskal et al. (1983) have outlined several possible relationships or "directions of association" between personality and psychopathology. The first considers personality to provide a characterological *predisposition* to the development of a disorder. In the eating disorders literature, this is illustrated by Bruch (1973) who has emphasized the premorbid personalities of eating disordered individuals as critical etiological factors in the development of AN.

A second hypothesized relationship between personality and psychopathology emphasized the *pathoplastic* or *modifying* influence of personality on the expression of psychological disorders.

As such, personality is not considered a predispositional or etiological factor in the disorder, but instead shapes and colours its expression. As Akiskal (1984) has noted, the depressed individual with hysterical personality features appears quite different from the depressed individual with obsessive-compulsive features. The pathoplastic hypothesis has recently been acknowledged in the eating disorder literature. Garfinkel and Garner (1982), for example, concluded that poorer outcome in AN was associated with unstable and neurotic personality traits, increased somatic and obsessional characterization, and bulimic symptoms.

A third way of conceptualizing the relationship of personality and disorder is to assume that personality factors are primarily *complications* of the disorder. Such a position implies that personality attributes are sequelae of a psychiatric disturbance and lack pathogenetic significance. Fairburn (1984) illustrates this by indicating that depression is improved in BN when the eating symptoms are brought under control. The complication hypothesis has been addressed to a limited extent in the eating disorders literature, for instance, by comparing the personality profiles of the same patients when malnourished and when weight is restored.

Finally, a fourth direction of association is the notion that personality attributes are simply *attenuated* or *subclinical* forms of actual psychiatric disorders. For example, Akiskal (1984) has stated that schizotypal personality disorder probably represents an attenuated form of schizophrenia. Although such a position highlights the contribution of underlying genetic processes, Hirschfeld and Klerman (1979) note that subclinical expression of psychiatric disorder may also present subclinical manifestations of familial or developmental processes. While it is possible to visualize a schizotypal personality disorder as an attenuated variant of a schizophrenic disorder, it is much more difficult to imagine personality characteristics associated with eating

disorders as a subclinical form to AN or BN. In the former instance, the differences can be concealed as quantitative, but in the latter they seem dramatically qualitative. Of the four relationships described, the subclinical hypothesis seems least relevant to the eating disorders (Swift & Wonderlich, 1988).

Personality is a very broad construct and I will approach it just in terms of personality disorders.

### **2.3.1. PERSONALITY DISORDERS IN EATING DISORDER PATIENTS**

While no single personality type has been associated with eating disorders (Garfinkel & Garner, 1982; Garner et al., 1985), clinical observation (e.g., Bruch, 1973) and psychometric studies (e.g., Garner, Olmsted, & Garfinkel, 1983 ; Garner et al., 1985; Strober, 1983) suggest that particular features of personality are common in subtypes of eating disordered patients. Extrapolation from these data to the area of personality disorders suggests that while there might be diversity of personality types associated with eating disorders, a few may be predominant. Early work in this area focused on grouping subjects into various personality patterns according to their predominant defence mechanisms and personality traits. For example, Lesser et al. (1960) categorized the “adjectival descriptions” from charts of 15 patients with AN into three personality categories: hysterical, obsessive-compulsive, and schizoid. Follow-up data suggested that hysterical features were prognostic of a better social adjustment than the other two categories. In a similar study, Goetz et al. (1977) used the same three personality categories to classify 30 anorexic patients. They also found that patients with hysterical features showed the best prognosis.

Using a slightly different set of personality categories, Dally (1969) conducted an extensive study of different personality types associated with AN. One hundred and forty anorexic patients were divided into obsessional (group O), hysterical (group H) and mixed etiology (group M)



categories. At follow-up, group H had the lowest rate of readmission, suggesting a better prognosis for this group.

While these three early studies provided preliminary information on the relationship of personality types and eating disorders, they are limited by methodological weakness. The lack of interrater agreement data on assignment to personality categories and to outcome categories leaves the reader unsure of the reliability of these variables. Furthermore, the lack of specific criteria and the high degree of inference necessary to assign subjects to the personality categories make reliable classification problematic. Also, the fact that these studies were conducted before the introduction of DSM-III makes it impossible to generalize to the current DSM personality disorders.

Studies which have examined the prevalence of DSM-III personality disorder in mixed samples of anorexic and bulimic subjects have obtained overall estimates of 27% (Herzog et al., 1992) to 93% (Kennedy, McVey, & Katz, 1990) for the presence of any personality disorder. Figures for bulimia nervosa have been distributed across ranges of 21% - 40% (Ames-Frankel et al, 1992), 41% - 60% (Rossiter et al., 1993; Steiger et al., 1991; Yates et al., 1989; Zanarini et al., 1990) and 61% - 85% (Gartner et al., 1989; Levin & Hyler, 1986; Norman, Blais, & Herzog, 1993; Schmidt & Telch, 1990; Steiger et al., 1992; Wonderlich et al., 1990).

Variability is also marked when cluster assignments and specific diagnoses are tabulated; for example, Borderline Personality Disorder (BPD) has been identified in from 2% to 47% of bulimics, 0% to 21% of restricting anorexics, and 12% to 55% of bulimic anorexics (e.g., Fahy et al., 1993; Gartner et al., 1989; 1993; McClelland et al., 1991; Wonderlich and Swift, 1990).

Summed over the 13 studies reporting on all DSM-III or DSM-III-R Axis II diagnosis (Ames-Frankel et al., 1992; Gartner et al., 1989; Gvirtzman et al., 1983; Herzog et al., 1992; Kennedy et al., 1990; Levin & Hyler, 1986; Piran et al. 1988; Powers et al., 1988; Schmidt & Telch, 1990; Wonderlich et al., 1990; Yager et al. 1989; Yates et al., 1989; and Zanarini et al., 1990), the rate of one or more personality disorders in eating disordered subjects is 59%. Among inpatients, the overall rate is 74%; among outpatients or volunteers, the rate is 54%.

Additionally, it has been suggested that BN and AN are differently characterized by specific personality disorders and distinct personality clusters: bulimic behaviour with the dramatic-eratic DSM-III-R personality clusters, particularly histrionic personality disorders, and AN with the anxious-fearful personality cluster, particularly obsessive-compulsive and avoidant personality disorders (Wonderlich et al., 1990; Piran et al., 1988). Other studies, however, do not support these associations (Gartner et al., 1989; Pope et al., 1987).

Using the Personality Assessment Schedule (Tyrer & Alexander, 1979) in an eating disorder sample, McClelland, Mynors-Wallis, Fahy, and Treasure (1991) found personality disorders in 26 patients (52%). These patients had a significantly longer duration of illness than those without a personality disorder. In another study, Fahy, Eisler and Russell (1993) assessed personality disorders in 39 BN patients evaluated with the PAS. Sixteen (41%) were given a personality disorder diagnosis and 10 (26%) had more than one. The results showed that patients with both disorders did worse in treatment than those without personality disorder (Fahy, Eisler, & Russell, 1991; Johnson, Tobin, and Dennis, 1990).

It is clear that a good start has been made in sorting out the relationship between personality



disorders and the eating disorders. Nevertheless, much work needs to be done in three particular realms. First, the directions of association between these two phenomena should be further investigated. Second, emphasis should be placed on determining clinically relevant subtypes of AN and BN based upon distinctive personality constellations. Third, the impact of personality disorder on treatment of eating disorders should be further examined.

Methodological problems including small sample size, selected populations (i.e., inpatients), use of either self-report measures or unstructured clinical interviews, and examination of only one personality disorder limit comparisons across studies and generalizability of the findings.

Specific family characteristics have also been shown to be linked to the diagnosis of some PDs in eating disorder patients. Wonderlich and Swift (1990), for example, found that the borderline personality disorder subgroup of anorexics and bulimics perceive their parents as abnormally hostile. It will be of particular interest to determine whether family EE is associated with PDs in patients with eating disorders.

Personality pathology has been found to be significantly related to poor outcome. Tyrer and Seivewright (1988) and Reich and Green (1991) reviewed treatment response in patients studied with the Personality Assessment Schedule (PAS) and instruments other than the PAS, respectively. They found poor response to treatment in patients with a secondary diagnosis of personality pathology, including those who were diagnosed as depressed, anxious, alcoholic, and social phobic. Like other diagnostic groups, eating disorders may have a poor prognosis when accompanied by personality disorders. Using the PAS, Fahy et al (1993) have shown that personality disorders are associated with a poorer response to cognitive-behavioural treatment for bulimia. In the more specific area of borderline personality disorder, Johnson et al. (1990)

showed that bulimics with this concurrent diagnosis were less likely to benefit from psychodynamic treatment. A negative prognostic effect has not been firmly established, however (Ames-Frankel et al., 1992; Fahy et al., 1993; Fallon et al., 1991).

The present study builds on the foundation of previous studies by reporting on the nature and prevalence of personality disorders in an outpatient sample of women seeking treatment for AN or BN. Based on these studies, several predictions are made. First, AN patients will be diagnosed with the inhibited PAS cluster more often than BN patients, who are predicted to be more likely to meet criteria for the dependent PAS cluster. Second, a histrionic PD will be more common in bulimic than in anorexic patients, who will be characterized more by anxious PD. Finally, families of eating disorder patients who have been diagnosed as PD will be more critical to the patients than families of eating disorder patients without PD. The study further examines the impact of PD on treatment of eating disorders. Several questions seem relevant: does personality pathology predict unfavourable outcome of treatment in eating disorder patients? If so, are certain PDs signs of poorer prognosis? And for what kind of therapy PD could be the best predictor of treatment outcome?

## **2.4. PATIENT'S RESPONSE STYLE TO THERAPIST AND THERAPY**

Self-disclosure has been the focus of considerable theoretical and empirical inquiry in recent years. Much of the research interest stems from the role of self-disclosure in theories of psychological adjustment, the psychotherapeutic process, and interactional behavior. In this section a brief review of the conceptual background of self-disclosure and the basic parameters of the disclosing process will be presented. The current body of theoretical and empirical knowledge that bears on the relationship of self-disclosure with personality characteristics and psychotherapy will also be

reviewed.

Psychotherapists of all persuasions, on the other hand, agree that nonverbal aspects of disclosure, voice quality, facial expressions, and body posture are important factors of psychotherapeutic communication, both for the therapist and the patient (e.g., Altman & Taylor, 1973; Argyle & Dean, 1965; Montgomery, 1981, 1984; Patterson, 1990; Shapiro, Krauss, & Truax, 1969). As nonverbal aspects of self-disclosure, emotional engagement which refers to the quality of a patient's nonverbal participation and engagement in therapeutic interaction will be briefly addressed.

#### **2.4.1. SELF-DISCLOSURE: CONCEPTUAL BACKGROUND**

Chelune (1979) in his review of self-disclosure research has noted that the concept of self-disclosure is derived primarily from existential and phenomenological theory. To disclose means to make known or to show. Self-disclosure, therefore, is the process by which we make ourselves known to other persons by verbally disclosing personal information.

Self-disclosure has often been postulated to have important consequences for psychological adjustment and interpersonal functioning. Fromm (1955) has referred to self-disclosure as a means for decreasing both phenomenological distance and alienation from self as well as others. The concept of self-disclosing behavior plays a central role in Rogers's theory of personality change (1961). For Rogers, self-disclosure is a characteristic of the acceptance of self and the means to achieve this end. Mowrer (1961) formulated a theory of psychopathology in which guilt from not disclosing one's perceived transgressions is the underlying cause of emotional disturbance. Only by the disclosure of one's "sins" to another person can one attain emotional

health. Jourard (1964) has been a leading advocate of the positive aspects of self-disclosure. For Jourard, self-disclosure is both a symptom of personality health and a means to achieve "real-self being" and interpersonal effectiveness.

The role of self-disclosure in interpersonal relationships has been examined extensively within the context of social exchange and social penetration theories and has been reviewed by Cozby (1973). According to social penetration theory, the development of an interpersonal relationship is a joint outcome of situational determinants, interpersonal reward/cost factors, and personality characteristics (Altman & Taylor, 1973; Taylor, 1968; Taylor, Altman, & Sorrentino, 1969). Relationships are thought to proceed from nonintimate to intimate areas of exchange via verbal disclosure, activities jointly engaged in, and nonverbal communication (Cozby, 1973). Within relationships the amount of information disclosed to a given individual has been found to be highly correlated with the amount of information received from that person (Jourard, 1959; Jourard and Richman, 1963; Levinger & Senn, 1967). This reciprocity or "dyadic effect" has been interpreted as a result of a social exchange process in which self-disclosure functions as a social reward (Certner, 1973; Worthy, Gary, and Kahn, 1969).

The concept of self-disclosure has also received considerable attention with respect to the psychotherapeutic process. "Most clinical and theoretical description of the psychotherapeutic process have focused upon...self-disclosure...as one of the central happenings" (Truax & Carkhuff, 1965, p.3). Both Ellis (1959) and Sullivan (1954) have commented on the therapeutic value of client disclosure and the nontherapeutic value of abstract interactions devoid of personal disclosure. Truax (1961) and Truax and Carkhuff (1964,1965) have noted that both the client's and the therapist's disclosures play important roles in the successful outcome of therapy. Yalom

(1970, p. 271) has suggested that "self-disclosure is prerequisite for the formation of meaningful interpersonal relationships in a dyadic or in a group situation."

#### **2.4.2. DEFINITION AND PARAMETERS OF SELF-DISCLOSURE**

As defined by Sidney Jourard (1964), self-disclosure refers to the process of telling another person about oneself, honestly sharing thoughts and feelings that may be very personal and private. Jourard was one of the first psychologists to develop the area of self-disclosure for extended research and discussion, and his name is still strongly associated with the area. He believed that the physical and psychological health of individuals and the success of relationships requires adequate self-disclosure to strip away restrictive social masks.

Derlega and Grzelak (1979) define self-disclosure as including "any information exchange that refers to the self, including personal states, dispositions, events in the past, and plans for the future" (p. 152). Self-disclosure is, thus, the communication of information about one's affects, behaviours, and cognitions with the implication that the material disclosed is either secret, intimate, or emotionally charged.

Definitions of self-disclosure usually emphasize either information conveyed to another person or the process of making oneself known to others. We might consider, however, the degree to which measures of self-disclosure tap a personality variable. Cozby (1973) acknowledged that self-disclosure refers to "both a personality construct and a process which occurs during interaction" (p. 73). Mahon (1982) defined self-disclosure as "a personal variable people bring to encounters with others" (p. 334). It seems reasonable to assume that people vary in the degree to which they are open and accessible to others and that measures of self-disclosure reflect, to

some degree at least, this individuals difference.

Viewing self-disclosure as an individual difference variable in no way negates the importance of situational factors. Situational factors certainly influence the expression of other variables (e.g., extroversion) that are commonly considered traitlike individual difference constructs.

The concept of self-disclosure is quite complex, since it encompasses both the qualifying of verbalizations and the assessment of the content and direction of verbalizations. Cozby (1973) has proposed that the dimensions of self-disclosure are 1) the breadth or amount of information disclosed, 2) depth or intimacy of this information, and 3) the duration of time spent in disclosure. Two different parameters suggested by chelune (1975a) are 4) the affective manner of presentation of the disclosed material and 5) the flexibility of the disclosure pattern. In addition, other authors have focused on the positive-negative self-evaluative aspect of the content of disclosures (e.g., Sarason & Ganzer, 1972).

### **2.4.3. SELF-DISCLOSURE AND PERSONALITY**

Several authors (Jourard, 1964; Mowerer, 1961; Rogers, 1961) viewed self-disclosure as both a sign and a cause of a healthy personality. Disclosure was viewed as a relatively stable personality characteristic that was related to other positive personality characteristics. Since the early 1970s, personality and social psychologists have produced a plethora of empirical research on self-disclosure (Altman & Taylor, 1973; Chelune, 1979; Derlega & Chaikin, 1977; Jourard, 1971; Strassberg et al., 1977). According to Archer (1983), however, most of the studies have focused on the amount of information disclosed and the content of that information to the exclusion of personal and situational factors involved in the self-disclosure process. In his proposed

attributional model of personal and situational factors involved, Archer includes perceiver variables such as dispositions and expectations as one set of factors. Similarly, Derlega and Grzelak (1979) propose that individual differences in a "need for intimate disclosure" (p. 175) may play a significant role in the regulation of privacy and disclosure, and thus "the value of self-disclosure may depend on a person's system of personal preferences" (p. 175).

Correlations between personality measures and self-disclosure scores have generally been low and often equivocal. Altman and Taylor (1973) suggested that the search for relationships between trait disclosure and other personality measures is unrealistic. They proposed that self-disclosure be examined within the context of specific relationships and settings. Several consistencies in the literature are worthy of note, however. From the studies summarized by Cozby (1973) there appears to be a positive relationship between self-disclosure and measures of extroversion and sociability. Taylor and Oberlander (1969) have also found significant positive relationships between self-disclosure and perceptual sensitization and field dependence. A differential relationship between repression-sensitization and self-disclosure for males and females has been reported by Chelune (1975b) and partially replicated in a later study (Chelune, 1975c). Other investigators have demonstrated that sensitizers elaborate more and spend more time verbalizing their emotional experiences than do repressors (Axtell and Cole, 1971; Davis and Sloan, 1974; Kaplan, 1967).

In an extensive review of the disclosure literature, Cozby (1973) hypothesized that "mentally healthy" persons are characterized by high disclosure to those close to them and moderate disclosure to others in their social environment. Direct links between disclosure and personality have not been effectively substantiated and who is a high- or low-disclosing individual has

remained unclear.

#### **2.4.4. SELF-DISCLOSURE AND OUTCOME OF THERAPY**

Different models of psychotherapy agree with the notion that patient self-disclosure makes an important contribution to the progress of treatment. Self-disclosure, especially early in the treatment process, enables the therapist to understand the patient's problems and the social context in which they occur. Nonverbal sources of information, such as behavioural observation, physiological assessment, and the congruence or incongruence among channels of communication, should further enrich the therapist's understanding of the patient's experience. Since many therapies presumably adapt to the needs of the patient, this information base directly affects the formulation of treatment plans, roles, and goals. Patient self-disclosure, thus, is viewed as a major process variable in psychotherapy.

Generally, the research literature describes a positive relationship between self-disclosure and the outcome of therapy. Studies reporting this finding have focused on the content of patient disclosure (Blau, 1950, 1953; Braaten, 1961; Kirtner & Cartwright, 1958; Seeman, 1949; Steele, 1948; Truax & Wittmer, 1971; Wolfson, 1949), the depth of patient disclosure (Gendlin et al., 1968; Kiesler, 1971; Kiesler, Mathieu, & Kiesler, 1969; Mitchell et al., 1973; Schauble & Pierce, 1974; Stoler, 1963; Tomlinson & Hart, 1962; Tomlinson & Stoler, 1967; Truax & Wargo, 1969; Van der Veen, 1967; Walker, Rablen, & Rogers, 1960), and increasing disclosure over time (Gendlin et al., 1968; Kiesler, Mathieu, & Klein, 1967; Tomlinson, 1967; Tomlinson & Hart, 1962). However, other studies have failed to confirm these relationships (Auld & Wilkinson, 1973; Craig, 1973; Kiesler, 1971; Prager, 1971; Staples et al., 1976; Tomlinson, 1967; Truax, Wargo, & Volksdorf, 1970; Van der Veen, 1967).



In summary, self-disclosure is seen as a process in which patients learn to understand, develop, and express themselves and their potential. Models that do not regard self-disclosure as a curative factor in therapy nevertheless implicate it in the successful outcome of treatment.

#### **2.4.5. EMOTIONAL ENGAGEMENT**

Research in the area of communications generally indicates that, when presented with contradictory verbal and nonverbal messages, individuals generally give greater credibility to the nonverbal aspects of the interaction (Stuart, 1980). In fact, nonverbal signals may convey upwards of 75% of the information people receive from others (Mehrabian, 1972). Emotional engagement (EEn) refers to the quality of a patient's nonverbal participation and engagement in therapeutic interaction. Some people respond more to nonverbal cues than others. Although nonverbal cues are complex, they are all significant pieces of information that should be integrated into the course of therapy.

The inclusion of nonverbal behaviour considerably expands the concept of self-disclosure and the range of interpersonal activities to be observed. In presenting a scale of nonverbal self-disclosure of feelings and emotions, Shapiro, Krauss, and Truax (1969) argue that this type of disclosure is as important as verbal disclosure. One person may disclose to another through a smile, a touch, withdrawing, or physical assault. Indeed, nonverbal disclosure alone may express a person's feelings or needs sufficiently or it may confirm or contradict verbal self-disclosure.

#### **2.4.6. RESPONSE STYLE TO THERAPIST AND THERAPY IN EATING DISORDER PATIENTS**

It is important to consider how self-disclosure and emotional engagement may operate together

in patient's response style to therapist and therapy. It is hypothesized that both verbal and nonverbal aspects of a patient's response style to therapist and therapy are positively related to outcome of therapy. To measure both self-disclosure and emotional engagement in this project, a new instrument, the Patient Response Style Scale (PRSS), is developed (see section B of chapter 3). Using the PRSS, this study will address four issues:

- a) the PRSS ratings in a sample of eating disorder patients
- b) the prognostic value of the PRSS
- c) the implications of these measures for therapists
- d) the reliability of the PRSS

## **2.5. SELF- AND OTHER-BLAME**

Theoretical discussion of self-blame suggests that it can be rational or irrational and functional or maladaptive, depending on the circumstances. For example, sometimes self-blame may seem "rational" if it is based on assumption of responsibility for transgressions. Alternatively, the psychological significance of self-blame may lie less in its rational relationship to responsibility for a bad outcome and more in a felt capacity to control that outcome. Thus, Janoff-Bulman (1979) found that "behavioural" self-blame (blame about things one did or failed to do that are under one's control) was not correlated with depression whereas "characterological" self-blame (blame about one's basic nature, presumably less controllable or changeable) was. Using related reasoning, Weiss (1975) proposed that children's self-blame in the context of parental separation is an alternative to feeling powerless in the family situation. For the child, guilt may be empowering, in that the divorce is then presumed to be the result of something the child did or failed to do. The child can, then, gain a sense of control and avoid a sense of helplessness by feeling self-blame.

Triadic relations in families are those in which a child feels caught or torn between two parents. The child may be asked by both parents - directly or indirectly - for her or his exclusive loyalty. Family systems theorists see this configuration as a common one in families in distress (see Haley, 1959; Minuchin, 1974, 1984; Napier, 1978).

### **2.5.1. CHARACTEROLOGICAL AND BEHAVIOURAL SELF-BLAME**

Recognizing that self-blame may be both adaptive and maladaptive is a first step towards the conclusion that there are two different types of self-blame. Janoff-Bulman (1979) distinguished between two types of self-blame: *self-behavioural blame*, which involves an attribution to a controllable or modifiable aspect of the self, and *self-character blame*, which involves an attribution to uncontrollable aspects of the self, such as one's traits or dispositions.

Further research (e.g., Janoff-Bulman, 1982; Janoff-Bulman, Timko, & Carli, 1985) has demonstrated that behavioural self-blame attributions are generally made in the past tense such as "she should not have let him kiss her" (Janoff-Bulman, et al., 1985, p. 166), whereas characterological self-blame attributions are generally made in the present tense such as "she is the type of person who gives in easily to men; they are able to persuade her to do things she really doesn't want to do" (Janoff-Bulman et al., 1985, p. 167), reflecting implicit differences in the extent to which the attributional factors continue to define the person.

For instance, it is characterological self-blame, and not behavioural self-blame, that is associated with depression (e.g., Janoff-Bulman, 1979; Major, Muller, & Hildebrandt, 1985; Peterson, Schwartz, & Seligman, 1981). Janoff-Bulman (1979) hypothesized that characterological blame resolves the "depressive paradox" (Abramson & Sackeim, 1977; Peterson, 1979), that is, the

tendency of depressives to feel guilty over events about which they also feel helpless: bad character is blamable but uncontrollable. The “paradox in depression” is readily resolved when one recognizes that depressed individuals blame themselves characterologically, and not behaviourally; that is, they do not blame themselves for outcomes that they regard as controllable. Rather, as has been pointed out elsewhere, “In the case of personal failure, the characterological blames will point to deficits in themselves that are believed to account for these failures. The deficits are likely to lie in the realm of characteristics that generally define them, characteristics that are relatively nonmodifiable, stable, and global” (Janoff-Bulman, 1979, p. 1801).

Work specifically on self-blame, however, has demonstrated that the nature of the self-attribution is important: Behavioural self-blame roughly coincides with an internal attribution that is unstable, controllable, and specific; characterological self-blame, on the other hand, coincides with an internal attribution that is stable, uncontrollable, and global. An important distinction to be drawn between behavioural and characterological self-blame is the different implication of each attribution for perceived vulnerability.

Characterological self-blame is related to self-esteem and is associated with a belief that one is personally deserving of past negative outcomes, whereas behavioural self-blame is related to control and is associated with a belief that one can, in the future, avoid a negative outcome (Janoff-Bulman, 1979; 1982). A person who can point to a particular past behaviour in order to account for a negative outcome is able to maintain a belief in the future avoidability of a similar misfortune. On the other hand, a person who attributes the negative outcome to a more or less stable character trait will not be afforded a similar belief in future invulnerability. It is behavioural self-blame in the absence of characterological self-blame that is adaptive.

In a theoretical paper on self-blame among victims of battering, Miller and Porter (1983) noted that victims of spouse abuse, unlike the victims of rape or other criminal behaviour, are faced with inferring “causal responsibility” from multiple observations repeated over time. This difference forms the basis for Miller and Porter’s perspective suggestion that various victims groups may differ both in the degree of self-blame they assume and in the meaning of that self-blame. For example, a victim of rape “is unlikely to blame herself for being a *cause* of the rape, only for being an *occasion* for the rape. The cause of the rape will most commonly be seen to reside in her assailant” (p. 143). By contrast, “a battered woman who says ‘it’s my fault’ may actually be exonerating her assailant for his actions, at least partially” (p. 144).

In addition to discussing the possible meaning of self-blame given repeated instances of victimization, Miller and Porter pointed out that the distinction between behavioural and characterological blame may vanish over time; “Specifically, there would appear to be little difference between blaming repeated acts of violence on one’s recurrent behaviour (e.g., I am always critical of him) and blaming them on one’s traits or character (e.g., I am a critical person)” (pp. 147-148). Lastly, Miller and Porter noted that the relation between attributions and adjustment may differ across different kinds of victimization, depending on the importance to the victim of finding a causal explanation. As conceptually detailed as Miller and Porter’s analysis was, they nevertheless made the error of describing the relation between attributions and adjustment as both “the link between causal explanation and emotional state” (p. 149) and “the relationship between blame assignment and adjustment” (p. 149).

### **3.5.2. SELF- AND OTHER-BLAME IN EATING DISORDER PATIENTS**

Self/other-blame as a response to victimization, illness, and physical disability has been receiving

increasing attention in the psychological literature. Many accident victims (Bulman & Wortman, 1977; Frey, Rogner, Schuler, & Korte, 1985; Heinemann, Bulka, & Smetak, 1988), people suffering from renal failure (Witenberg et al., 1983), cancer patients (Taylor, Litchman, & Wood, 1984; Timko & Janoof-Bulman, 1985; Weisman & Worden, 1976), rape victims (Janoof-Bulman, 1979; Meyer & Taylor, 1986), heart attack patients (Affleck, Tennen, Croog, & Levine, 1987a, 1987b; Croog & Levine, 1982), and patients with insulin-dependent diabetes (Tennen, Affleck, Allen, McGrade, & Ratzan, 1984) engage in some form of self/other-blame. A relatively high incidence of self-blame has also been reported in studies of parents of acutely ill or handicapped infants (Affleck, Allen, McGrade, & McQueeney, 1982; Affleck, McGrade, Allen, & McQueeney, 1985; Tennen, Affleck, & Gershman, 1986) and children with insulin-dependent diabetes (Affleck, Allen, Tennen, McGrade, & Ratzan, 1985).

Bulman and Wortman (1977) found that accident victims with spinal cord injury who blamed themselves coped better than those who blamed others or who felt that the accident could have been avoided. The authors speculated that self-blame may be adaptive, however, only in cases where it may facilitate action to remedy the situation.

The link between blaming one's own behaviour and adaptation, however, has been inconsistent across a series of studies that focus on the role of self-blame in adapting to threatening events. Behavioural self-blame has been associated with positive adaptational outcomes among accident victims (Bulman & Wortman, 1977), rape victims (Janoof-Bulman, 1979), cancer patients (Timko & Janoff-bulman, 1985), mothers of acutely ill infants (Affleck, McGrade, Allen, & McQueeney, 1985), and children with diabetes (Tennen, Affleck, Allen, McGrade, & Ratzan, 1984). However, among end-stage renal failure patients (Witenberg et al., 1983), another group of cancer patients

(Taylor, Litchman, & Wood, 1984), and mothers of diabetic children (Affleck, Allen, Tennen, McGrade, & Ratzan, 1985), behavioural self-blame showed no association with adaptation. In one group of rape victims (Meyer & Taylor, 1986), one group of children of divorcing patients (Healy, Stewart & Copeland, 1993) and in two other groups of accident victims (Frey, Rogner, Schuler, & Korte, 1985; Nielson & McDonald, 1988), it was associated with poorer adaptation.

In an extensive literature review, Tennen & Affleck (1990), found a reliable association between blaming another person and poor adjustment. Nineteen out of 24 studies in which participants did perceive another person as a cause, demonstrated an association between blaming others and less positive adaptation. In none of these investigations was other-blame associated with more positive adaptation. Among the five studies in which there was no association between blaming others and adaptation, three (Affleck, Pfeiffer, et al., 1987; Witenberg et al, 1983) found no association between any attribution and adaptation, and one other (Meyer & Taylor, 1986) found only 1 of 12 statistically significant correlations between any attribution and adaptation. In another study, Witenberg et al. (1983) reported no correlation between other-blame and adjustment. Rather, they reported that after entering situational factors and other appraisals into a regression statement, blaming others did not make a unique contribution to adjustment. However, as Mendola et al. (1990) demonstrated, a significant association between other-blame and poor adjustment may be attenuated when factors such as event severity and other event appraisals are controlled statistically. Finally, Affleck, Pfeiffer, et al. (1987) found no association between other-blame and adjustment to rheumatoid arthritis, but only 7 patients (7%) blamed someone else. This low endorsement rate made it difficult for a significant relation to emerge between blaming others and adjustment.

Clinical observations indicate that in eating disordered families there tends to be a high level of feelings of guilt and self blame (Dare et al., 1994; Dare, Eisler, Russell, and Szmulker, 1990; Le Grange et al., 1992b; Szmulker et al., 1985). Mothers appear more likely to express these attitudes than fathers (Szmulker et al., 1985). Bulimic patients also experience greater blame in their families than do anorexic patients (Humphrey, 1988). Very little is known about the nature and prevalence of self- and other-blame among families with an eating disorder patients. More importantly, we do not know anything about the prognostic value of SB and OB. It is hypothesized that SB and OB are positively associated with EOI and CC, respectively. It is also hypothesized that mothers of eating disorder patients would be more likely to engage in SB attitudes than do fathers.

This study designed to extend the results of the research reviewed earlier and to expand their generality to a different group of psychiatric patients. As part of the present project, a new instrument, the Self- and Other-Blame Scale (SOBS), is developed to measure feelings of self- and other-blame between family members (see section C of chapter 3). Using the SOBS, this study will address five issues:

- a) the prevalence of self- and other-blame among families with an eating disorder patient
- b) the link between self- and other-blame and therapeutic outcome
- c) the association between self- and other-blame and psychological well-being
- d) the implications of these measures for therapists
- e) the reliability of the SOBS



## **CHAPTER 3**

### **3.A. DESIGN AND METHODOLOGY OF THE STUDY**

This chapter consists of three sections. Section A describes the design of the study and methodological issues forwarded by presenting the research purposes and hypotheses. The development of the two new observational measures, the PRSS and the SOBS, will be described in sections B and C, respectively.

#### **3.A.1. PURPOSE OF THE RESEARCH**

The purpose of this research is to investigate the relationship between personality-family factors and eating disorders, and their prognostic value in relation to engagement-outcome in different types of psychological treatments.

The importance of the influence of family factors in eating disorders, as measured by EE variables, is recognized. However, very few clinical data exist which clarify the nature of the relationship between family EE and eating disorders, as well as the prognostic value of the EE variables. This study investigates to what extent treatment outcome can be predicted from family EE.

Clinical observations have reported a high level of feelings of guilt and self-blame in families containing an eating disorder patient. However, very little is known about the nature and prevalence of self- and other-blaming attributes among the families, and to the best of my knowledge, no report has been published on the prognostic value of these factors, nor any clinical instrument to measure these feelings. To achieve these purposes, a reliable observational instrument, the Self- and Other-Blame Scale (SOBS), has been developed. This instrument allows

us to measure self and other-blaming attitudes among family members objectively, and examine the prognostic value of these factors.

Although Self-Disclosure (SD) has been emphasized as one of the central issues of the psychotherapeutic process, no research has been done to investigate this issue in a sample of eating disorder patients. One objective of the present research is to develop a reliable observational instrument to examine the patient's style of involvement in therapeutic interaction. The Patient Response Style Scale (PRSS) has been developed specifically to measure SD patterns as a patient-related factor in a sample of eating disorder patients. Moreover, the PRSS explores the prognostic value of the patient's self-disclosing style.

This project further investigates the impact of personality disorder on treatment outcome in a sample of eating disorder patients, as measured by the PAS. The study will use a combination of self-report and observational measures.

### **3.A.2. PRESENTATION OF THE RESEARCH HYPOTHESES**

The present study was aimed at evaluating the following hypotheses:

- Changes in family expressed emotion will be related to symptomatic changes in the patient during the course of therapy.
- Personality disorders of anorexic patients will be different from those of bulimic patients.
- Families of eating disorder patients who have personality disorder are more critical to the patients than families of eating disorder patients without personality disorder.
- The measures of self-disclosure and emotional engagement are positively related to therapeutic outcome.

- Mothers of eating disorder patients will be more likely to engage in self-blaming attitudes than fathers.
- Self-blame will be positively associated with emotional over-involvement, and other-blame will be positively associated with critical comments and hostility and negatively associated with warmth.
- Mothers' self-blame is negatively associated with therapeutic outcome

### **3.A.3. METHOD**

#### **3.A.3.1. SAMPLE**

Seventy-eight consecutive female referrals to the Maudsley Hospital Eating Disorder Clinic, referred for eating disorders who met DSM-III-R (APA, 1987) and ICD-10 (WHO, 1992) criteria for AN and BN, and their families were included in this study. The patients had to be 18 years or older. Table 3.A.1. demonstrates the patients characteristics. The mean age for the sample was 26.1 years (s.d.= 6.58; Range= 18-45). Subjects were divided into 59 (75.6%) anorexics and 19 (24.4%) bulimics. The average body weight, expressed as a percentage of the mean population weight matched for age and height (Diem, K. & Lentner, C., 1970), for the entire sample was 71.82% (s.d.= 7.87). Patients were excluded from the study if their weight fell to a dangerous level (usually 35 Kg in females of average height) or their mental or physical state at assessment was considered so dangerous as to require urgent admission to hospital. Fifty-eight patients (74%) had received previous treatment for the eating disorder elsewhere. Fifty-seven (73%) subjects were single, 15 (19.2%) were married at the time of the study, and 6 (7.7%) were separated or divorced. Family data that could be used for this study was available for 58 families. The eventual sample of 88 relatives consisted of 45 mothers, 31 fathers (15 patients came to the assessment with mother alone and one patient came accompanied only by her father), and 12 husbands. Permission to participate in the study and informed consent was obtained before their initial clinical interview (see Appendix A).

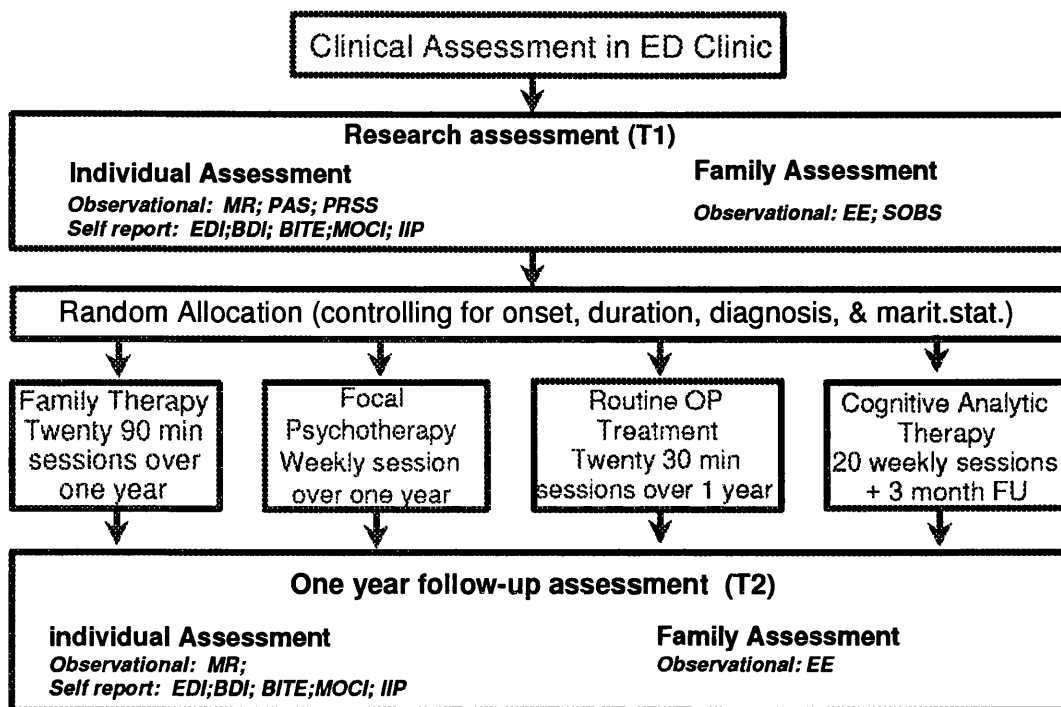
**Table 3.A.1. Characteristics of 78 Female Eating Disorder Patients**

<b>Diagnosis N(%)</b>	<b>AN:</b>	<b>59 (75.6)</b>
	<b>BN:</b>	<b>19 (24.4)</b>
<b>Age at presentation in Years M(s.d.)</b>		<b>26.1 (6.58)</b>
<b>Age at onset of illness (Years)</b>		<b>18.69 (5.24)</b>
<b>Duration of illness (Years)</b>		<b>6.48 (5.96)</b>
<b>Average Body Weight</b>		<b>71.82 (7.87)</b>
<b>Previous treatment N(%)</b>		<b>58 (74)</b>
<b>Marital status N(%)</b>	<b>Single:</b>	<b>57 (73)</b>
	<b>Married:</b>	<b>15 (19)</b>
	<b>Divorced:</b>	<b>6 (7.7)</b>

**3.A.3.2. THE CLINICAL ASSESSMENT PROCEDURE**

The design of the study is pictured in Figure 3.A.1. This study is part of a larger investigation in which three types of outpatient psychological treatment (family therapy, individual focal psychoanalytic psychotherapy and cognitive analytic therapy) for adults suffering from eating disorders are being compared with a fourth control treatment (supportive therapy) (see Dare & Eisler, 1995). An extensive pre-treatment evaluation was made of the patients and their families by independent researchers. Once the patient had satisfied DSM-III-R (APA, 1987) and ICD-10 (WHO, 1992) criteria for AN or BN, the patient and her family were assessed. The family interviews (using Standardized Clinical Family Interview - SCFI; Kinston and Loader, 1984) were video-recorded and later used for the rating of family EE following the standard rules (Leff and Vaughan, 1985; Vaughan and Leff, 1976). The family EE was again rated at the end of 12 months treatment using the same procedure. The interviews and ratings were conducted independently by two researchers, Dr. Ivan Eisler and the author, respectively. The author received his training in the rating of EE at the Medical Research Council (MRC) Social Psychiatry Unit at the Institute of Psychiatry in London.

**Figure 3.A.1. Research design**



**EE= Expressed Emotion**  
**SOBS= Self- and Other-Blame Scale**  
**MR= Morgan-Russell Assessment Schedule**  
**PAS= Personality Assessment Schedule**  
**PRSS= Patient Response Style Scale**  
**EDI= Eating Disorder Inventory**  
**BDI= Beck Depression Inventory**  
**BITE= Bulimic Inventory Test, Edinburgh**  
**MOCI= Maudsley Obsessional-Compulsive Inventory**  
**IIP= Inventory of Interpersonal Problem**

To assess self- and other-blaming attributions experienced by patients and their families, an observer-based instrument, the Self- and Other-Blame Scale (SOBS), was developed. Relatives' and patients' feelings and attitudes on self- and other-blame were elicited in a semi-structured manner during the SOBS segment of the SCFI interview for 51 eating disorder patients and 80 relatives. This part of the interview lasted about 10 minutes on average, while the family was directed to discuss and express their thoughts and feelings on self- and other-blaming (see section C of this chapter).

All patients were interviewed individually to obtain details of their psychiatric history and eating disorder. A standardised personality assessment interview, the Personality Assessment Schedule (PAS; Tyrer & Alexander, 1979; Tyrer, 1988) was also administered at the same time. It was stressed to subjects and informants that the assessment of personality referred to the period before the development of the eating disorder. The interviews were video-recorded and later used for the rating of personality characteristics by following the PAS rating criteria. The interviews and ratings were conducted independently by two researchers, Dr. Christopher Dare and the author, respectively. The author received his training in the rating of the PAS at St. Marys' Hospital Medical School at the University of London.

To assess both verbal and nonverbal communicative aspects of the patient's attitudes and behaviours, an observer-based rating instrument, the Patient Response Style Scale (PRSS), was also developed. All 76 subjects were interviewed using a clinical/research interview designed for patients suffering from eating disorders. The interviews were video-recorded and later used for the rating of PRSS by following the PRSS scoring instructions. This was conducted by the author and an independent rater for 30 randomly selected tapes, and by the author for the rest. For each

PRSS scale - Self-Disclosure (SD) and Emotional Engagement (EEn) - a score of 4 or 5 indicates high-SD/EEn, a score of 3 indicates moderate-SD/EEn, and a score of 0-2 indicates low-SD/EEn. Mixed-SD/EEn consists of those in which PRSS scales is rated on two different levels, one as high or low and the other as either low or moderate (see section D of this chapter).

The Morgan-Russell Outcome Assessment Schedule (Morgan & Hayward, 1988; Morgan & Russell, 1975) was used at the beginning and end of treatment to assess the clinical progress of the patients.

All patients were asked to complete a battery of psychometric instruments. A test battery assessed self-report symptoms of disordered eating, symptoms of bulimia or binge-eating, depression, obsessive-compulsive symptoms, and interpersonal difficulties. This battery included 5 widely used and well-standardized inventories of clinical symptomatology: The Eating Disorder Inventory (EDI; Garner, Olmstead, & Polivy, 1983); The Bulimic Inventory Test, Edinburgh (BITE; Henderson & Freeman, 1987); The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961 ); The Maudsley Obsessional-Compulsive Inventory (MOCI; Hodgson & Rachman, 1977); The Inventory of Interpersonal Problems (IIP; Horowitz, Rosenberg, Bare, Ureno, & Villasenor, 1988; Horowitz, Rosenberg, Ureno, Kalehzan, & O'Halloran, 1989). A detailed description of the self-report measures will be presented later in this chapter.

Following the family and personality interviews all patients were randomly allocated to either family therapy, individual focal psychoanalytic psychotherapy, cognitive analytic therapy, or a control treatment, supportive therapy for up to one year.

As soon as treatment had terminated, both patients and relatives were invited to a post-treatment

assessment meeting (T2) similar to the pre-treatment assessment (T1).

### **3.A.3.3. INSTRUMENTS**

#### **3.A.3.3.1. OBSERVATIONAL MEASURES**

Observational instruments are divided into individual and family measures. The former includes the Morgan-Russell scales, the Personality Assessment Schedule (PAS), and the Patient Response Style Scale (PRSS). The latter consists of the Standardized Clinical Family Interview (SCFI) and the Self- and Other-Blame Scale (SOBS). Detailed description of the two newly developed instruments, The PRSS and the SOBS, will be presented in sections B and C of this chapter, respectively. The following is a brief description of the other measures.

##### **a) The Morgan-Russell Assessment Schedule**

The Morgan-Russell Assessment Schedule (Morgan & Russell, 1975) (see Appendix B) is a structured interview designed to assess outcome as comprehensively as possible and is widely accepted in research into AN (Morgan & Hayward, 1988).

The Morgan-Russell scale includes five subscales which may be averaged and presented as an average outcome score: subscale A (food intake), subscale B (menstrual pattern in previous six months), subscale C (mental state as observed at interview and reported abnormalities at any time in previous six months), subscale D (psychosexual state), and subscale E (social and family relationships). Each subscale is rated on a 0-12 scale.

The assessments and the scores are based on the six month period preceding the interview. The schedule includes a global measure of outcome with a threefold categorization: Good Outcome; Intermediate Outcome; Poor Outcome. For the purpose of assessment of general outcome in this study, more specifically because of the chronic nature of the eating disorders in this sample, this



global scale was modified with the poor outcome category being divided into two groups. The new classification of general outcome was labelled as:

- 1) Recovered: Body weight is maintained within 15% of ABW and menstrual cycles are regular, or there are no bulimic symptoms (no episodic overeating nor self-induced vomiting).
- 2) Significantly Improved: Body weight has risen to within 15% of ABW, but amenorrhea persists, or bulimic symptoms are present but occur once a month or less.
- 3) Improved: Body weight has risen to within 25% of the ABW and amenorrhea persists, or bulimic symptoms occur up to once a week.
- 4) Poor: The patient is less than 15% below ABW or has developed bulimic symptoms, which occur more frequently than once weekly.

The Morgan-Russell scales for the study of AN outcome have documented reasonable to good psychometric properties (Morgan & Hayward, 1988).

#### **b) The Personality Assessment Schedule**

The Personality Assessment Schedule (PAS; Tyrer & Alexander, 1979) is a semistructured interview in which 24 personality variables are rated on a 9-point scale, with the scores determined by the extent of social maladjustment produced by each personality trait. Each of these can be rated by interview with the subject and interview with an informant. This interview is designed to formalize the assessment of personality disorder and may be used with any subject irrespective of psychiatric status. The way in which the schedule is used will depend on the current mental state of the patient and an assessment of this is a necessary precursor to the personality ratings.

The schedule takes 30-45 minutes to administer, and yields personality diagnoses that are roughly equivalent to DSM-III personality disorder clusters. It was developed by Tyrer (Tyrer &

Alexander, 1979; Tyrer et al., 1979, 1983) based on a cluster analysis of personality traits of personality-disordered patients. The specific dimensions yielded are: schizoid (roughly equivalent to DSM-III cluster I of schizoid, schizotypal, and paranoid), sociopathic (roughly equivalent to the DSM-III cluster II of borderline, histrionic, narcissistic, and antisocial), passive dependent (roughly equivalent to DSM-III dependent, avoidant, and passive-aggressive), and anankastic (equivalent to DSM-III compulsive).

The PAS has been shown to be fairly reliable in terms of inter-rater and temporal reliability (Tyrer et al., 1979, 1983). One of the major strengths of the PAS is the reliability of an overall personality disorder diagnosis. A blind 2.75-year test-retest of psychiatric patients yielded a weighted kappa of .64 for the presence or absence of a personality disorder (Tyrer et al., 1983).

### **c) The Standardised Clinical Family Interview**

The Standardised Clinical Family Interview(SCFI; Kinston & Loader, 1984)(see Appendix C) is designed to be used with a wide range of labelled and non-labelled families, in different stages of the family life cycle, and of varying composition.

The SCFI has a semistructured format, which allows a flexible use of standard questions, probes and statements about family life. The SCFI focuses on the family, while encouraging family members to interact spontaneously. It consists of a series of questions and statements about family life. The topics covered consist of the way the family sees itself, the degree of togetherness, who does what with whom, how families are alike and how they are different, views about stages of the family life cycle, the issue of the roles and responsibilities, areas of conflict and ways of handling these, issues of discipline and ways of making decisions.

### **3.A.3.3.2. SELF-REPORT MEASURES**

#### **a) The Eating Disorder Inventory**

The Eating Disorder Inventory (EDI; Garner, Olmstead, & Polivy, 1983) is a 64-item self-report questionnaire, which assesses the multidimensional nature of anorexia and bulimia nervosa. The EDI comprises eight subscales representing different psychological constructs essential to eating disorders. These subscales are: Drive for Thinness (DT, 7 items); Bulimia (B, 7 items); Body Dissatisfaction (BD, 9 items); Ineffectiveness (I, 10 items); Perfectionism (P, 6 items); Interpersonal Distrust (ID, 7 items); Interoceptive Awareness (IA, 10 items); and Maturity Fears (MF, 8 items)

Internal consistency of the EDI was good (Garner et al., 1982; Raciti & Norcross, 1987; Shore & Porter, 1990) and test-retest reliability in a sample of 70 undergraduate students over a period of 3 weeks was very high (Wear & Pratz, 1987).

#### **b) The Bulimic Inventory Test, Edinburgh**

The Bulimic Inventory Test, Edinburgh (BITE; Henderson & Freeman, 1987) (see Appendix D) is a 33-item self-report questionnaire, designed to identify subjects with symptoms of bulimia or binge-eating. It can be used to identify binge-eaters in a given population or as a screening instrument for use in a clinical setting. In addition, it serves as a useful measure of severity and response to treatment. The BITE consists of two subscales and an overall scale. Henderson and Freeman suggest “cut-off” scores for each scale, where a score at or above that level is “indicative of the presence of a severely disordered eating pattern.” The symptom subscale (30 items; overall range: 0-30; cut-off score = 20) asks the subject to rate the presence or absence of a set of attitudes and behaviours that are related to bulimia. The severity subscale (six items; overall range: 0-39; cut-off score = 5) measures the extent of those behaviours. Scores on the symptom scale

can be subdivided into three groups: high, medium and low scores. Those subjects achieving a high score have a high probability of meeting the DSM-III criteria for bulimia and Russell's (1979) criteria for bulimia nervosa. The two subscales can be added to provide a total score (overall range: 0-69; cut-off score = 25).

### **c) The Beck Depression Inventory**

The Beck Depression Inventory (BDI), first introduced in 1961 (Beck, Rush, Shaw, & Emery, 1979) and later revised (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), is a 21-item self-report depression scale. Scoring is completed by summing the severity of individual symptoms rated from 0 to 3 so that overall scores can range from 0 to 63. This scale is one of the most frequently used measures of the severity of depression symptoms, and many studies have demonstrated its reliability and validity (Beck, Steer, & Garbine, 1988).

### **D. The Inventory of Interpersonal Problems**

The Inventory of Interpersonal Problems (IIP; Horowitz, Rosenberg, Bare, Ureno, & Villasenor, 1988; Horowitz, Rosenberg, Ureno, Kalehzan, & O'Halloran, 1989) (see Appendix E) is a 127-item inventory designed to assess interpersonal difficulties in a broad cross-section of interpersonal domains. The IIP comprises 78 items phrased as: 'It is hard for me to', followed by, for example, 'trust other people', or 'say "no" to other people'. The remaining 49 items are phrased as: 'There are things I do too much', followed by, for example, 'I fight with other people too much', or 'I am too sensitive to criticism'. Subjects are asked to describe the amount of distress that they have experienced from each interpersonal problem on a 5-point scale ranging from not at all (0) to extremely (4). The IIP has demonstrated acceptable reliability, and sensitivity to changes that occur during psychotherapy (Horowitz et al., 1988; 1989).

#### **e) The Maudsley Obsessional-Compulsive Inventory**

The Maudsley Obsessional-Compulsive Inventory (MOCI; Hodgson & Rachman, 1977) (see Appendix F) is a self report measure that consists of 30 true/false items designed to assess the extent of various obsessive-compulsive symptoms. A total score as well as washing (11 items), checking (9 items), slowness (7 items) and doubting (7 items) subscale scores may be determined. The questionnaire effectively distinguishes obsessional from non-obsessional neurotics, (it has good test-retest reliability and the total score correlates significantly (0.6) with the Leyton Symptom Score. Control data were obtained from norms established for a non-student sample by Dent and Salkovskis (1986)).

This instrument has been shown to be both internally consistent and stable over time within a college student sample (Sternberger & Burns, 1990).

#### **3.A.3.4 . STATISTICAL ANALYSIS**

In order to investigate possible relationships between family EE variables and patient-related variables, as well as changes of EE levels and scales during the course of therapy, Pearson correlations, t-tests, and ANOVA as parametric statistics, and Spearman correlations, Chi-Square, Wilcoxon Matched-Pairs Signed-Ranks and McNemar Tests as nonparametric statistics will be used. The questions concerning the prediction of outcome will be addressed using forward logistic regression analysis. In this analysis the general outcome categories (GOC) are the dependent nominal measures and the family EE variables are independent continuous measures. In one set of analyses other possible prognostic variables, namely patient-related factors, will be included as independent variables in order to determine which variables are the most accurate predictors of outcome.

Similar analyses will be presented to assess what connections exist within the sample between

personality disorders, eating disorder characteristics and family EE variables. To this end, the sample is split into patients with personality disorder (PD) and those without personality disorder (NPD). A forward logistic regression analysis to evaluate the prognostic value of the PAS personality diagnoses for outcome of therapy in which the GOC is the dependent nominal measure and the PAS personality diagnoses are independent nominal measures, is performed. In order to determine which one of the personality and family factors would be the most accurate predictors of outcome, personality disorder and family EE (CC) will be introduced into the model. Again these analyses will be repeated to assess possible relationships within the sample between patient's response style to therapist and therapy, self- and other-blaming attitudes, family EE variables and treatment outcome.

### **3.B. THE PATIENT RESPONSE STYLE SCALE (PRSS)**

The fact that most forms of psychotherapy produce positive changes in a substantial proportion of patients has been established in recent years (see Luborsky, Singer, & Luborsky, 1975; and Smith, Glass, & Miller, 1980, for an extensive review). However, the nature of the curative factors is only vaguely understood and provides an ongoing challenge for psychotherapy researchers. Within the last 20 years, a number of variables, each at one time or another considered essential, have been shown to account for far less of the outcome variance than previously believed. These include pretherapy patient and therapist factors, as well as technique and type of treatment variables (Luborsky & McLellan, 1981; Luborsky et al., 1980; Luborsky et al., 1975; Sloane et al., 1975; Smith et al., 1980; Strupp & Hadley, 1979).

As communication of information about one's affects, behaviours, and cognitions, self-disclosure has been emphasized as one of the central issues of the psychotherapeutic process. It seems reasonable to assume that people vary in the degree to which they are open and accessible to others and that measures of SD reflect, to some degree at least, this individual difference. In order to determine why individuals differ in the extent to which they disclose to others, researchers have typically relied on a number of self-disclosure measures (e.g., Chelune, 1976; Jourard, 1964; Pederson & Breglio, 1968). These scales, however, have had serious problems with predictive validity. For example, in its various forms the classic Jourard Self-Disclosure Questionnaire (JSDQ) has been shown to be positively related (Jourard & Resnick, 1970; Pedersen & Breglio, 1968; Taylor, 1968), not at all related (Burhenne & Mirels, 1970; Ehrlich & Graeven, 1971; Vondracek, 1969a, 1969b), and even negatively related to self-disclosure (Doster & Strickland, 1971). The more recent Chelune Self-Disclosure Situations Survey (Chelune, 1976) fares little better. Though this measure has been shown to be positively related to self-disclosure for men in

an interview situation, it was actually negatively related for women in the same situation (Chelune, 1976). In addition, a new social psychological disclosure measure, the Self-Disclosure Index (Miller, Berg, & Archer, 1983) shows considerable promise. While these inventories have tended to be widely used, they remain subject to the fundamental problems common to many instruments based on self-report.

Similarly, nonverbal aspects of disclosure are important factors of psychotherapeutic communication, both for the therapist and the patient (e.g., Altman & Taylor, 1973; Montgomery, 1981, 1984; Patterson, 1990). An important barrier in investigating nonverbal aspects of disclosure has been the absence of standardized measures of individual accuracy in interpreting and conveying nonverbal cues in various modalities, or channels. Obviously, well-validated measures of decoding and encoding skills would make it much easier to study such issues as individual differences in nonverbal skills and in the use of different type of disclosure. Researchers interested in these kinds of questions have had to make up their own measuring instruments. Without such measures, it is hard to learn whether people with well-developed nonverbal skills differ from other people and, if so, in what ways. It would be important to know, for example, whether those who are better at sending or receiving nonverbal cues are more likely to benefit from psychotherapy. Although the methodological problems in studying these nonverbal levels and types of disclosure are difficult, they desperately need to be addressed for the field to develop comprehensive theories of psychotherapeutic change.

One objective of current research in this area is to develop a reliable observational instrument to examine empirically and objectively the patient's response style to therapist and therapy. The Patient Response Style Scale (PRSS) was developed specifically as part of the fourth Maudsley



study of psychological treatments for eating disorders in which three types of outpatient psychological treatment ( family therapy, individual focal psychoanalytic psychotherapy and cognitive analytic therapy) for adults suffering from eating disorders were compared with a fourth control treatment (supportive therapy; Dare & Eisler, 1995). The procedure was not to be specifically linked to any theoretical orientation.

In this study, thus, self-disclosure refers to the process by which the patients make themselves known to the therapist by verbally disclosing personal information. As nonverbal aspects of self-disclosure, emotional engagement refers to the quality of a patient's nonverbal participation and engagement in therapeutic interaction. By nonverbal disclosure, I mean positive changes in tonal aspects and enthusiasm shown in therapist and therapy by the patient when talking about his/her problem.

**3.B.1. A MEASURE OF PATIENT'S RESPONSE STYLE TO THERAPIST AND THERAPY: The Development of the Patient Response Style Scale (PRSS)**

The PRSS is an observer-based rating instrument designed to assess both verbal and nonverbal communicative aspects of the patient's attitudes and behaviours that are expected to facilitate or impede progress in psychotherapy. The PRSS describes the patient's style of involvement in the interaction and predicts the ability to participate in a therapeutic interaction. This instrument is designed to be applied to videotape recordings of a psychotherapy assessment interview. The interview consists of a series of questions, probes and statements about the patient's personal and family life, problem, and treatment. The main topics covered include issues such as the patient's family, social, intimate, and sexual relationships. The PRSS presently is organized in two subscales, Self-Disclosure (SD) and Emotional Engagement (EEn). Each subscale taps specific

aspects. "Simple Response", "Elaboration", "Spontaneity", and level of "Self-Personal Information" are considered as self-disclosure variables. Emotional engagement consists of two variables, "Tone of Voice" and "Interest in the Therapist and Therapy". All of these variables are meant to tap what is assumed to be an important general quality of a patient's engagement in psychotherapy.

### **3.B.2. The PRSS Scales**

#### **3.B.2.1. SELF-DISCLOSURE**

As a process which occurs during interaction with others, self-disclosure refers to the ability or willingness to reveal one's feelings and thoughts to another. Self-disclosure may be defined as any information about oneself including "Personal states, dispositions, events in the past, and plans for the future" (Derlega and Grzelak, 1979, p.152). The breadth or amount of information disclosed, and depth or intimacy of this information are proposed as two dimensions of self-disclosure (Cozby, 1973).

##### **3.B.2.1.1. Self-Disclosure Rating Criteria**

The following criteria were used for the rating of self-disclosure:

###### **a) Simple Response**

Simple response is a short and limited phrase which is usually of three types: 1) indicates agreement, acknowledgement, understanding, or approval of what the therapist has said; 2) indicates disagreement or disapproval with what the therapist has said; or 3) responds briefly to a therapist's question with specific information or facts. In examples 1 and 2 the patients present simple responses throughout the psychotherapy assessment interviews.

### ***Example 1***

T: from your point of view, do you have an idea about the real nature of your problem?

P: Umm, no.

T: You must have a theory about it.

P: I haven't got any theory.

T: Haven't you?

P: Not, really.

T: How does it effect in you at the moment?

P: Umm, what do you mean?

T: What's your main problem?

P: Weight.

T: Yes, yes. And how you look.

P: Yeah.

T: And what you eat.

P: Yeah.

T: So it's more how you look and how much you weigh.

P: Yeah.

T: Are you a sort of person who likes to talk through a problem in general?

P: Umm, yes, I think so.

T: How do you get on with your family?

P: Well I think.

T: Most of people have some balance with their parents. What's the sort of balance in your relationship with your parents?

P: It's OK.

T: Do you live on your own or are you living at home?

P: I'm living on my own.

T: How do you get on with your parents?

P: It's OK.

T: Would you say your family is a close family or a family where it is more important to be getting on with your own business?

P: Both, really. We respect each other.

T: How often do you see your parents?

T: Probably about three times a week.

T: Have you got close friends?

P: Yeah, I suppose so.

T: Perhaps would you discuss this problem with your closest friend?

P: No.

T: Have you got a boyfriend at the moment?

P: No, I haven't.

T: When you had boyfriend, did you have any sexual relationship?

P: No, I don't sex with boyfriend.  
T: Is that because you are not interested or?  
P: Because I'm not enough . . . (unclear).  
T: You do feel sexy, but it doesn't happen.  
P: I think so.  
T: Why?  
P: I don't know.  
T: I'm trying to find out about your sex drive. So I want to know whether you feel that you are sexual?  
P: Yeah.  
T: Do you enjoy sex generally?  
P: Yes.  
T: So you are not avoiding sex.  
P: No.

### ***Example 2***

T: So, what sort of problem do you see it is? Physical problem or psychological problem?  
P: I think it's not a real physical problem.  
T: Is that psychological?  
P: Umm, yes.  
T: So, in your mind, what sort of treatment do you see yourself as ...?  
P: I don't know really.  
T: I asked about friends, I didn't asked about girlfriends. Do you have a girlfriend?  
P: Not for a long time.  
T: Have you had a sexual relationship?  
P: No.  
T: Is that your religious belief?  
P: Yes, I believe in being virgin until marriage.

### **b) Elaboration**

Responses placed in this category are detailed accounts of events. They present a series of descriptive statements that serve to provide extended and often factual information about seemingly related incidents. The content is a purposeful elaboration of the patient's feelings and thoughts . The elaboration must be clearly related to the initial proposition and must contain inner references that reflect the patient's problems. Elaboration can be seen in the following brief

excerpts (examples 3-6) from the interviews.

### **Example 3**

T: Why did you stop (drinking alcohol)?

P: Umm, I suppose I didn't want being accused.

T: Who was accusing you?

P: My husband. I didn't like being blamed for things that I didn't do wrong. I wanted to be understood which I wasn't. I suppose I wanted to show myself that I could give up . . . . And there was a weekend we had a party . . . .

### **Example 4**

T: When did your problem with your eating start?

P: I've always been picky . I don't really know, to be honest. Dr T said I'm anorexic, but I thought anorexic was being sick all the time, but I'm not sick all the time. ... I'm vegetarian.

P: I don't know what I'm doing on this chair. I've been pushed by my husband, my parents, my work, and my friends. So I'm here, but I don't want to be here. (she is laughing and looks very relaxed at this point).

T: How old were you when your period started?

P: I was about 11 or 12. I remember that because we were going to France for a week. . . . . Teachers in school were saying "take something because you won't be upset or whatever." So I took something . . . .

### **Example 5**

T: What, from your point of view, is the nature of the problem. Because we are interested in the way how people's view about their problem is effected by treatment in a years time. So, from your point of view, what's the essence of the disorder?

P: Umm, the way I understand that it effects me, it's as a cycle. Sometimes I'm on the top of the cycle, sometimes on the side and on the bottom. Not necessarily related to depression, not, in any way, related to happiness. Possibly at the moment its related to stress. But I can't think more than that ...

T: So, how do you call it. Do you call it anorexia?

P: Oh, yes. Yes, I do. The way it effects me certain days, umm, it doesn't seem actually to be related to food. Food isn't the way that I tend to relate to certain things. Umm, that's just the way I show it.

T: Do you think there must be a reason why food is important for you?

P: Yes, I think so, possibly. I actually quite enjoy to do it. It's a social thing, you know. I like to eat without people. But one sort of behavioural thing as well, I think it is.

T: What does that mean?

P: Well, I think the way, for instance, if I'm having a difficult day, I will - help it or not help it, or consciously or subconsciously - put a sort of limit on what I eat on certain time of the day. It

doesn't necessarily mean, it doesn't seem to be to me that I'm trying to create myself as a certain shape. I've never consciously dieted. Never consciously worried about a particular shape or size or . . . .

#### **Example 6**

T: One of things that I'm trying to find out is how people themselves think they develop the problem. What's your understanding of your anorexia nervosa.

P: Well, when I, first, went in the hospital I didn't realize that, but now looking back, umm, there are the other periods in my life where I lost and gain weight. I was very chubby when I was in junior school. Umm, so around the age of 11, when I went to secondary school I lost a lot of weight before going to secondary school.

### **c) Self-Personal Information (Intimacy of Disclosure)**

As a basic parameter of self-disclosure, self-personal information (intimacy of information) refers to the explicit communication to others of some topics which are of such a nature that the individual is not likely to disclose it to everyone who asks for it. Intimate disclosure is a straightforward revelation of feelings and thoughts about an issue of personal importance to someone who is receptive. Based on a clinical/research interview the following topics have been rated as quite intimate and private:

**c.1. Family Personal Information-** more private information about the patient's family, the nature of family relationships, and feelings about family members (examples 7-9)

#### **Example 7**

T: Can you tell me a little bit about your family?

P: Well, my mum and my dad are divorced. My dad left when I was 7. So my mum had to bring up myself and my brother and it was hard time for her, really. She's married again when I was 11. He (stepfather) is lovely. He is really nice to do anything for anybody. I love him very much. First I didn't like him because he was in my dad's place, but he is lovely. . . .

T: What was your childhood like?

P: Umm, I really don't remember much before my dad left, to be honest. I can't remember myself as a baby. Umm, yeah, when my dad left, my mum blamed me and I blamed my mum, you know. I hated my mum. Then, on the other hand, my mum needed somebody to talk to and she talked

to me. So I grew up a lot, actually. I wasn't really a kid any longer. By doing that we began to be friends. We be there to support each other, really, all the time. You know, she started smoking, she does smoke now. She was just shocked about what happening. ... My dad was very immature, you know. My mum's older than my dad. (Describes two stories of her relationship with her father ...). He just didn't understand kids. I forgive him now. That was all part of me growing up and part of what I am today. ... I can't really umm ... she (her mum) had to go out to work. ... Child minder was a friend of mum's ... She (child minder) used to make me go down the shops every morning before school and buy bloody packs of stuff, and then when I came home from school that's go down the shops again bloody shopping. (Getting a bit sad and distressed and says to T: I apologise. T: No, that's all right. P: Sometimes, you can see, how I feel (T: Yeah, sure.)). it's not a really good part of my life. I told Dr. T so I don't care to tell you. You know, and then she (child minder) started ... (another negative story) ... and I was really so awful. I didn't know what to do. And I never told my mum because my mum had enough problems. And then she started to steal my stuff. .... She (child minder) never then used to feed me much either. .... I didn't tell my mum because she used to go work. . . .

T: Have you changed your feelings about that?

P: No, I still feel disgusting to think about it. I feel sick. You know, somebody in the position of trust and responsibility, you do that. I feel sorry for her now. Although I hate her "so much". I'm not a sort of person who can hate anybody. But, her, I hate her "very much", "very much", you know. She basically was a part of our lives, I just can't forget. I'm still nervous about it.

### **Example 8**

T: How do you describe your relationship with your husband?

P: Very, I mean good friends, basically. We were very good friends before we married. Umm, there is not a big issue with us as far as we talked about it several times. But I do actually gain ... that my problem is some sort of fear of pregnancy or whatever.

We explored that together. But having children is, in fact, very important to either of us.

T: How long have you been with each other?

P: Fourteen years. ... We married 1987. We worked very closely together. He was my boss, actually. I married a boss.

T: What was the relationship in your family like?

P: Good. Very busy. My father was always coming and going trying to run two jobs, and my mother was, a sort of, was always there for us. She wanted to be a full time mother.

T: So there were two domains. Father outside and mother inside.

P: Oh, yes. Pretty traditional for that time.

### **Example 9**

T: How did you get on with your parents and your brothers and sisters?

P: ... He (patient's father) wanted a boy and I was a girl, as I was a girl he was more for my sister than he was for me, and I felt I could relate, "I-could-relate" more to my mother than my father. My father many times frightened me.

**c.2. Sexual Matters-** private information about sexual experiences, the nature of these experiences, and feelings of sexual adequacy (examples 10-11)

***Example 10***

T: Have you (patient and her husband) had any sexual relationship?

P: Yes, we did, but we haven't made love since September. I feel such an awful person because I can't be a proper wife for my husband. But he is, to be honest, the most understanding person I've ever met, you know. He loves the way I am. He loves me to be OK. . . . I can't do it. I feel awful.

***Example 11***

T: How long has your relationship with your husband being stressed by anorexia?

P: Umm, sexual side has waned for the past few years. Perhaps sexual drive is effected by anorexia.

T: And that's distressed your husband?

P: Umm, he is extremely patient. Extremely patient all around, as far as sexual side is concerned, but also ...

T: Do you like sex?

P: OH, yeah. Yeah, ....

T: How often do you make love?

P: Not enough. Once every six weeks. And my husband is extremely patient (laughing).

T: Do you enjoy it?

P: Fairly, yes.

**d. Spontaneity**

Spontaneity refers to volunteered information of the type described under "Elaboration" and/or "Self-Personal Information". To be counted it must be associated with spontaneous comments that have some relevance to the patient's problem. The spontaneous self-disclosure tends to result in a higher rating. Some patients go well beyond what is strictly required to answer a question to disclose information about themselves. Conversely, the failure to disclose information where opportunities to do so exist would tend to lower the rating. The following excerpts (examples 12-



13) from the interviews illustrate this type of information.

### ***Example 12***

T: What was your childhood like?

P: Umm, I really don't remember much before my dad left, to be honest. I can't remember myself as a baby. Umm, yeah, when my dad left, my mum blamed me and I blamed my mum, you know. I hated my mum. Then, on the other hand, my mum needed somebody to talk to and she talked to me.

T: What helped you that you know that (patient's problem)?

P: My husband and my friends. We are very close friends. Long talk about using antidepressant and her medical history regarding eating problem . . . .

### ***Example 13***

T: So, this is what you know anorexia is like for some people?

P: Yes. For some people. Umm, and for me it is the way I can control myself.

T: How long has your relationship with your husband been stressed by anorexia?

P: Umm, sexual side has waned for the past few years. Perhaps sexual drive is effected by anorexia.

T: Do you think there must be a reason why food is important for you?

P: Yes, I think so, possibly. I actually quite enjoy to do it. It's a social thing, you know. I like to eat without people. But one sort of behavioural thing as well, I think it is.

T: What does that mean?

P: Well, I think the way, for instance, if I'm having a difficult day, I will - help it or not help it, or consciously or subconsciously - put a sort of limit on what I eat on certain time of the day. It doesn't necessarily mean, it doesn't seem to be to me that I'm trying to create myself as certain shape. I've never consciously dieted. Never consciously worried about a particular shape or size or . . . .

### **3.B.2.2. EMOTIONAL ENGAGEMENT**

The concept of "Emotional Engagement" refers to the quality of a patient's nonverbal participation and engagement in the therapeutic interaction.

### 3.B.2.2.1. Emotional Engagement Rating Criteria

Emotional engagement may be shown in the interview by "Positive Tone of Voice" and "Interest in the Therapist and Therapy".

#### a) Positive Tone of Voice

Positive changes in tonal aspects and enthusiasm shown in the therapist and therapy by the patient when talking about his/her problem, is perhaps the most important criterion on which the rating of emotional engagement is based.

#### b) Interest in the Therapist and therapy

Enthusiasm for and interest in the therapist and therapy are also relevant, provided that they are shown in relation to the therapist and therapy as such. Patients' warmth and enthusiasm can be seen in examples 15 and 16.

#### *Example 15*

T: I see. Do you think it is a psychological or physical problem?

P: I think it's probably psychological, but obviously, in fact, when I went to the GP two, three years ago asking for help, I went to get his help on the physical side, probably.

T: Yes, I understand. That seems very important because obviously we are offering psychological help (P: Yeah, right) and obviously I want to know how people's view about what sort of help they need influences what sort of help they best respond to.

P: Right, yeah. I know that, for instance, I do have what I would imagine or have read subsequently to realizing that I have been labelled as anorexic. I do recognize certain traits in myself, but I don't feel I'm doing a sort of, you know excessive amount of exercise to achieve a certain physical shape. ...

T: If you could imagine the ideal treatment, from your point of view?

P: Umm, I think probably some sort of psychological talking does help. ....

T: Are you a sort of person who likes to talk problems through?

P: Not naturally, but I suppose I tend to like to communicate. ...

T: Can I, umm, I want to ask some question about ...

P (interrupting): Yes, of course do.

T: On the whole over the last 6 months, have you been stable or ....?

P: Stable, overall, if you look at the graph. Well, on the weight side of things. On the mental side of things certainly going uphill.

T: Going uphill.

P: Going uphill, yeah.

T: That means?

P: More positive. More positive in terms of accepting help, I suppose.

T: Yes.

P: Going in search of help, allowing myself to be labelled. And that's, you know, a sort of accepting help. Coming here for instance.

### ***Example 16***

patient (to therapist): I apologise (she is distressed at this point).

T: No, that's all right.

P: Sometimes, you can see, how I feel (T: Yeah, sure.). it's not a really good part of my life. I told Dr. T. so I don't care to tell you.

Conversely, there may be an absence of the warmth and interest shown either by considerable negative behaviour (negative attitude and behaviour, dissatisfaction, displeased, criticism, and even hostility) or simply an absence of positive behaviours in the form of flatness and coldness, is regarded as lack of emotional engagement and is balanced against any evidence of emotional engagement when making the final overall judgment (example 17).

### ***Example 17***

T: I'm seeing you here in order to evaluate what sort of treatment program might work for you. I've to explain that as well as that being a clinical program, there is a research component attached. We try to find out what sorts of treatment help what sorts of people. So I want to ask you about it ... I can't involve you in the research aspect of the interview without your permission. Umm, and what I would be asking you to commit yourself too is to answering questions as best as you can. And also to see me in a year's time to see how have you got on. So I'm asking your

permission.

P: No, I can understand in several aspects, but I have had a lot of pain. I'd like to get out of pain. I don't want it to take another year of pain for another year.

T: Well, I'm not in the position until I talk to you...

P: It would take a year?

T: I don't know. I'm asking you for your permission and if you can give it I'd ask you to sign because we can't subject anyone to research without permission. So I would like you to look at the consent form and see what you think about it and ask me any question you have (T gives the consent form to the patient)

P: But I'm not anorexic (negative tone).

T: Umm, you ...

P (interrupting T): Anorexic, isn't that a person who eats and becomes sick?

T: No.

P: Who is anorexic?

T: Someone who has a poor appetite to eat and to get a very usual body weight.

P: But I'm eating more or less the same as I was eating.

T: But you are not in your usual weight. I don't like to talk about this because Dr. T. has seen you ...

P: Yes, I could understand that (negative tone).

T: Because ...

P (interrupting T): "OK", "OK", I lost weight (critical tone). (patient is reading the form) But I'm not eating disorder.

T: You are not committing yourself as an eating disordered. You are committing yourself to answer the question and again see me in a year.

P: But it says eating disorder clinic.

....

T: Other things that I'm interested in is how

P: Excuse me for the moment. Why did you ask my sister's name and my husband's name and not my daughter's name (she becomes angry at this point)?

T: Oh, I lost it.

P: No, you going to my husband, my ex-husband, my sister, what about my daughter?

....

T: One other thing that we are interested in is how people's experience of relationships effects what sort of treatment is helpful.

P: Yes, but lets face it, when I split up from my ex-husband life was hell and I literally hated every man, but things are changed. So how can you go back, I really get upset talking about the past ... (critical tone and nervousness) I'm not going back years.

T: But of course, I don't know either, but what I'm saying is that it seems that people's experience of relationships effects what treatments are helpful. And I'm wanting to know something

P (unwillingly): OK, OK. ... (turning face)

### **3.B.3. Allocation of Scores on the PRSS Scales**

Taking into account the interview as a whole, the following general principles should be used to determine the scores for a particular subscale of the PRSS:

**Self-Disclosure-** Self-disclosure is rated on a 6-point global scale from 0 to 5 (0= none; 1= little; 2= some; 3= moderate; 4= high; 5= marked).

High (4) or Marked (5) Self-Disclosure- Instances in which there are definite and clear-cut "Elaboration", "Spontaneity", and "Self-Personal Information", are rated " high self-disclosure" (4) or "marked self-disclosure" (5), according to the amount and depth of information and spontaneity disclosed.

Some (2) or Moderate (3) Self-Disclosure- Instances in which there are definite "Elaboration", but none or only limited "Spontaneity" or "Self-personal Information", are rated "some self-disclosure" (2) or "moderate self-disclosure" (3), according to the amount of information disclosed.

No (0) or Little (1) Self-Disclosure- In the absence of the rest of the self-disclosure criteria, if "Simple Response" is the predominant patient's mode of responding during the interview, " little self-disclosure" (1) can be rated. The rating of "no self-disclosure" (0) is reserved for patients who show a complete absence of the characteristics of self-disclosure.

**Emotional Engagement-** Emotional engagement is measured on a 6-point global scale from 0 to 5 (0= none; 1= little; 2= some; 3= moderate; 4= high; 5= marked).

High (4) or Marked (5) Emotional Engagement- Instances in which there are definite and clear-cut tonal warmth, concern and enthusiasm about and interest in the therapist and therapy are rated "high emotional engagement" (4) or "marked emotional engagement" (5), according to the amount of emotional engagement expressed.

Some (2) or Moderate (3) Emotional Engagement- Instances in which there are definite emotional

involvement and concern about or interest in the therapist and therapy, but any or only limited warmth of tone, are rated "some emotional engagement" (2) or "moderate emotional engagement" (3), according to the amount of concern and interest expressed.

No (0) or Little (1) Emotional Engagement- If there is only a slight amount of emotional engagement qualities like concern about or interest in the therapist and therapy, " little emotional engagement" (1) can be rated. The rating of "no emotional engagement" (0) is reserved for patients who show a complete absence of the qualities of emotional engagement as defined or negative attitude and behaviour, and dissatisfaction.

### 3.B.4. THE PRSS RELIABILITY

Table 3.B.1. shows the PRSS reliabilities for two raters (A & B) in 30 ED patients. Interrater reliability of the first rater and the second rater, calculated by Intra-Class Correlation (I.C.C.), for the two subsales are extremely high (0.92 - 0.94) over 30 randomly selected tapes.

**Table 3.B.1. Distribution of the PRSS reliabilities for two raters (A & B) in 30 ED patients**

	A	B	
PRSS Scales	Mean(s.d.)	Mean(s.d.)	r*
SD	3.300(1.208)	3.367(1.098)	0.92
EEn	3.100(1.348)	2.967(1.450)	0.94

**PRSS= Patient Response Style Scale; SD= Self-Disclosure; EEn= Emotional Engagement**  
**\* Intra-Class Correlation**

Although assessment of the PRSS validity is far from the purpose of the present study, preliminary results give support to both the construct and predictive validity of the PRSS. This issue will be briefly presented in chapter 5 under the “discussion of the results.”

It is important to note that in rating of the PRSS, first, both raters were experienced clinical psychologists and one of them had already been trained in rating of EE, and second, the PRSS has specific and clear guidelines, which can be easily replicated. Having said this, however, it is possible that the SD ratings have influenced the EEn ratings (SD rating criteria are more objective than EEn rating criteria) and this has raised artificially the PRSS reliability. Therefore, a word of caution is advisable about the high interrater reliability found for the PRSS scales in this study. A further reliability study using independent raters rating only one of the scales at a time is needed. It is, however, unlikely that reliability would be reduced significantly.

### **3.C. THE SELF- AND OTHER-BLAME SCALE (SOBS)**

Feelings of guilt or self-blame are somewhat difficult to assess. Nevertheless, some operationalizations include a focus on affect (rather than judgment or belief) which is negatively toned and which includes a sense of responsibility or causality. Although these elements can be distinguished, they often co-occur when people blame themselves for negative outcomes (see Shaver, 1985).

There is considerable variability in measurement of self/other-blaming attributions. Some investigators included self-report questionnaires (e.g., Klass, 1983, 1987; Meyer & Taylor, 1986). Others interviewed subjects (e.g., Bulman & Wortman, 1977; Kiecolt-Glaser & Williams, 1987; Tylor, Lichtman, & Wood, 1984) or their relatives (e.g., Affleck, Allen, McGrade, & McQueen, 1982; Affleck, McGrade, Allen, & McQueen, 1985; Affleck, Tennen, Croog, & Levine, 1987; Tennen, Affleck Gershman, 1986) or both subject and relative, separately (e.g., Healy, Stewart, & Copeland, 1993). Some studies included composite measures of self-report and observer ratings (Affleck, Tennen, Croog, & Levine, 1987; Bulman & Wortman, 1977; Frey, Rogner, Schuler, & Korte, 1985; Tylor, Lichtman, & Wood, 1984).

Most studies relied on content coding, rating scales, or both. Studies utilizing more than one measure do not support convergence between measures (e.g., Bulman & Wortman, 1977; Taylor, Lichtman, & Wood, 1984), hence the validity of the measures remains questionable (Turnquist, Harvey, & Anderson, 1988). None of these studies assessed self/other-blaming attributions in the context of a family interaction. To do this, a reliable observational instrument would have to be developed. Lack of such a reliable measure and the practical needs of the fourth Maudsley study (Dare & Eisler, 1995), are other reasons for developing the SOBS.



### **3.C.1. A Measure of Self- and Other-Blame: The Development of The Self- and Other-Blame Scales (SOBS)**

The SOBS is an observer-based rating instrument designed to assess self- and other-blaming attributions and guilt feelings experienced by patients and their families. As a reflection of psychological problems, self-blame expresses one's notion of responsibility. He/she is prone to hold him/herself responsible for any difficulties or problems that he/she encounters. Hence, attribution of responsibility is a key construct for understanding and rating of self-blame or guilt. Causality, negative feeling, and negative tone of voice are regarded as other elements of this phenomenon.

The term other-blame refers to the belief that another person is the cause of one's problem or is in some way responsible or blameworthy for an unwanted outcome.

This instrument is designed to be applied to videotape recordings of a family assessment interview. The interview consists of a series of questions and probes about family relationships. The main topics covered include the issues such as interpersonal conflicts, feelings of responsibility, causality, guilt, and blame between members of the family in terms of their connection to the patient's eating problem.

### **3.C.2. The SOBS Rating Criteria**

The following are relevant criteria for the rating of self and other-blame:

#### **a) Causality**

Causality is defined as an "antecedent or a subset of antecedents that is sufficient for the

occurrence of an effect" (Shaver and Drown, 1986, p.701). The following excerpts (examples 1-7) from the family assessment interviews refer to causality.

### ***Example 1***

T (to parents): My impression is that at different points you tend to blame yourselves.

M: Of course, yeah (crying).

F: Yes. My instinct tells me there must be a cause.

M (interrupting father): Yes, of course we (mother and father) feel it is our fault.

F: And L. (Patient) has made it very clear that she is, has been, not so much now, but has been unhappy with her relationship with her parents, and she has blamed us because of our fault in different ways.

T (to patient): Do you blame your parents?

P: (short pause) There isn't any point that I've tried to find someone or something to blame. Yes I tried to blame my mum. I blame myself too.

M: Yes I behaved her in a very bad way, actually.

### ***Example 2***

T: V. (Patient) Do you think that your parents a lot of times think that what they have done is wrong.

P: Mum, mum, my mum.

M: Yes, I think I did wrong.

T (to mother): Do you think actually you feel some of what you did, has caused anorexia.

M: Yes.

T: You do?

M: Yes.

### ***Example 3***

T (to parents): In a number of points both of you have talked as if you blame yourselves because of B's (patient) illness.

M: Yes it is (crying). Well, who else is attend to blame (father is laughing). You know, one has to ...

F: I'm not sure ... (laying back on the chair).

T: I'm struck that you actually you feel you caused anorexia, but parents don't cause anorexia.

M: No, I know that.

F (to mother): I suppose your feeling is the balance between ...

M: No, I suppose the thing is that he (father) has so much, but she's turned out like this, and (tearfully) after all she is my child, but I'm afraid I can't help her.

T: ... that's not the same as saying it's your fault.

M: No, but it's difficult to separate the two. I suppose I'm a sort of person who does tend to feel guilty if something's going wrong and I feel involved, whatever I'm doing.

#### **Example 4**

T: I suppose, you know, parents do tend to blame themselves. I suppose I've been struck by feeling that maybe you do feel that somehow you've caused it (patient's problem).

M: Well, yes, I suppose we do.

T: Do you?

M: Yes.

T (to father): Do you feel that as well?

F: Umm, yes, but I had put all the reasonable arguments I can before the accident, but I had no response. It's years and years and years I've tried to say, hey look, hang on, you think that's fine ok, but just listen to this point of view and I put that point of view upon him, but he didn't accept that what I'm saying is true. What I'm saying has got some sense to it (negative tone).

#### **Example 5**

T (to patient) How do you feel?

P: I feel guilty.

T: Guilty or ... Do you agree with what your mum said?

P: I suppose I do blame my mother because my problem started when she left my dad.

M (to therapist): Did you ask S. (Patient) if "she" feels guilty or blame...

P: (interrupting mother) Guilt and blame.

T: Both.

M (to patient): You don't really blame me, do you?

P: I do. I just feel that.

M: I want to know how much does she blame me and how much she feels angry about me.

#### **Example 6**

T: It sounds that all three of you blame yourselves.

F, M, & P (all together): Oh, yes.

F: I think we (father and mother) have done something wrong in her upbringing.

P: I think we hate each other.

F and M: Oh, no.

P: Not hate but dislike.

T (to patient): Each other or yourselves?

P: Each other.

F: May be hard to show much love but

M (interrupting father): may be hard to say but I love you, but I often don't like you.

P (to therapist, proving her point): yeah.

M: But my love is much stronger than dislike. I love you (she is crying at this point), but for some of the things you do I don't like you, you see.

F (to patient): You know you are loved. Every thing we've done we have done for you.

P: Yes I know I am loved.

F: Then we do love each other.

P: Yes.

T (to patient): Your mum said she didn't like what you do, but you see it as she dislikes you.

P: I see now as more I'd do it's me. So it's me.

T: You can't separate them.

P: No.

F: No one yet really knows. It's a nice thing to say oh we not to blame. It's your self-presentation, but I see something happened to my daughter, what caused this to happen, is it us? Have we been too loving? Have we been too protecting?

### ***Example 7***

T (to parents): At several points both of you talked as if you blame yourselves, as if you feel it is your fault.

M: Umm, yes as far as you (father) see and I see, anyway, there is a reason behind this sort of thing. I really must be a really bad parent to not notice something had happened to my little girl when she was ill. I blame myself for that (showing negative feeling, expressing negative tone)

T: Do you?

M: Yeah. I also blame myself for when she was a little girl she was quite tubby and I was when I was a child. So I wanted to, I used to make sure ...

## **b) Responsibility**

The term responsibility refers to the moral evaluation of an individual action (Shultz et al., 1981).

Responsibility attributions differ from causal attributions because they involve an evaluative component that compares the behaviour with normative criteria (Fincham, Beach, and Baucom, 1987). Responsibility is addressed in examples 8-10 below.

### **Example 8**

T: I've had the impression that all family members in some way blame themselves.

P: I don't think it would apply to my brother and sister.

T: To your dad?

P: I could say yes. I feel very much, umm, my dad doesn't want to face a lot of things.

T: Do you think he blames himself?

P: If he would allow himself to do so I think he might be afraid that he could, ...

T (to father): Do you blame yourself?

F: No I don't do. I mean, we have this discussion many times and right at the beginning with Dr. A and this was put very clearly to H. (Patient) Is it because your dad left home? Is this, you know,

T (interrupting father): No, no, no, I'm not in any way implying that you are to blame or that your (to patient) mother is to blame.

F: You are saying do "I" feel to blame.

T: Parents don't cause anorexia, I know as parents you did not make H. (Patient) anorexic. I'm not talking about a sort of rational response, I'm asking about what it feels like. H. Is saying that she thinks you wouldn't allow yourself to feel that, I'm wondering whether she is right.

F: Well, I don't know whether she is right or she is wrong. All I am saying is that the whole way I live my life, I believe in what I do. I have not been settled with a sense of responsibility of that H's condition at all. I put responsibility firmly on H. ... I've lived my life on that basis, you make up your mind, what you are going to do and do it. Whatever it is, right or wrong it's your responsibility, nobody else's.

### **Example 9**

T: ... What was your first reaction (to patient's illness)?

M: (spontaneously) I personally felt guilty. That was my first reaction. That in some way, umm I'd not let her (patient) down, but perhaps I pushed her trying to make her being more than what she wanted to be.

### **Example 10**

T (to mother): At several points you talked as if in some way you blame yourself.

M: Umm, yeah.

T: Do you?

M: Yeah.

T: You do.

M: Yeah. (while laughing) I don't put a finger on it saying this is only our fault, but it has written in so many ways, people said it in so many ways that the mother is the king pin of the family. The mother's role is to feed family. Therefore, children see mother and food as just the same thing, which is probably is not true in my case because I was always feeding P. (Patient) When she was seem to need it for her life. I'm a very mumsy mum, unfortunately. So I feel I want to lunch P. Now, it's a bit late, unfortunately. But I just think I perhaps cocooned her for too long.

### **c) Negative Feeling**

Negative feelings may involve a statement against self or other. One could express some dissatisfaction or regret concerning his/her or others mistakes, either he/she takes causal (the extent to which an agent is the cause of some outcome) or moral responsibility (the extent to which the agent is deserving of blame for the outcome), or simply accepts some mistakes. In the latter instance one (usually patient's relative) blames him/herself to display sympathy towards the patient. Negative feelings are shown in examples 11-14.

#### ***Example 11***

T (to father): Do you blame yourself as well?

F: Umm, I suppose maybe in the back of my mind I was thinking I did something wrong, I don't know. I suppose yes.

#### ***Example 12***

T: At several points the way you talked about what happened I had the impression that you blame yourselves.

F: I think I feel a bit guilty about that, but I didn't know what was going on in her own.

T (to mother): Do you blame yourself as well?

M: Umm, well, yes. ..., but we couldn't put finger on it.

#### ***Example 13***

T (to husband): My impression is that perhaps she (Patient) blames you.

H: Yes, it's obvious.

T: Do you blame yourself as well?

H: Umm, well yes, I suppose that's right.

#### ***Example 14***

T (to mother): Do you sometimes feel guilty?

M: Well, if her (C.) unhappiness is according to, is as a result of what has happened to her in her childhood, I must be a bad mother.

T: The question is not whether you are a bad mother or not. I want to know whether that's the way you felt.

M: Umm, I honestly feel that I didn't do too bad.

Further examples can be seen in numbers 1, 3, and 5.

#### **d) Negative Tone of voice**

Negative changes in tonal aspects when blaming is regarded as a criterion which rating of self- and other-blame is based. Self/other-blame, however, is defined primarily by its content (including causality, responsibility, and negative feeling), its depth and severity is determined by the tone in which it is spoken. Implying that something bad has happened, negative tone of voice may be used only to define the depth and severity of self- and other-blaming. The following are examples (15-17) of presenting negative tone of voice.

##### ***Example 15***

T (to parents): My impression is that at different points you tend to blame yourselves.

M: Of course, yeah (crying).

F: Yes. My instinct tells me there must be a cause.

M (interrupting father): Yes, of course we (mother and father) feel it is our fault.

F: And L. (Patient) has made it very clear that she is, has been, not so much now, but has been unhappy with her relationship with her parents, and she has blamed us because of our fault in different ways.

M: Yes I behaved her in a very bad way, actually.

##### ***Example 16***

T (to parents): In a number of points both of you have talked as if you blame yourselves because of B's (patient) illness.

M: Yes it is (crying). Well, who else is one ment to blame (father is laughing). You know, one has to ...

M: No, I suppose the thing is that he (father) has so much, but she's turned out like this, and (tearfully) after all she is my child, but I'm afraid I cant help her.

T (to father): Do you blame yourself?

F: No I don't do. I mean, we have this discussion many times and right at the beginning with Dr. A and this was put very clear to H. (Patient) Is it because your dad left home? Is this, you know.

T (interrupting father): No, no, no, I'm not in any way implying that you are to blame or that your (patient) mother is to blame.

F: You are saying don't do "I" feel to blame.

T: Parents don't cause anorexia, I know as parent you did not make H. (Patient) anorexic. I'm not talking about a sort of rational response, I'm asking about what it feels like. H. Is saying that

she thinks you wouldn't allow yourself to feel that, I'm wondering whether she is right.

F: Well, I don't know whether she is right or she is wrong. All I am saying is that the whole way I live my life, I believe in what I do. I have not been settled with a sense of responsibility of that H's condition at all. I put responsibility firmly on H. ... I've lived my life on that basis, you make up your mind, what you are going to do and do it. Whatever it is, right or wrong it's your responsibility, nobody else's.

### ***Example 17***

T (to patient) How do you feel?

P: I feel guilty.

T: Guilty or ... Do you agree with what your mum said?

P: I suppose I do blame my mother because my problem started when she left my dad.

M (to therapist): Did you ask S. If "she" feels guilty or blame...

P: (interrupting mother) Guilt and blame.

T: Both.

M (to patient): You don't really blame me, do you?

P: I do. I just feeling that.

M: I want to know how much does she blame me and how much she feels angry about me.

### **3.C.3. Allocation of Scores on the SOBS**

Self- and other-blame is measured on a 6-point scale from 0 to 5 (0= none; 1= little; 2= some; 3= moderate; 4= high; 5= marked).

Taking into account the whole SOBS segment of the interview, the following general principles should be used to determine the scores for self/other-blame (SOB):

High (4) or Marked (5) SOB- Instances in which there are definite and clear-cut SOB related characteristics, "Causality"; "Responsibility"; "Negative Feeling"; and "Negative Tone of Voice", are rated "high SOB" (4) or "marked SOB" (5), according to the amount and depth of SOB qualities expressed.

Moderate (3) SOB- Instances in which there are definite "Causality" or "Responsibility", but any or only limited "Negative feeling" and "Negative Tone of Voice", are rated "moderate SOB".



Little (1) or Some (2) SOB- Instances in which there are definite "Negative Feeling" and/or "Negative Tone of Voice", but any "Causality" and "Responsibility", can be rated " little SOB" (1) or "some SOB" (2), according to the amount of negative tone and feeling shown in the interview.

No (0) SOB- The rating of "no SOB" is reserved for subjects who express a complete absence of the characteristics of SOB.

### 3.C.4. The SOBS Reliability

The distribution of the SOBS in 36 families (including 36 patients, 28 mothers, 19 fathers, and 8 husbands) with an eating disorder patient is presented in Table 3.C.1. Interrater reliability of the first rater and the second rater, calculated by Intra-Class Correlation (I.C.C.), for all SOBS components are high or extremely high (0.80 - 0.98) over 36 randomly selected families.

**Table 3.C.1. Distribution of the SOBS reliabilities for two raters (A & B) in 91 ED patients and their relatives**

	A	B	
	Mean(s.d.)	Mean(s.d.)	r*
<b>SOBS</b>			
<u><b>Self-Blame</b></u>			
<b>Mother</b>	3.000 (1.901)	3.000 (1.861)	0.98
<b>Father</b>	1.684 (1.734)	1.737 (1.759)	0.97
<b>Husband</b>	2.444 (1.130)	2.444 (1.130)	0.80
<b>Patient</b>	1.972 (1.920)	1.917 (1.903)	0.98
<u><b>Other-Blame</b></u>			
<b>Mother to Patient</b>	0.333 (0.832)	0.370 (0.926)	0.97
<b>Father to Patient</b>	0.421 (0.902)	0.526 (1.124)	0.94
<b>Husband to Patient</b>	0.222 (0.667)	0.222 (0.667)	1.00
<b>Patient to Mother</b>	0.778 (1.625)	0.556 (1.396)	0.80
<b>Patient to Father</b>	0.526 (1.349)	0.579 (1.427)	0.98
<b>Patient to Husband</b>	0.556 (1.667)	0.500 (1.581)	1.00

\* r= Intra-Class Correlation; SOBS= Self- and Other-Blame Scale

This study is not in the position of testing the validity of the newly developed instrument. Preliminary results, however, lend support to both the construct and predictive validity of the SOBS. These results will be reported in chapter 5 under the “discussion of the results.”

**PART II**

**CHAPTER 4**

**PRESENTATION OF THE RESULTS**

Chapter 4 presents the results of this investigation in five sections. Section A reports levels of family EE at first assessment and at the termination of treatment. The changes of family EE during the course of therapy, clinical features of the diagnostic groups, and the distribution of treatment outcome for AN and BN patients are described. Section B presents the distribution of personality disorder (PD) ratings. Comparison of PDs in AN and BN patients, and the relationship between family Expressed Emotion (EE) and PD are described. The following two sections demonstrate findings based on the two newly developed rating instruments, the PRSS and the SOBS. Sections C and D report on the results of the PRSS and the SOBS in association with clinical and family factors, respectively. Section E presents the prognostic value of family EE, personality disorders, the PRSS variables, and the SOBS scale.

**4.A. EXPRESSED EMOTION**

**4.A.1. Clinical Features of the Diagnostic Groups**

Clinical variables were examined for their association with the two diagnostic groups, AN and BN. As can be seen in Table 4.A.1., there were no statistically significant differences between anorexics and bulimics in terms of age at presentation, age of eating disorder onset, duration of illness, BMI, BDI, average score of the Morgan-Russel scales, IIP scores, and MOCI scores. As would be expected, the BN patients had significantly higher scores on the BITE and the bulimic subscale of the BDI (see Table 4.A.1.).

**Table 4.A.1. Clinical Features of AN and BN patients**

<b>Variables</b>	<b>Diagnostic Groups</b>		<b>P*</b>
	<b>AN(M/s.d.)</b>	<b>BN(M/s.d.)</b>	
<b>Age at Presentation</b>	<b>25.93(6.69)</b>	<b>26.47(6.37)</b>	<b>.75</b>
<b>Age at Onset of ED</b>	<b>18.69(4.88)</b>	<b>18.68(6.37)</b>	<b>.99</b>
<b>Duration of ED</b>	<b>6.04(5.77)</b>	<b>7.78(6.52)</b>	<b>.30</b>
<b>Body Mass Index</b>	<b>15.33(1.65)</b>	<b>15.51(1.57)</b>	<b>.68</b>
<b>Beck Depression Inventory</b>	<b>25.70(12.66)</b>	<b>27.40(11.59)</b>	<b>.70</b>
<b>MRAV</b>	<b>5.83(1.64)</b>	<b>5.43(1.99)</b>	<b>.43</b>
<b>MOCI</b>	<b>9.19(5.39)</b>	<b>7.36(3.38)</b>	<b>.20</b>
<b>The Inventory of Interpersonal Problem</b>	<b>1.88(.58)</b>	<b>2.05(.26)</b>	<b>.37</b>
<b>BITE Total</b>	<b>15.26(7.86)</b>	<b>32.90(8.30)</b>	<b>.001</b>
<b>BITE Severity</b>	<b>2.96(3.25)</b>	<b>10.70(3.09)</b>	<b>.001</b>
<b>BITE Symptoms</b>	<b>12.30(5.63)</b>	<b>22.20(7.82)</b>	<b>.003</b>
<b>EDI Interoceptive Awareness</b>	<b>9.43(6.68)</b>	<b>16.75(8.84)</b>	<b>.05</b>
<b>EDI Bulimia</b>	<b>.93(1.23)</b>	<b>8.12(6.22)</b>	<b>.01</b>
<b>EDI Maturity Fear</b>	<b>2.63(2.31)</b>	<b>6.87(5.38)</b>	<b>.06</b>
<b>EDI Body dissatisfaction</b>	<b>13.76(6.65)</b>	<b>14.00(6.25)</b>	<b>.92</b>
<b>EDI Interpersonal Distrust</b>	<b>7.06(5.05)</b>	<b>10.37(5.15)</b>	<b>.13</b>
<b>EDI Ineffectiveness</b>	<b>9.66(6.52)</b>	<b>13.87(7.86)</b>	<b>.19</b>
<b>EDI Perfectionism</b>	<b>7.83(5.20)</b>	<b>6.75(5.72)</b>	<b>.63</b>
<b>EDI Drive for Thinness</b>	<b>11.20(6.31)</b>	<b>12.87(5.13)</b>	<b>.45</b>

**\* t-test for independent samples; AN= Anorexia Nervosa; BN= Bulimia Nervosa; MRAV= Average Score of the Morgan-Russell Scales; MOCI= The Maudsley Obsessional-Compulsive Inventory; BITE= The Bulimic Inventory Test, Edinburgh; EDI= The Eating Disorder Inventory**

4.A.2. The Distribution of Family Expressed Emotion

The distribution of EE ratings is presented in Table 4.A.2.

Table 4.A.2. Distribution of Expressed Emotion among ED patients’ relatives at Intake

EE Scales	Mean (s.d.)
<b><u>Mothers (n= 45)</u></b>	
CC	1.60(2.02)
HOS	0.08(0.35)
EOI	1.84(1.22)
W	3.77(0.85)
PR	1.31(1.22)
<b><u>Fathers (n=31)</u></b>	
CC	1.29(1.46)
HOS	0.03(0.18)
EOI	0.74(0.93)
W	3.41(0.84)
PR	0.83(0.73)
<b><u>Husbands (n=12)</u></b>	
CC	2.66(2.74)
HOS	0.25(0.86)
EOI	0.83(0.71)
W	3.16(1.26)
PR	0.83(0.93)

ED= Eating Disorders; EE= Expressed Emotion; CC= Critical Comments; HOS= Hostility; EOI= Emotional Over-Involvement; W= Warmth; PR= Positive Remarks

The levels of Critical Comments (CC), Hostility (HOS), Emotional Over-Involvement (EOI) and Positive Remarks (PR) were rated low; 34.5% of relatives scored no CC, 92.2% no HOS, and 21.6% no EOI, 25.9% made only one CC, and 39.2% made only one EOI. The relatives were rated as being moderately warm in the way they related to the patient during the interview.

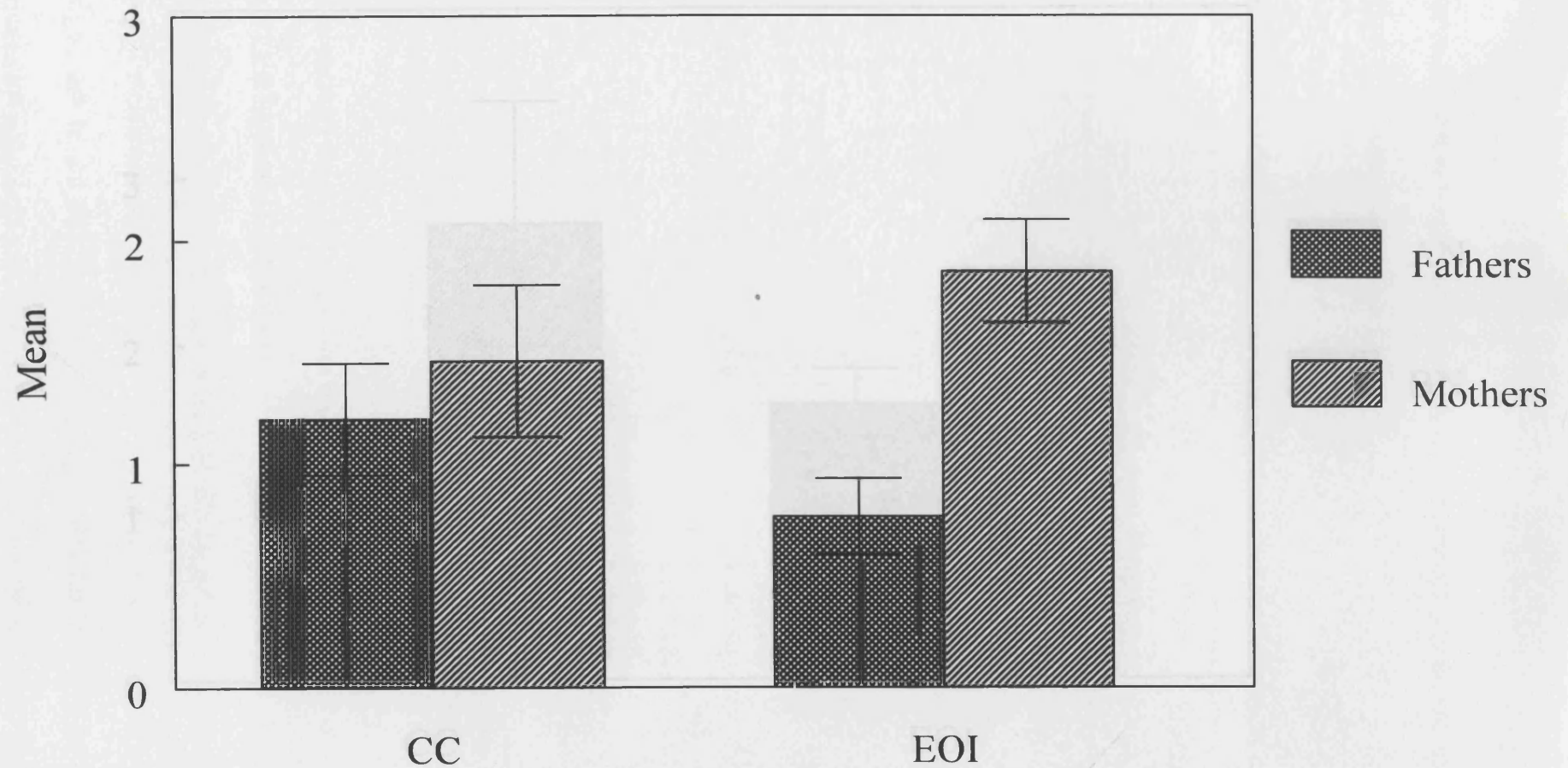
A comparison between mothers and fathers revealed that the mothers’s score on EOI was significantly more than fathers’ (t= - 4.31, df= 29, p < .001). Mothers also scored higher on Warmth (W) (t= - 3.25, df= 29, p < .003) and PR (t= - 2.83, df= 29, p < .008) than did fathers.

There was no significant difference among relatives in their scores of CC. Distribution of parental CC and EOI is presented in Figure 4.A.1.

A comparison between single- and two-parent families showed that the level of CC in the former (M= 2.45, s.d.= 2.42) setting is higher than the latter (M= 1.33, s.d.= 1.36) and the difference reached levels of statistical significance ( $t= 1.97$ ,  $df= 41.86$ ,  $p < .05$ ).

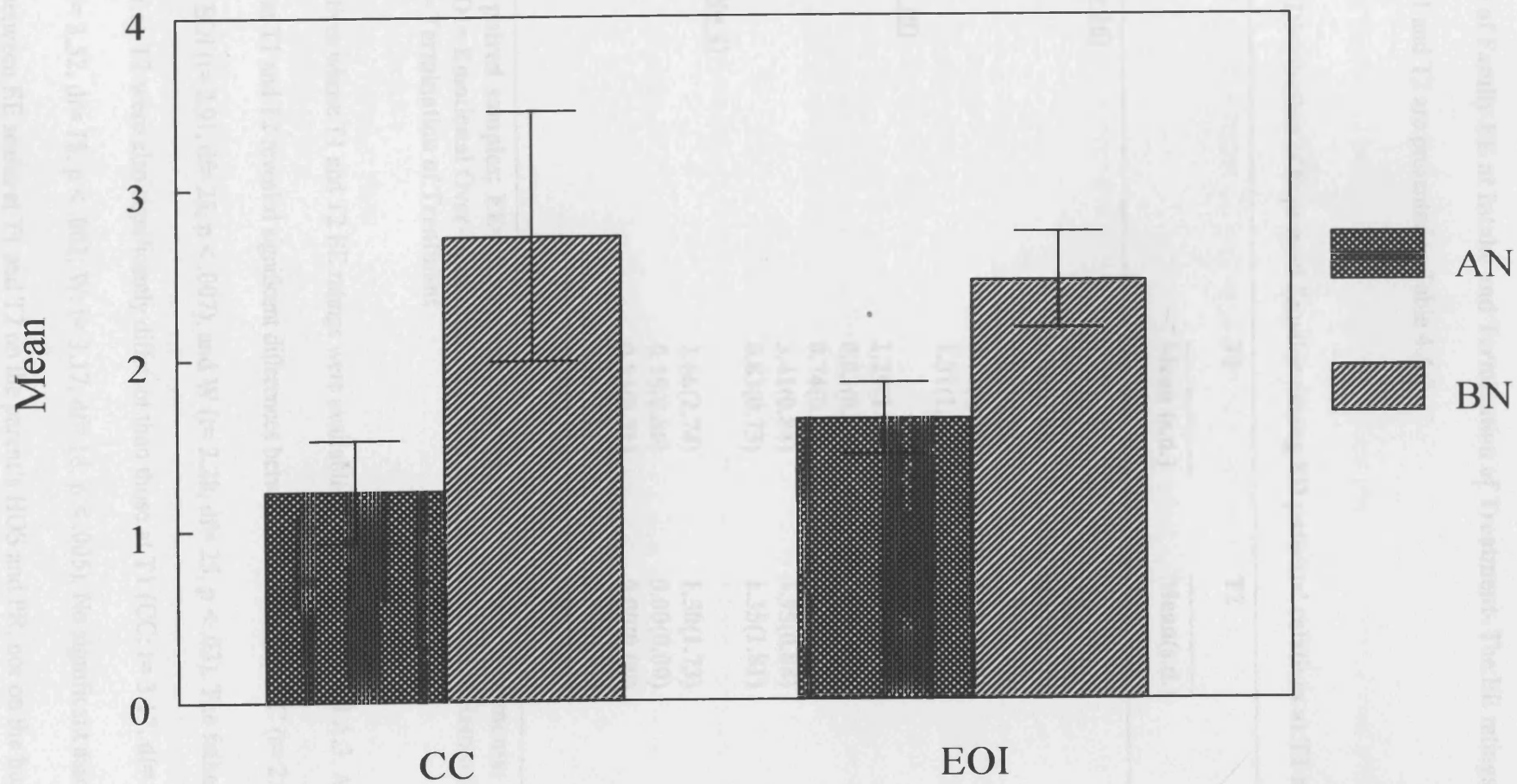
**The Association Between Family EE and Eating Disorder Subgroups-** Family EE variables were associated with diagnosis in some aspects. Mothers of bulimic patients scored higher on CC (M= 2.72, s.d.= 2.45) than did mothers of anorexic patients (M= 1.23, s.d.= 1.75). This difference, however, just failed to reach statistical significance ( $t= -1.87$ ,  $df= 13.49$ ,  $p < .08$ ). Mothers of bulimics also showed significantly more EOI (M= 2.45, s.d.= .93) than mothers of anorexics (M= 1.64, s.d.= 1.25;  $t= -2.28$ ,  $df= 22.69$ ,  $p < .03$ ). Figure 4.A.2. presents the distribution of mothers' CC and EOI in AN and BN patients.

Figure 4.A.1. Distribution of Parents'  
CC and EOI in ED Patients at T1



CC= Critical Comments; EOI= Emotional Over-Involvement; ED= Eating Disorders; T1= Intake

Figure 4.A.2. Distribution of Mothers' CC and EOI in AN and BN Patients



CC= Critical Comments; EOI= Emotional Over-Involvement; AN= Anorexia Nervosa; BN= Bulimia Nervosa



**Comparison of Family EE at Intake and Termination of Treatment-** The EE ratings of the families at T1 and T2 are presented in Table 4.A.3.

**Table 4.A.3. Distribution of Expressed Emotion among ED patients’ relatives at T1 and T2**

	T1	T2	
EE Scales	Mean (s.d.)	Mean(s.d.)	P*
<b><u>Mothers(N= 26)</u></b>			
CC	1.60(2.02)	0.69(1.46)	.01
HOS	0.08(0.35)	0.07(0.27)	.57
EOI	1.84(1.22)	1.03(1.11)	.007
W	3.77(0.85)	4.15(0.88)	.03
PR	1.31(1.22)	1.19(0.84)	.87
<b><u>Fathers(N= 19)</u></b>			
CC	1.29(1.46)	0.35(0.74)	.005
HOS	0.03(0.18)	0.00(0.00)	.33
EOI	0.74(0.93)	0.20(0.41)	.002
W	3.41(0.84)	3.95(0.88)	.005
PR	0.83(0.73)	1.35(1.81)	.18
<b><u>Husbands(N= 4)</u></b>			
CC	2.66(2.74)	1.50(1.73)	.08
HOS	0.25(0.86)	0.00(0.00)	--
EOI	0.83(0.71)	0.00(0.00)	.21
W	3.16(1.26)	3.00(0.81)	1.00
PR	0.83(0.93)	0.25(0.50)	--

\* t-test for paired samples; EE= Expressed Emotion; CC= Critical Comments; HOS= Hostility; EOI= Emotional Over-Involvement; W= Warmth; PR= Positive Remarks; T1= Intake; T2= Termination of Treatment

Only the relatives whose T1 and T2 EE ratings were available are shown in Table 4.A.3. Analysis of EE scales at T1 and T2 revealed significant differences between the mother’s CC (t= 2.62, df= 25, p < .01), EOI (t= 2.91, df= 25, p < .007), and W (t= 2.28, df= 25, p < .03). The father’s CC, EOI, and W at T2 were also significantly different than those at T1 (CC: t= 3.17, df= 18, p < .005; EOI: t= 3.52, df= 18, p < .002; W: t= 3.17, df= 18, p < .005). No significant association was found between EE scales at T1 and T2 on the parent’s HOS and PR, nor on the husband’s

EE scales. The distribution of parental CC and EOI is presented in Figure 4.A.3.

**Correlational Patterns of Family EE Scales-** Findings concerning intercorrelational patterns of family EE scales showed a significant positive correlation between CC and EOI for both mothers and fathers; those with higher CC expressed more EOI (see Table 4.A.4.).

**Table 4.A.4. Intercorrelations of family EE scales**

EE Scales	r*	p
Mother’s CC - Mother’s EOI	0.50	0.001
Fathers’ CC - Fathers’ EOI	0.51	0.003
Husbands’ CC - Husbands EOI	0.34	0.26

**\* Spearman Correlation Coefficients; EE= Expressed Emotion; CC= Critical Comments; EOI= Emotional Over-Involvement**

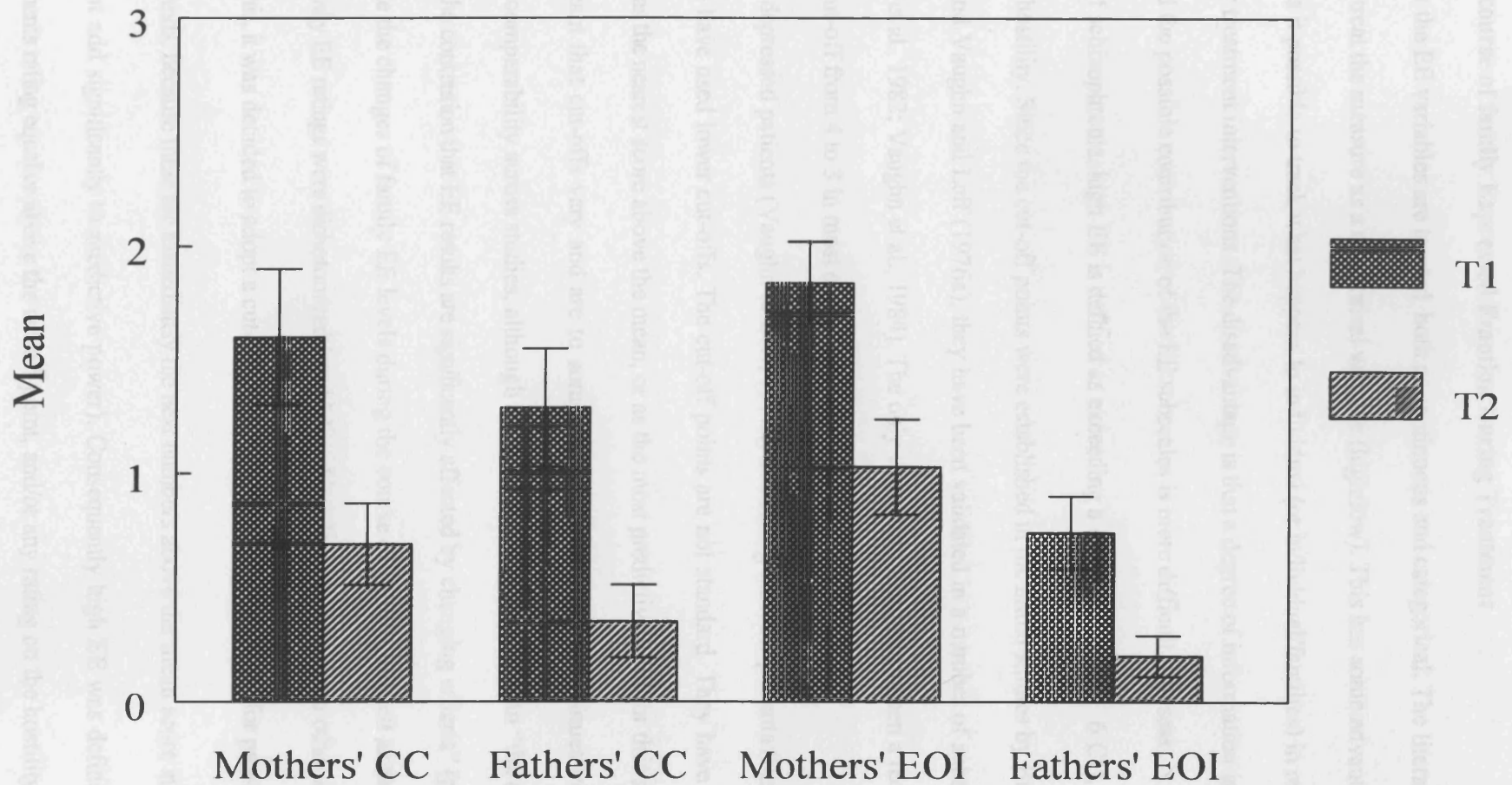
The examination of correlations between EE scales for maternal and paternal pairs also revealed significant correlations for CC, HOS, W, and PR, but not for EOI (see Table 4.A.5.).

**Table 4.A.5. Correlations of family EE scales**

EE Scales	r	p
Mothers’ CC - Fathers’ CC	0.35*	0.05
Mothers’s HOS - Fathers’ HOS	1.00**	0.001
Mothers’ EOI - Fathers’ EOI	0.18**	0.31
Mothers’ W - Fathers’ W	0.55**	0.001
Mothers’ PR - Fathers’ PR	0.40*	0.02

**\* Pearson Correlation Coefficients; \*\* Spearman Correlation Coefficients; EE= Expressed Emotion; CC= Critical Comments; HOS= Hostility; EOI= Emotional Over-involvement; W= Warmth; PR= Positive Remarks**

Figure 4.A.3. Distribution of Parents' CC and EOI in ED Patients at T1 and T2



CC= Critical Comments; EOI= Emotional Over-Involvement; ED= Eating Disorder; T1= Intake; T2= Termination of Treatment

#### **4.A.3. The course of family Expressed Emotion During Treatment**

In this study the EE variables are treated both as continuous and categorical. The literature on EE tends to treat the measure as a categorical variable (high/low). This has some advantages in that it makes it possible to track what happens to individual (or individual/families) in response to particular treatment interventions. The disadvantage is that a degree of information is lost in that way and the possible contribution of the EE subscales is more difficult to assess.

In studies of schizophrenia, high EE is defined as exceeding a cut-off point of • 6 CC, or • 4 EOI, or any hostility. Since the cut-off points were established in the initial studies by Brown et al. (1972) and Vaughn and Leff (1976a), they have been validated in a number of subsequent studies (Leff et al., 1982; Vaughn et al., 1984). The only consistent change has been a reduction in the EOI cut-off from 4 to 3 in most of the research published after 1986.

Studies with depressed patients (Vaughn & Leff, 1976a) and eating disorder patients (Szmukler et al., 1985) have used lower cut-offs. The cut-off points are not standard. They have mainly arisen either as the nearest score above the mean, or as the most predictive level for that sample.

This has meant that cut-offs vary and are to some extent arbitrary. This continues to cause problems of comparability across studies, although as Kavanagh (1992) points out “the data do not support the contention that EE results are significantly affected by changing criteria” (p. 603).

To investigate the changes of family EE levels during the course of therapy, as well as the other purposes, family EE ratings were dichotomized into high and low in a way similar to other studies.

To achieve this, it was decided to adopt a cut-off point of • 2 critical comments for parents and

• 3 for husbands, because these are immediately the next numbers above the mean score at intake (EOI did not add significantly to predictive power). Consequently high EE was defined as a critical comments rating equal or above the cut-off point, and/or any rating on the hostility scale.

Using this criterion, 28.9% (13/45) of the mothers and 25.8% (8/31) of the fathers could be considered as high EE at intake.

Using the same criterion, 19.2% (5/25) of the mothers and 15% (3/20) of the fathers were rated as high EE at the termination.

A small number of patients were married and their family data therefore relates to their husbands rather than parents. They are treated as a separate group for the purpose of analysis of family data.

Table 4.A.6. shows the changes of family EE levels during the course of therapy (T1-T2). Sixty-seven per cent of the mothers (4/6) and fathers (4/6) changed from high EE to low EE in the course of treatment. This change, however, was not statistically significant comparing to 33% (2/6) of those parents whose EE ratings during the course of treatment remained unchanged (McNemar Two-Tailed Exact p : mothers= .68; Fathers= .37). In three families, the EE level changed from low to high. Patients in each of these families had poor progress at the end of treatment period. The husbands' EE levels during the course of therapy remained unchanged (see Table 4.A.6.).

**Table 4.A.6. The course of family EE Levels over treatment period (T1-T2)**

	T2		
T1	Low EE	High EE	Total
<u>Mothers</u>			
Low EE	18	2	20
High EE	4	2	6
Total	22	4	26
	McNemar Two-Tailed Exact p = .68		
<u>Fathers</u>			
Low EE	12	1	13
High EE	4	2	6
Total	16	3	19
	McNemar Two-Tailed Exact p = .37		
<u>Husband</u>			
Low EE	2	0	2
High EE	0	2	2
Total	2	2	4
	McNemar Two-Tailed Exact p = 1.00		

**EE= Expressed Emotion; T1= Intake; T2= Termination of Treatment**

Separate examination of the EE subscales, however, demonstrated significant changes. Wilcoxon Matched-Pairs Signed-Ranks Test revealed that the changes of EE scales during the course of therapy were statistically significant in terms of CC, EOI, and W ratings for the mothers' and fathers', but not for husbands' scores. The changes of HOS and PR were not found to be significant for any of the patients' relatives (see Table 4.A.7.).

**Table 4.A.7. The course of family EE Scales over the Treatment period (T1-T2)**

<b>EE Scales</b>	<b>T1-T2(Z score/Probability )*</b>
<b><u>Mothers(N= 26)</u></b>	
<b>CC</b>	<b>-2.42(.015)</b>
<b>HOS</b>	<b>-.57(.56)</b>
<b>EOI</b>	<b>-2.73(.006)</b>
<b>W</b>	<b>-2.08(.037)</b>
<b>PR</b>	<b>-.17(.86)</b>
<b><u>Fathers(N= 19)</u></b>	
<b>CC</b>	<b>-2.81(.004)</b>
<b>HOS</b>	<b>-1.00(.31)</b>
<b>EOI</b>	<b>-2.71(.006)</b>
<b>W</b>	<b>-2.62(.008)</b>
<b>PR</b>	<b>-1.41(.15)</b>
<b><u>Husband(N= 4)</u></b>	
<b>CC</b>	<b>-1.63(.10)</b>
<b>HOS</b>	<b>.00(1.00)</b>
<b>EOI</b>	<b>-1.34(.17)</b>
<b>W</b>	<b>.00(1.00)</b>
<b>PR</b>	<b>.00(1.00)</b>

**\* Wilcoxon Matched-Pairs Signed-Ranks Test; EE= Expressed Emotion; CC= Critical Comments; HOS= hostility; EOI= Emotional Over-Involvement; W= Warmth; PR= Positive Remarks; T1= Intake; T2= Termination of Treatment**

#### **4.A.4. The Distribution of Treatment Outcome for AN and BN Patients**

Table 4.A.8. presents the general outcome categories for the two diagnostic groups. Among the patients, 53% (26/49) of anorexics were rated as either recovered, significantly improved, or improved whereas 23% (3/13) of bulimics were rated in this way. A comparison between the two diagnostic groups revealed that AN patients showed significantly more progress during the course of treatment than did BN patients (Fisher's Exact  $p = .04$ ). Figure 4.A.4. shows the distribution of treatment outcome for AN and BN patients.

**Table 4.A.8. Distribution of ED Diagnostic Groups for General Outcome Categories**

Diagnosis	General Outcome Category (No. of patients)			
	Recovered	Sign. Improved	Improved	Poor
AN	5(10.2)	5(10.2)	16(32.7)	23(46.9)
BN	1(7.7)	2(15.4)	0(.00)	10(76.9)
Two-Tail Fisher's Exact p = .04*				

ED= Eating Disorder; AN= Anorexia Nervosa; BN= Bulimia Nervosa; N(%)

\* Fisher's Exact P for Tables larger than 2×2 (Fleiss, 1981; Joe, 1985; Mehta, 1983)

Next, to examine the relationship between eating disorder diagnoses, type of therapy and treatment outcome, similar analyses were performed. No significant relationships were found between the diagnostic groups and outcome categories for any of the therapy types when the poor progressed group was compared to the rest of the outcome categories (see Table 4.A.9.).

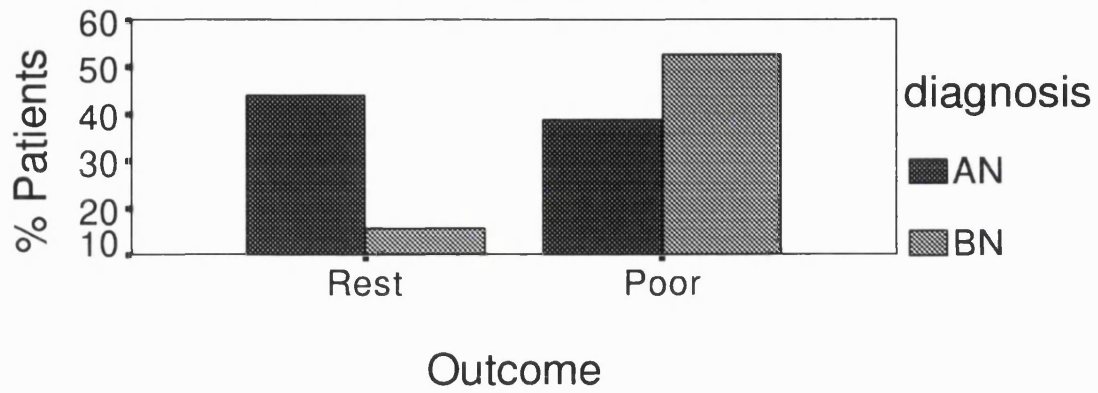
**Table 4.A.9. Distribution of the ED Diagnostic Groups and Type of Treatment for GOC**

Therapy & Diagnosis	General Outcome Category (No. of patients)			
	Recovered	Sign. Improved	Improved	Poor
<b><u>FOCAL</u></b>				
AN	2	2	7	3
BN	1	1	0	1
	NS			
<b><u>FAMILY</u></b>				
AN	1	1	4	7
BN	0	1	0	3
	NS			
<b><u>COGNITIVE</u></b>				
AN	2	2	3	6
BN	0	0	0	2
	NS			
<b><u>SUPPORTIVE</u></b>				
AN	0	0	2	7
BN	0	0	0	4
	NS			

ED= Eating Disorder; GOC= General Outcome Categories; AN= Anorexia Nervosa; BN= Bulimia Nervosa; NS= Not Significant



Figure 4.A.4. Distribution of Outcome for AN and BN Patients



**AN= Anorexia Nervosa**

**BN= Bulimia Nervosa**

**Outcome= Poor vs Rest (Recovered, Significantly Improved, Improved)**

## 4.B. PERSONALITY DISORDERS

### 4.B.1. The Distribution of the PAS Personality Disorder Diagnoses

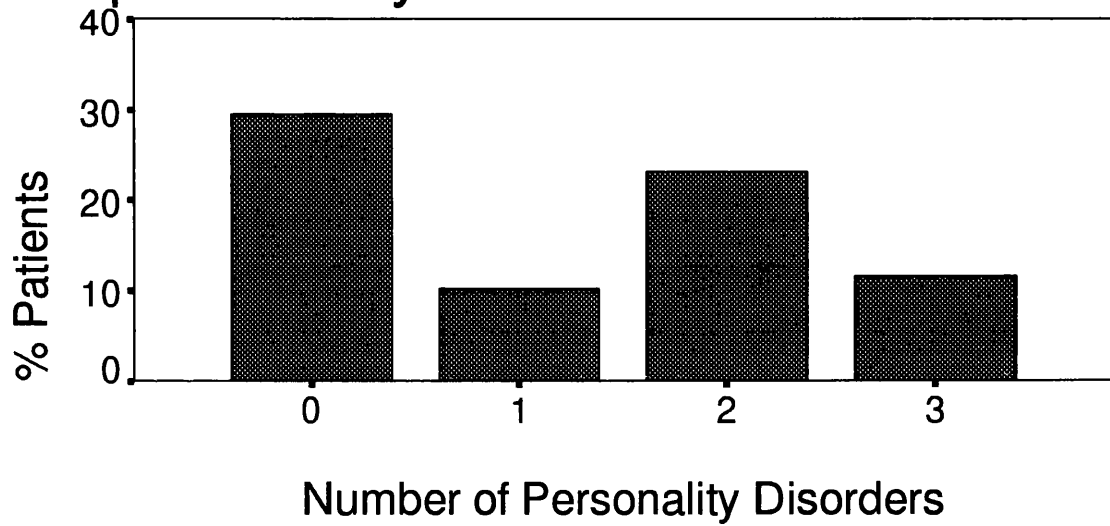
Overall, 23 subjects (39.7%) had no personality disturbances on the PAS. Eight subjects (13.8%) met the PAS criteria for personality difficulties, 18 (31%) for personality disorders, and 9 (15.5%) for severe personality disorders (see Figure 4.B.1.). In total, 27(46.5%) patients met the PAS criteria for at least one PD. The BN patients tended to fulfill the criteria more than the AN patients (53.3% vs. 44.2%) but the difference was not statistically significant. Twenty-seven patients (46.6%) had more than one personality disturbances. The distribution of personality disorder diagnoses is detailed in Table 4.B.1.

**Table 4.B.1. Distribution of the PAS Personality Disorder Diagnoses in Eating Disorder Patients**

	Eating Disorder Subtypes					
	All Patients(n= 58)		AN(n=43)		BN(n=15)	
PAS Personality Diagnoses	n	%	n	%	n	%
Normal	23	39.7	18	41.9	5	33.3
<b><u>Personality Abnormality</u></b>						
Personality Difficulty	8	13.8	6	14	2	13.3
Personality Disorder	18	31	12	27.9	6	40
Severe Personality Disorder	9	15.5	7	16.3	2	13.3
<b>&gt;1 Personality Abnormality</b>	<b>27</b>	<b>46.6</b>	<b>20</b>	<b>46.5</b>	<b>7</b>	<b>46.7</b>
<b><u>Personality Type</u></b>						
Anxious	9	15.5	7	16.3	2	13.3
Histrionic	5	8.6	1	2.3	4	26.7
Passive-Dependent	6	10.3	5	11.6	1	6.7
Sensitive-Aggressive	4	6.9	3	7	1	6.7
Avoidant	4	6.9	4	9.3	0	0
Paranoid	3	5.2	2	4.7	1	6.7
Dysthymic	2	3.4	2	4.7	0	0
Anankastic	2	3.4	1	2.3	1	6.7

AN= Anorexia Nervosa; BN= Bulimia Nervosa; PAS= Personality Assessment Schedule

Figure 4.B.1. distribution of the PAS  
personality disorders in ED Patients



**PAS= Personality Assessment Schedule**  
**ED= Eating Disorders**

As can be seen in Table 4.B.1., the most common diagnosis in the AN group was “anxious”, which occurred in 16.3% of the patients, and the most common diagnosis in the BN group was “histrionic”, which occurred in 26.7% of the patients. Only 2.3% of anorexics fulfilled the PAS criteria for histrionic personality disorder, a significant difference (Two-Tail Fisher’s Exact  $p < .01$ ). Figure 4.B.2. demonstrates the prevalence of anxious and histrionic PDs in AN and BN patients.

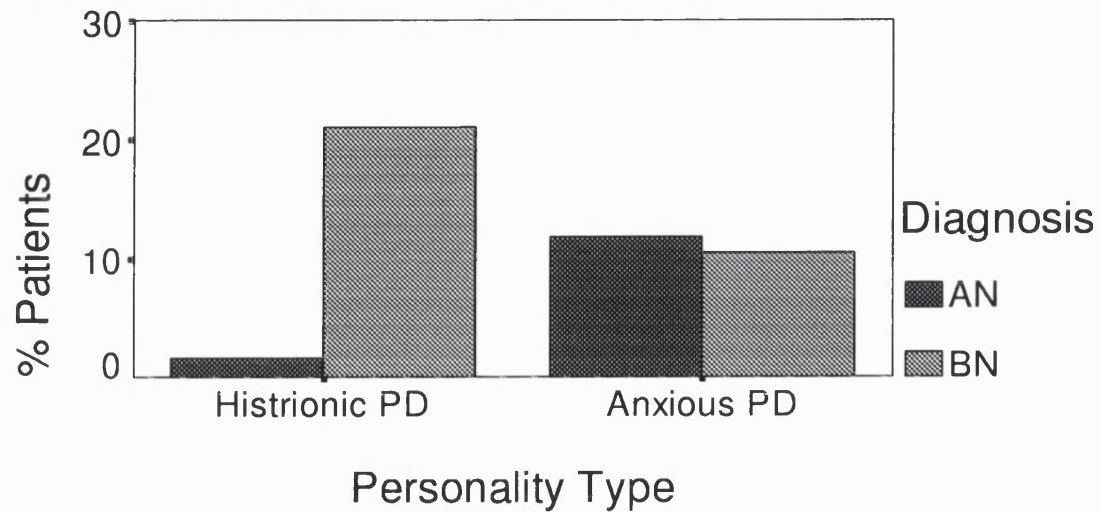
The differential distribution of the PAS personality disorder diagnoses in the AN and BN groups is presented in Table 4.B.2. When the four clusters of personality disorders suggested in the PAS are employed, 50% of the bulimics fell within the dependent PAS cluster, whereas 40% of anorexics fell within the inhibited PAS cluster. The difference, however, did not reach statistical significance. Of the total number of diagnoses 37.1% ( $n = 13$ ) were from the inhibited PAS cluster, 31.4% ( $n = 11$ ) from the dependent PAS cluster, 20% ( $n = 7$ ) from the withdrawn PAS cluster, and 11.4% ( $n = 4$ ) in the antisocial PAS cluster.

**Table 4.B.2. Distribution of the PAS personality disorder clusters in ED subtypes**

PAS Personality Clusters	Eating Disorder Subtypes					
	All Patients( $n=35$ )		AN( $n=25$ )		BN( $n=10$ )	
	n	%	n	%	n	%
Antisocial	4	11.4	3	12	1	10
Dependent	11	31.4	6	24	5	50
Inhibited	13	37.1	10	40	3	30
Withdrawn	7	2.00	6	24	1	10

**ED= Eating Disorder; AN= Anorexia Nervosa; BN= Bulimia Nervosa; PAS= Personality Assessment Schedule**

Figure 4.B.2. Distribution of Anxious and Histrionic PDs in EDs Patients



**PDs= Personality Disorders**

**AN= Anprexia Nervosa**

**BN= Bulimia Nervosa**

**4.B.2. Clinical Feature of the Patients and Family Factors Associated with Personality Disorders**

**Patient Factors-** personality disorders were examined for their association with clinical features of the patients. Table 4.B.3. presents the significant findings of clinical factors. Prior to treatment (T1) patients who had no personality disorders scored significantly higher on average score on the Morgan-Russell scales ( $t= 2.39$ ,  $df= 44.96$ ,  $p < .02$ ) than did patients without personality disorders. The latter group showed significantly more interpersonal distrust ( $t= -2.16$ ,  $df= 35.76$ ,  $p < .04$ ), and interpersonal problem ( $t= -3.18$ ,  $df= 7.96$ ,  $p < .01$ ) than did those without personality disorders, as measured by EDI and IIP, respectively.

**Table 4.B.3. Clinical Features of the patients with and without personality disorders**

Variables	No Personality Disorder	Personality Disorder	P*
Age at Onset of ED	20.54(5.86)	18.20(5.10)	.13
Duration of ED	4.40(5.52)	7.24(6.39)	.08
BMI	15.94(1.46)	15.16(1.74)	.07
BDI	26.15(12.73)	29.66(12.01)	.44
MRAV	6.31(1.60)	5.16(1.59)	.02
Interpersonal Distrust	5.91(4.42)	9.93(5.40)	.04
Interpersonal Problem	1.13(.58)	1.95(.52)	.01

\* t-test for independent samples; BMI= Body Mass Index; BDI= Beck Depression Inventory; MRAV= Average Score of the Morgan-Russell Scales; M(s.d.)

Those who met criteria for at least one personality disorder at T1 had a younger age of onset of eating disorder, a longer duration of illness, and scored higher on BDI, and lower on BMI. These differences, however did not reach statistical significance.

**Family Factors-** Investigation was performed for the association between family factors as measured on EE scales and personality disorders (see Table 4.B.4.).

**Table 4.B.4. Distribution of family EE and personality diagnoses in eating disorder patients**

<b>EE Scales</b>	<b>No Personality Disorder</b>	<b>Personality Disorder</b>	<b>P*</b>
<b><u>Mothers(N= 38)</u></b>			
<b>CC</b>	<b>.78(1.31)</b>	<b>2.04(2.43)</b>	<b>.05</b>
<b>HOS</b>	<b>.00(.00)</b>	<b>.09(.42)</b>	<b>.32</b>
<b>EOI</b>	<b>1.64(1.21)</b>	<b>2.00(1.15)</b>	<b>.38</b>
<b>W</b>	<b>3.92(.61)</b>	<b>3.72(.98)</b>	<b>.45</b>
<b>PR</b>	<b>1.21(1.12)</b>	<b>1.40(1.33)</b>	<b>.64</b>
<b><u>Fathers(N= 26)</u></b>			
<b>CC</b>	<b>.90(1.44)</b>	<b>1.25(1.18)</b>	<b>.53</b>
<b>HOS</b>	<b>.00(.00)</b>	<b>.00(.00)</b>	<b>--</b>
<b>EOI</b>	<b>.60(.84)</b>	<b>.75(.85)</b>	<b>.66</b>
<b>W</b>	<b>3.50(.85)</b>	<b>3.37(.95)</b>	<b>.73</b>
<b>PR</b>	<b>.70(.67)</b>	<b>.93(.77)</b>	<b>.41</b>
<b><u>Husbands(N= 9)</u></b>			
<b>CC</b>	<b>2.80(1.78)</b>	<b>3.50(4.35)</b>	<b>.77</b>
<b>HOS</b>	<b>.00(.00)</b>	<b>.75(1.50)</b>	<b>.39</b>
<b>EOI</b>	<b>1.00(.70)</b>	<b>.50(1.00)</b>	<b>.43</b>
<b>W</b>	<b>3.40(.89)</b>	<b>2.25(1.50)</b>	<b>.23</b>
<b>PR</b>	<b>1.40(.89)</b>	<b>.00(.00)</b>	<b>.02</b>

**\* Tow-Tail t-test; EE= Expressed Emotion; CC= Critical Comments; HOS= Hostility; EOI= Emotional Over-Involvement; W= Warmth; PR= Positive Remarks; M(s.d.)**

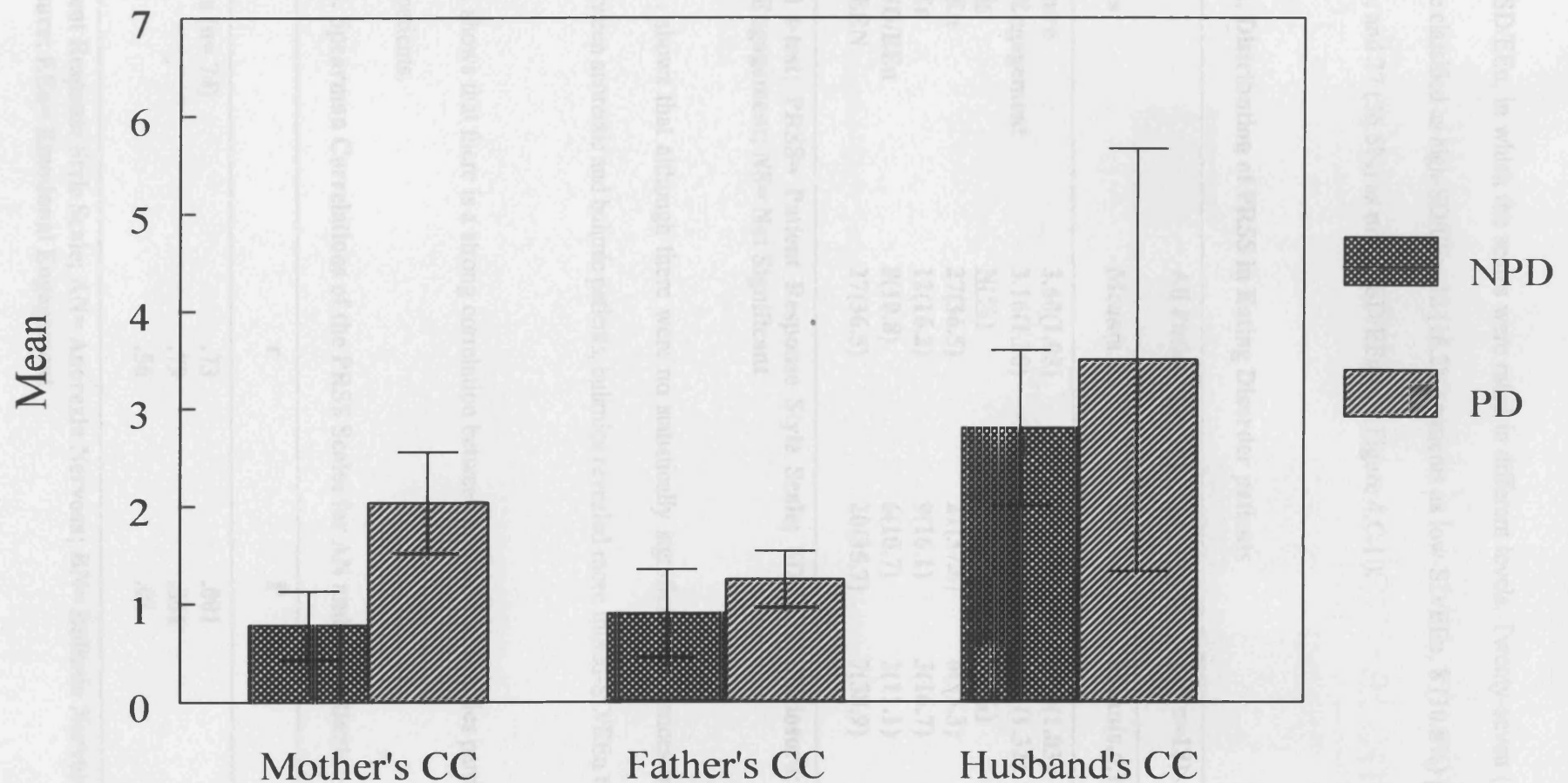
A comparison between relatives of personality disorder (PD) and non-personality disorder (NPD) groups at T1 revealed that mothers of PD patients were significantly more critical to the patients than mothers of NPD patients ( $t=-2.01$ ,  $df=33.31$ ,  $p < .05$ ). There were no significant differences among fathers and husbands of the two groups (see Figure 4.B.3.).

**4.C. PATIENT’S RESPONSE STYLE TO THERAPIST AND THERAPY**

**4.C.1. The Distribution of the PRSS**

The obtained measures of PRSS are summarized in Table 4.C.1. Four different patient’s response styles were designated: high-SD/EE<sub>n</sub>, in which both SD and EE<sub>n</sub> were high; low-SD/EE<sub>n</sub>, in which both SD and EE<sub>n</sub> were low; moderate-SD/EE<sub>n</sub>, in which both SD and EE<sub>n</sub> were moderate;

Figure 4.B.3. Distribution of Relatives' Critical Comments to NPD and PD Patients



NPD= No Personality Disorder; PD= Personality Disorder; CC= Critical Comments



and mixed-SD/EEEn, in which the scales were rated in different levels. Twenty-seven (36.5%) patients were classified as high-SD/EEEn, 12 (16.2%) patients as low-SD/EEEn, 8 (10.8%) patients as moderate, and 27 (36.5%) as mixed-SD/EEEn (see Figure 4.C.1.).

**Table 4.C.1. Distribution of PRSS in Eating Disorder patients**

	All Patients(n=74)	AN(n=56)	BN(n=18)	
PRSS Scales	Mean(s.d.)	Mean(s.d.)	Mean(s.d.)	P*
Self-Disclosure	3.60(1.03)	3.67(1.02)	3.38(1.03)	NS
Emotional Engagement	3.16(1.20)	3.17(1.17)	3.11(1.32)	NS
PRSS Levels	N(%)	N(%)	N(%)	
High-SD/EEEn	27(36.5)	21(37.5)	6(33.3)	NS
Low-SD/EEEn	12(16.2)	9(16.1)	3(16.7)	NS
Moderate-SD/EEEn	8(10.8)	6(10.7)	2(11.1)	NS
Mixed-SD/EEEn	27(36.5)	20(35.7)	7(38.9)	NS

\* Two-Tail t-test; PRSS= Patient Response Style Scale; SD= Self-Disclosure; EEEn= Emotional Engagement; NS= Not Significant

Table 4.C.1. shows that although there were no statistically significant differences on these measures between anorectic and bulimic patients, bulimics revealed more mixed-SD/EEEn than did anorexics.

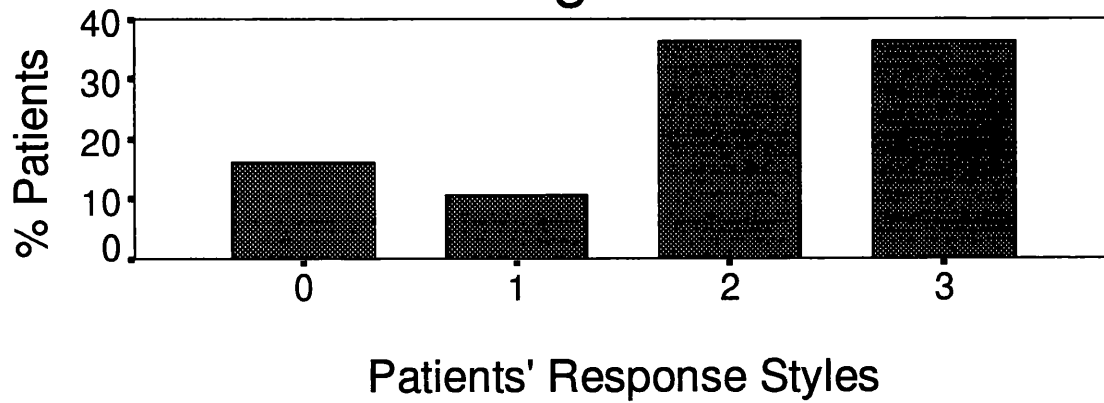
Table 4.C.2. shows that there is a strong correlation between the two PRSS scales particularly in anorexic patients.

**Table 4.C.2. Spearman Correlations of the PRSS Scales for AN and BN patients**

SD - EEEn	r	P
All Patients (n= 74)	.73	.001
AN (n= 56)	.79	.001
BN (n= 18)	.56	.01

PRSS= Patient Response Style Scale; AN= Anorexia Nervosa; BN= Bulimia Nervosa; SD= Self-Disclosure; EEEn= Emotional Engagement

Figure 4.C.1. Distribution of the PRSS in Eating Disorder Patients



**PRSS= Patient Response Style Scale**  
**0= Low-SD/EE**  
**1= Moderate-SD/EE**  
**2= High-SD/EE**  
**3= Mixed-SD/EE**

#### 4.C.2. Clinical Features of the Patient and Family Factors Associated with the PRSS

**Patient Factors-** The PRSS levels were examined for their association with clinical features of the patients. The ANOVA analyses revealed that BMI was significantly different for the four levels of the PRSS ( $F_{3,69} = 3.25, p = .02$ ). Comparison between different levels of the PRSS, using contrast, revealed significant difference only between High and low-SD/EEn groups ( $t = 2.25, df = 69.0, p = .02$ ), and between high- and mixed-SD/EEn ( $t = -2.72, df = 69.0, p = .008$ ). Patients who rated as high-SD/EEn had a significantly higher BMI than did patients with low-SD/EEn and mixed-SD/EEn (see Table 4.C.3.).

**Table 4.C.3. Distribution of Clinical Features of ED patients for the PRSS Levels**

Variables	PRSS				P*
	High	Low	Moderate	Mixed	
Age at Presentation	25.11(4.25)	27.00(9.04)	25.12(5.61)	27.37(7.67)	.59
Age at Onset of ED	17.85(4.26)	19.66(6.51)	20.15(3.94)	18.33(5.89)	.62
Duration of ED	6.31(5.52)	6.00(7.09)	5.00(5.39)	7.96(6.26)	.55
BMI	16.10(1.54)	14.87(1.90)	14.92(1.92)	14.92(1.31)	.02
BDI	22.68(13.29)	38.75(6.60)	27.20(12.57)	24.78(11.03)	.13
MRAV	6.12(1.34)	5.28(1.99)	5.20(1.83)	5.62(2.04)	.41

\* Oneway ANOVA; ED= Eating Disorder; PRSS= Patient Response Style Scale; SD= Self-Disclosure; EEn= Emotional Engagement; BMI= Body Mass Index; BDI= Beck Depression Inventory; MRAV= Average Score of the Morgan-Russell Scales

There were no statistically significant differences between the four groups on any of the following variables: age at presentation, age of eating disorder onset, duration of illness, BDI scores, average score of the Morgan-Russell scales, and EDI scores.

To explore the correlations of the PRSS scales, self-disclosure (SD) and emotional engagement (EEn), with clinical factors, Spearman correlational analyses were conducted. Findings revealed no significant association between the PRSS scales and any of the clinical factors mentioned

above. Again, although the PRSS scales were found to be significantly correlated with BMI, it was low (SD:  $r = .30$ ,  $p = .01$ ; EEn:  $r = .25$ ,  $p = .03$ ).

Further analysis was performed for the association between the PRSS scales and personality disorders (see Table 4.C.4.). Patients without personality disorder revealed more SD ( $M = 3.65$ ,  $s.d. = 1.11$ ) than did patients with personality disorder ( $M = 3.41$ ,  $s.d. = 0.98$ ). The former group also showed more EEn ( $M = 3.30$ ,  $s.d. = 1.29$ ) than did the latter group ( $M = 3.05$ ,  $s.d. = 1.17$ ). These differences, however, failed to reach statistical significance (SD:  $t = .84$ ,  $df = 43.50$ ,  $p < .40$ ; EEn:  $t = .73$ ,  $df = 44.32$ ,  $p < .47$ ).

**Table 4.C.4. Distribution of the PRSS Scales for the PAS Personality Abnormality**

PRSS Scales	No Personality Abnormality	Personality Abnormality	P*
SD	3.65(1.11)	3.41(.98)	.40
EEn	3.30(1.29)	3.05(1.17)	.47

\* t-test for independent samples; PRSS= Patient Response Style Scale; SD= Self-Disclosure; EEn= Emotional Engagement; PAS= Personality Assessment Schedule; M(s.d.)

**Family Factors-** Investigation was performed for the correlations between the PRSS scales and family EE ratings. As presented in Table 4.C., the only significant correlation was found between SD and warmth which was moderate for mothers ( $r = .40$ ,  $p = .006$ ).

**Table 4.C.5. Spearman Correlations of the PRSS Scales with Family EE Scales**

<b>EE Scales</b>	<b>SD(r/P)</b>	<b>EEn(r/P)</b>
<b><u>Mothers(N= 45)</u></b>		
<b>CC</b>	<b>-.25(.09)</b>	<b>-.15(.32)</b>
<b>HOS</b>	<b>.02(.84)</b>	<b>.11(.46)</b>
<b>EOI</b>	<b>-.10(.47)</b>	<b>-.06(.69)</b>
<b>W</b>	<b>.40(.006)</b>	<b>.24(.10)</b>
<b>PR</b>	<b>.10(.51)</b>	<b>.09(.54)</b>
<b><u>Fathers(N= 31)</u></b>		
<b>CC</b>	<b>.02(.91)</b>	<b>-.03(.84)</b>
<b>HOS</b>	<b>.25(.16)</b>	<b>.27(.13)</b>
<b>EOI</b>	<b>.32(.07)</b>	<b>.26(.15)</b>
<b>W</b>	<b>.11(.54)</b>	<b>.04(.81)</b>
<b>PR</b>	<b>.02(.90)</b>	<b>-.17(.34)</b>
<b><u>Husbands(N= 12)</u></b>		
<b>CC</b>	<b>.03(.92)</b>	<b>-.14(.64)</b>
<b>HOS</b>	<b>.00(.00)</b>	<b>.17(.57)</b>
<b>EOI</b>	<b>.24(.43)</b>	<b>-.04(.89)</b>
<b>W</b>	<b>.44(.15)</b>	<b>.39(.19)</b>
<b>PR</b>	<b>.22(.47)</b>	<b>-.02(.93)</b>

**PRSS= Patient Response Style Scale; AN= Anorexia Nervosa; BN= Bulimia Nervosa; SD= Self-Disclosure; EEn= Emotional Engagement; EE= Expressed Emotion; CC= Critical Comments; HOS= Hostility; EOI= Emotional Over-Involvement; W= Warmth; PR= Positive Remarks**

Next, to examine the association between the PRSS levels and family EE scales, further analyses were performed. The ANOVA analyses revealed that the mothers’ warmth and positive remarks ratings were significantly different for the four levels of the PRSS (W:  $F(3,41)= 5.32, p= .003$ ; PR:  $F(3,41)= 2.80, p= .05$ ). Comparison between different levels of the PRSS, using contrast, revealed significant difference only between High and low-SD/EEn groups ( $t= 2.51, df= 41.0, p= .01$ ) in terms of the mothers’ warmth scores. Mothers of patients with high-SD/EEn showed significantly more W towards the patients than did mothers of patients with low-SD/EEn (see Table 4.C.6.).

**Table 4.C.6. Distribution of Family EE Scales for the PRSS Levels**

EE Scales	PRSS Levels				
	High	Low	Moderate	Mixed	P*
<u>Mothers(N= 45)</u>					
CC	1.25(1.73)	2.25(2.76)	1.80(2.04)	1.56(1.99)	.72
HOS	.12(.34)	.25(.70)	.00(.00)	.00(.00)	.39
EOI	1.93(1.38)	2.12(.83)	1.80(1.48)	1.62(1.20)	.80
W	4.18(.54)	3.37(.91)	2.80(.83)	3.87(.80)	.003
PR	1.81(1.37)	1.62(.74)	.40(.54)	.93(1.18)	.05
<u>Fathers(N= 31)</u>					
CC	1.27(1.84)	1.00(1.09)	2.33(2.08)	1.18(1.07)	.63
HOS	.09(.30)	.00(.00)	.00(.00)	.00(.00)	.63
EOI	1.00(1.00)	.50(.83)	.33(.57)	.72(1.00)	.62
W	3.36(.80)	3.16(.98)	2.66(1.52)	3.81(.40)	.14
PR	.63(.67)	.83(.40)	.66(.57)	1.09(.94)	.53
<u>Husbands(N= 12)</u>					
CC	3.20(3.49)	2.00(2.64)	--	2.50(2.38)	.85
HOS	.60(1.34)	.00(.00)	--	.00(.00)	.54
EOI	.80(.83)	.66(.57)	--	1.00(.81)	.85
W	3.20(1.92)	3.00(1.00)	--	3.25(.50)	.97
PR	.80(.83)	.66(1.15)	--	1.00(1.15)	.91

\* Oneway ANOVA; PRSS= Patient Response Style Scales; EE= Expressed Emotion; CC= Critical Comments; HOS= Hostility; EOI= Emotional Over-Involvement; W= Warmth; PR= Positive Remarks

#### **4.D. SELF- AND OTHER-BLAME**

##### **4.D.1. The Distribution of the SOBS**

Table 4.D.1. contains descriptive statistics of the SOBS for eating disordered patients and their relatives. The mean scores of the SOBS suggest that mothers blamed themselves the most and blamed the patients the least.

**Table 4.D.1. Descriptive statistics of the SOBS among ED patients and their relatives**

<b>SOBS Scales</b>	<b>Mean (s.d.)</b>
<b><u>Self-Blame</u></b>	
<b>Mother(N= 39)</b>	<b>2.71(1.90)</b>
<b>Father(N= 29)</b>	<b>1.51(1.55)</b>
<b>Husband(N= 8)</b>	<b>2.50(1.19)</b>
<b>Patient(N= 49)</b>	<b>2.18(1.79)</b>
<b><u>Other-Blame</u></b>	
<b>Mother to Patient(N= 39)</b>	<b>0.42(1.01)</b>
<b>Father to Patient(N= 29)</b>	<b>0.62(1.47)</b>
<b>Husband to Patient(N= 8)</b>	<b>1.22(1.56)</b>
<b>Patient to Mother(N= 39)</b>	<b>1.05(1.70)</b>
<b>Patient to Father(N=29)</b>	<b>1.13(1.97)</b>
<b>Patient to Husband(N= 8)</b>	<b>0.55(1.66)</b>

**SOBS= Self- and Other-Blame Scales; ED= Eating Disorder**

More than 22% of relatives and 30.6% of patients reported no feelings of self-blame (SB). A majority of 71.4% of relatives and 76 % of patients reported no feelings of other-blame (OB). In general, the number of relatives who reported at least some levels of SB for patients’ eating problems was quite high. It was actually about the same as the number of patients reporting SB for their eating problems in the interview. Similarly, the proportion of relatives reporting any levels of OB was about the same as the proportion of patients reporting such feelings in the interview.

A t-test for paired samples comparing the SOBS measures revealed that self-blame scores were all greater than other-blame scores, and statistically significant both for parents and patients (all  $P_s < .01$ ) except SOBS scores of husbands and wives which did not reach levels of statistical significance.

Similar results were found when separate analyses were performed for families with an anorexic patient and families containing a bulimic patient.

A comparison between mothers and fathers showed mothers to be significantly more self-blaming than fathers ( $t = -5.08$ ,  $df = 29$ ,  $p < .001$ ). No statistically significant difference was found between mothers and fathers in terms of the other-blame scale. Figure 4.D.1. demonstrates the distribution of the parental self- and other-blaming attitudes in eating disorder patients.

A comparison of relatives' and patients' other-blame scores revealed that patients expressed significantly more other-blaming attitudes towards their mothers ( $t = -2.63$ ,  $df = 38$ ,  $p < .01$ ) and fathers ( $t = -2.10$ ,  $df = 28$ ,  $p < .04$ ) than did parents. No statistically significant difference was found between husbands and wives in terms of the other-blame scale.

The SOBS scores were examined in order to compare family self-blame and other-blame in anorexic and bulimic patients. The SOBS scores were slightly higher in families with a bulimic patient but the difference did not reach levels of statistical significance.

Bulimic patients were more likely to blame their parents specifically the mothers (AN:  $M = .79$ ,  $s.d. = 1.44$ ; BN:  $M = 1.80$ ,  $s.d. = 2.20$ ;  $t = -1.35$ ,  $df = 11.80$ ,  $P < .19$ ), whereas anorexic patients tended to blame themselves (AN:  $M = 2.28$ ,  $s.d. = 1.82$ ; BN:  $M = 1.81$ ,  $s.d. = 1.72$ ;  $t = .79$ ,  $df = 17.11$ ,  $p < .44$ ) rather than their parents. These differences, however were not statistically significant. The distribution of the SOBS scores for AN and BN patients is presented in Figure 4.D.2.

Findings concerning intercorrelational patterns of the SOBS scales showed no statistically significant correlation between self-blame (SB) and other-blame (OB) either for relatives or for patients (Table 4.D.2.). This indicates SB and OB as two separate scales measuring two different attitudes. Only a low significant positive correlation was found between patients' SB and patients



blaming mothers ( $r = .36, p = .02$ ).

**Table 4.D.2. Intercorrelations of the SOBS scales**

SOBS Scales	r*	p
<b>Mothers' SB - Mother Blaming Patient</b>	<b>-.02</b>	<b>.88</b>
<b>Fathers' SB - Father Blaming Patient</b>	<b>.25</b>	<b>.18</b>
<b>Husbands' SB - Husband Blaming Patient</b>	<b>-.05</b>	<b>.89</b>
<b>Patients' SB - Patient Blaming Mother</b>	<b>.36</b>	<b>.02</b>
<b>Patients' SB - Patient Blaming Father</b>	<b>-.07</b>	<b>.71</b>
<b>Patients' SB - Patient Blaming Husband</b>	<b>-.42</b>	<b>.25</b>

**\* Spearman Correlations; SOBS= Self- and Other-Blame Scale; SB= Self-Blame**

The examination of correlations between the SOBS scales for parental pairs revealed significant correlations for both SB and OB (see Table 4.D.3.).

**Table 4.D.3. Spearman correlations of parental SOBS scales**

SOBS Scales	r	p
<b>Mothers' SB - Father's SB</b>	<b>.63</b>	<b>.001</b>
<b>Mothers' OB - Father's OB</b>	<b>.51</b>	<b>.006</b>

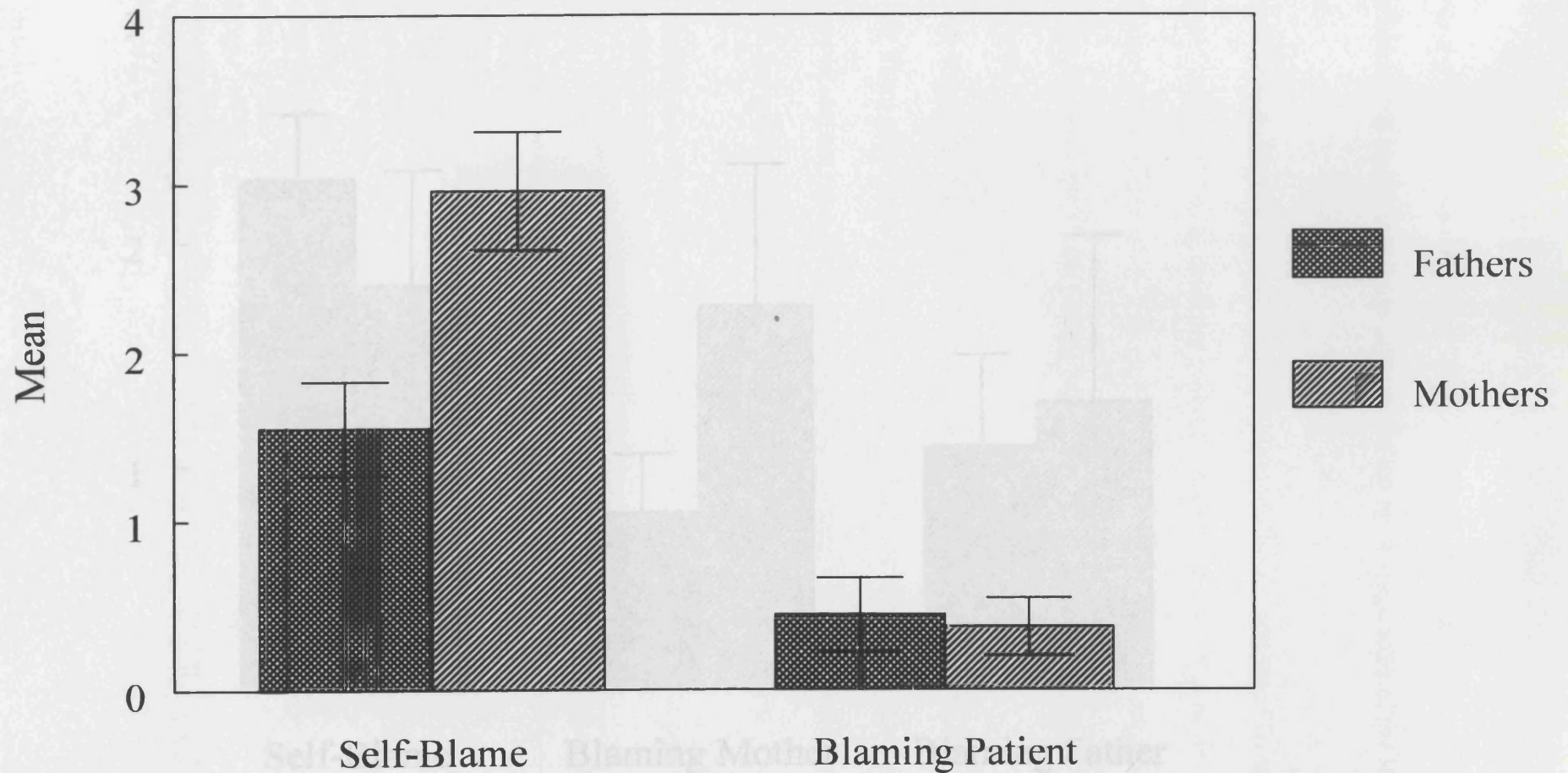
**SOBS= Self- and Other-Blame Scales; SB= Self-Blame; OB= Other-Blame**

**4.D.2. Clinical Features of the Patients and Family Factors Associated with the SOBS**

**Patient Factors-** Spearman correlations were performed to investigate the association of the SOBS measures with clinical features of the patients. The patients' blaming mother ratings revealed a significant negative correlation with their score on BMI ( $r = -.40, p = .01$ ) and with the age of onset of illness ( $r = -.30, p = .03$ ).

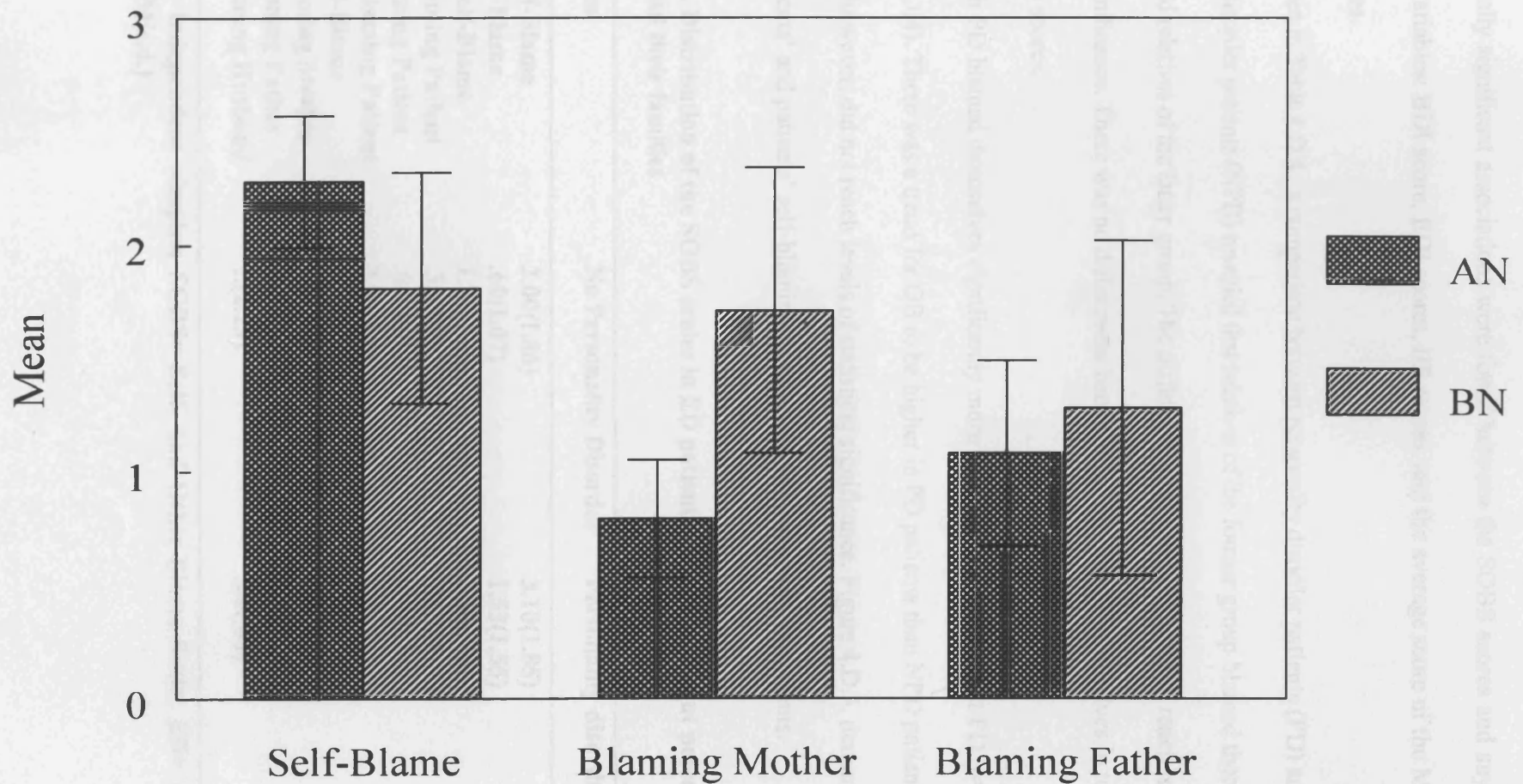
A low significant negative correlation was also found between mothers' self-blame and the patients' score on BMI ( $r = -.34, p = .02$ ).

Figure 4.D.1. Distribution of Parents' SOBS in Eating Disorder Patients



SOBS= Self- and Other-Blame Scales

Figure 4.D.2. Distribution of The SOBS  
for Eating Disorder Patients



SOBS= Self- and Other-Blame Scales; AN= Anorexia Nervosa; BN= Bulimia Nervosa

No statistically significant associations were found between the SOBS scores and any of the following variables: BDI score, EDI scores, IIP scores, and the average score of the Morgan-Russell scales.

As can be seen in Table 4.D.4., a comparison between personality disorder patients (PD) and non-personality disorder patients (NPD) revealed that relatives of the former group blamed themselves more than did relatives of the latter group. The difference, however, just failed to reach levels of statistical significance. There was no differences between the two groups of relatives in terms of other-blame scores.

Patients with PD blamed themselves significantly more than did patients without PD ( $t = -2.04$ ,  $df = 39$ ,  $p < .04$ ). There was a trend for OB to be higher in PD patients than NPD patients. The differences, however, did not reach levels of statistical significance. Figure 4.D.3. demonstrates both the parents' and patients' self-blaming attitudes for the two groups of patients.

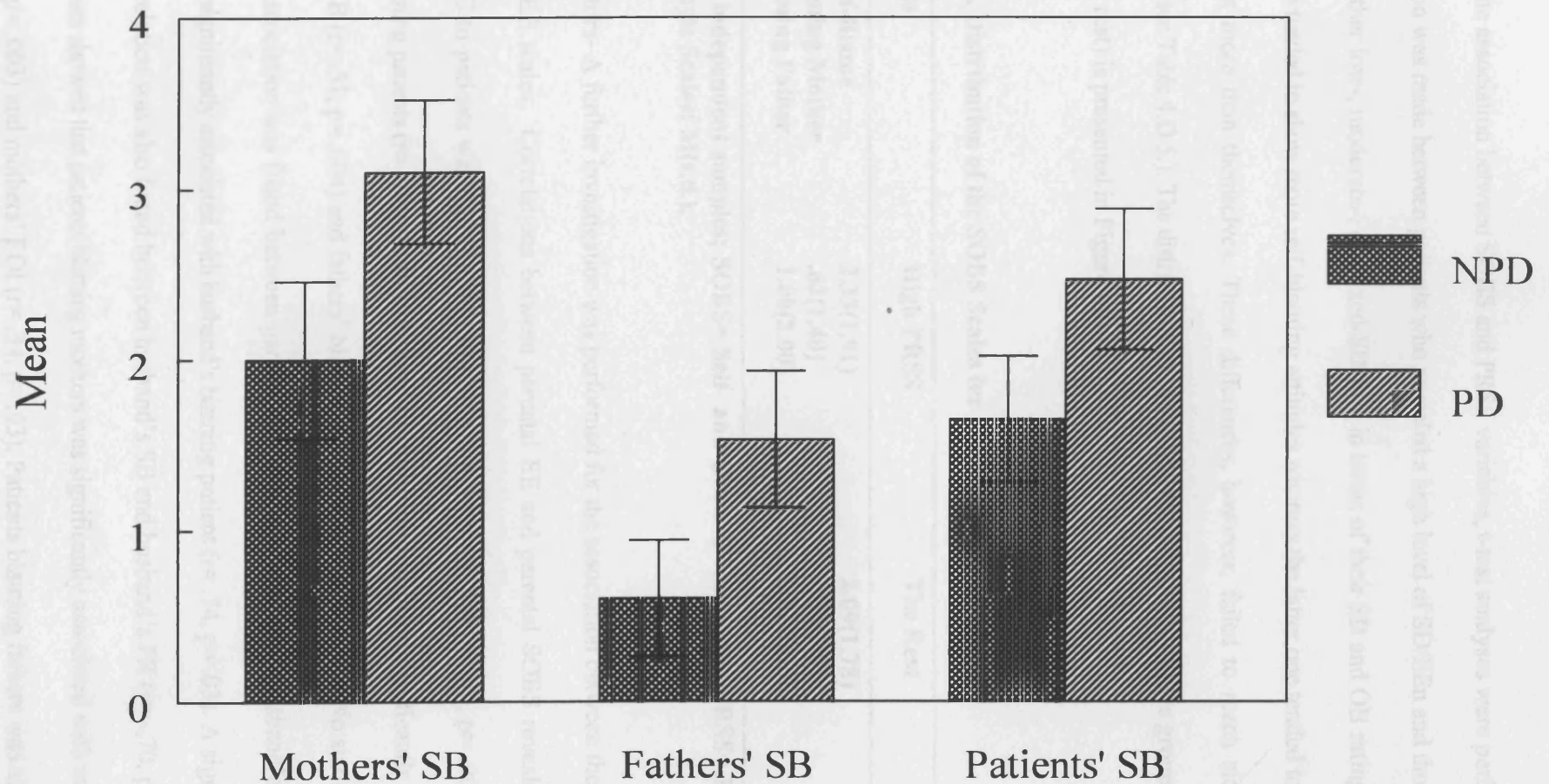
**Table 4.D.4. Distribution of the SOBS scales in ED patients with and without personality disorders and their families**

SOBS Scales	No Personality Disorder	Personality disorder	P*
Mother Self-Blame	2.00(1.86)	3.10(1.85)	.08
Father Self-Blame	.60(1.07)	1.53(1.55)	.08
Husband Self-Blame	1.75(1.25)	3.50(.70)	.10
Mother Blaming Patient	.35(.99)	.31(.74)	.90
Father Blaming Patient	.00(.00)	.80(1.74)	.09
Husband Blaming Patient	2.00(1.63)	1.50(2.12)	.80
Patient Self-Blame	1.37(1.58)	2.57(1.81)	.04
Patient Blaming Mother	.33(.90)	1.16(1.82)	.10
Patient Blaming Father	.60(1.57)	1.33(2.09)	.32
Patient Blaming Husband	1.(2.23)	.00(.00)	.37

\* t-test for independent samples; SOBS= Self- and Other-Blame Scale; ED= Eating Disorders; M(s.d.)



Figure 4.D.3. Distribution of the SOBS  
for NPD and PD Patients



SOBS= Self- and Other-Blame Scales; NPD= No Personality Disorder; PD= Personality Disorder; SB= Self-Blame

To explore the association between SOBS and PRSS variables, t-test analyses were performed. A comparison was made between patients who revealed a high level of SD/EE<sub>n</sub> and those who presented either low-, moderate-, or mixed-SD/EE<sub>n</sub> in terms of their SB and OB ratings. The former group tended to show more self-blaming attitudes whereas the latter one tended to blame their parents more than themselves. These differences, however, failed to reach statistical significance (see Table 4.D.5.). The distribution of the SOBS for the two respective groups (high PRSS vs the rest) is presented in Figure 4.D.4.

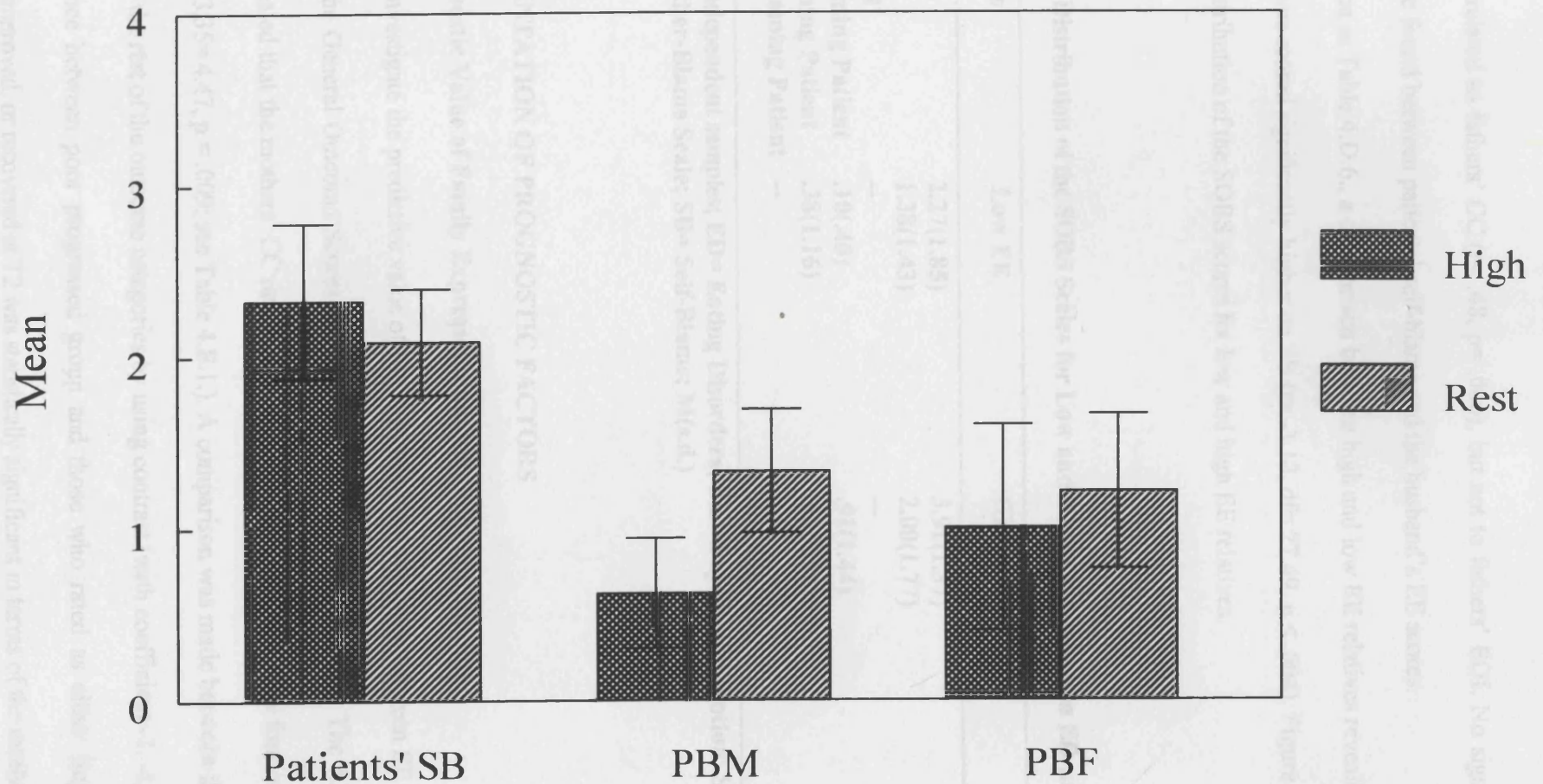
**Table 4.D.5. Distribution of the SOBS Scales for the PRSS Levels**

<b>SOBS Scales</b>	<b>High PRSS</b>	<b>The Rest</b>	<b>P*</b>
<b>Patient' Self-Blame</b>	<b>2.33(1.91)</b>	<b>2.09(1.75)</b>	<b>.67</b>
<b>Patient Blaming Mother</b>	<b>.62(1.40)</b>	<b>1.33(1.81)</b>	<b>.17</b>
<b>Patient Blaming Father</b>	<b>1.00(2.00)</b>	<b>1.21(1.96)</b>	<b>.78</b>

\* t-test for independent samples; SOBS= Self- and Other-Blame Scale; PRSS= Patient Response Style Scales; M(s.d.).

**Family Factors-** A further investigation was performed for the association between the SOBS and family EE scales. Correlations between parental EE and parental SOBS revealed that mothers' CC to patients was significantly associated with mothers' SB ( $r = .45$ ,  $p = .008$ ) and mothers' blaming patients ( $r = .48$ ,  $p = .002$ ). Fathers' CC to patients was also significantly related to fathers' SB ( $r = .51$ ,  $p = .004$ ) and fathers' blaming patients ( $r = .52$ ,  $p = .003$ ). No statistically significant association was found between parental SOBS and EOI ratings. Husband's CC to patients was significantly associated with husband's blaming patient ( $r = .74$ ,  $p = .03$ ). A significant negative correlation was also found between husband's SB and husband's PR ( $r = -.70$ ,  $p = .05$ ). Similar analyses showed that patients blaming mothers was significantly associated with mothers' CC ( $r = .43$ ,  $p = .003$ ) and mothers' EOI ( $r = .34$ ,  $p = .03$ ). Patients blaming fathers was also

Figure 4.D.4. Distribution of the SOBS for the PRSS Levels (High vs Rest)



SOBS= Self- and Other-Blame Scales; PRSS= Patient Response Style Scales; Rest= Low-, Moderate-, and Mixed-PRSS; PBM= Patient Blaming Mother; BPF= Patient Blaming Father

significantly related to fathers' CC ( $r = .48$ ,  $p = .006$ ), but not to fathers' EOI. No significant relations were found between patients' self-blame and the husband's EE scores.

As can be seen in Table 4.D.6., a comparison between high and low EE relatives revealed that high EE mothers scored significantly higher on SB ( $t = -3.12$ ,  $df = 27.49$ ,  $p < .004$ ). Figure 4.D.5. shows the distribution of the SOBS scores for low and high EE relatives.

**Table 4.D.6. Distribution of the SOBS Scales for Low and High EE families in ED patients**

SOBS Scales	Low EE	High EE	P*
Mother SB	2.27(1.85)	3.91(1.37)	.004
Father SB	1.38(1.43)	2.00(1.77)	.39
Husband SB	--	--	--
Mother Blaming Patient	.10(.40)	.91(1.44)	.07
Father Blaming Patient	.38(1.16)	.62(1.40)	.67
Husband Blaming Patient	--	--	--

\* t-test for independent samples; ED= Eating Disorders; EE= Expressed Emotion; SOBS= Self- and Other-Blame Scale; SB= Self-Blame; M(s.d.)

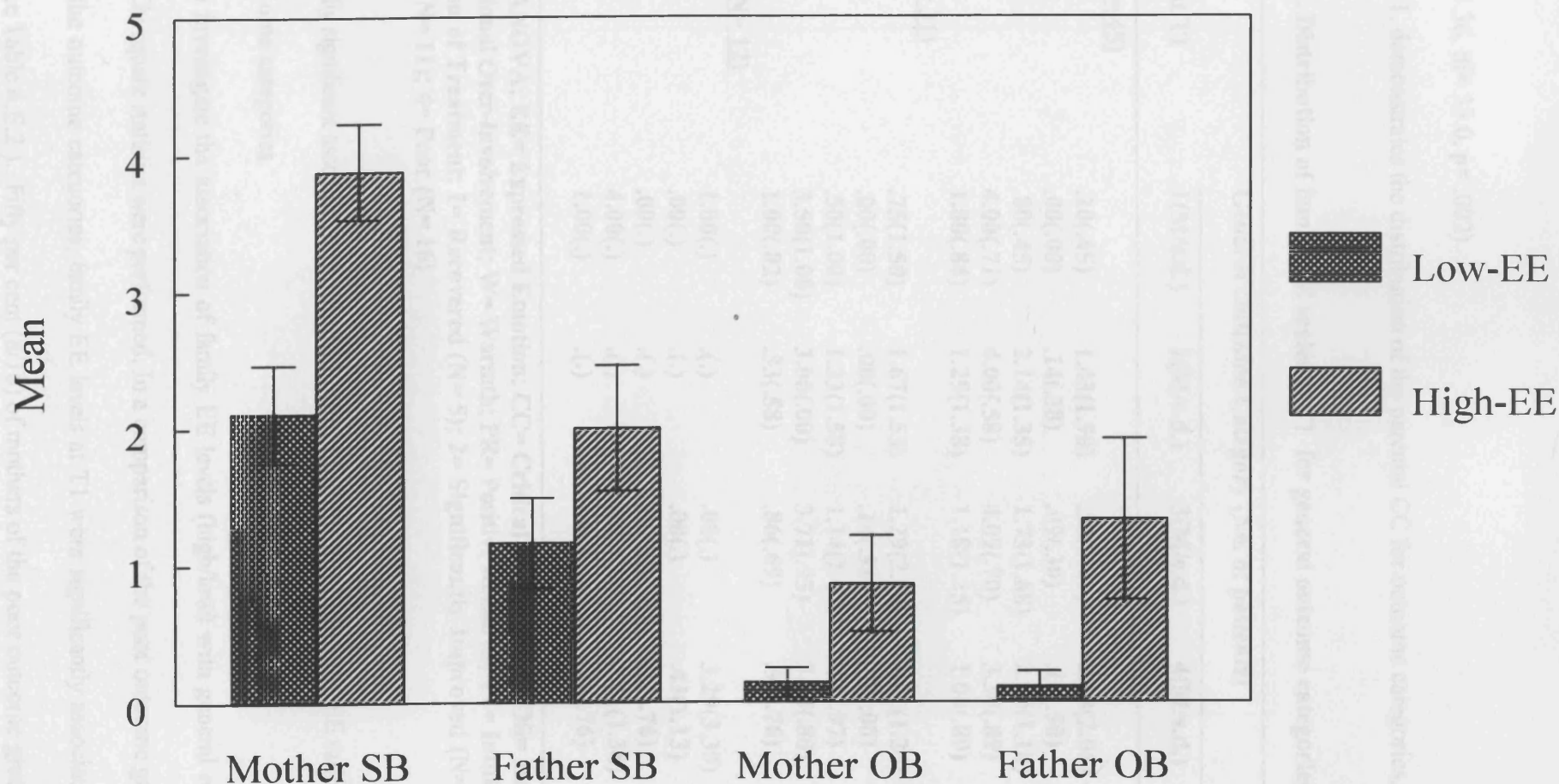
## 4.E. PRESENTATION OF PROGNOSTIC FACTORS

### 4.E.1. Prognostic Value of Family Expressed Emotion

In order to investigate the predictive value of EE, a comparison was made between EE ratings at T1 and the General Outcome Scores (GOS; Morgan-Russell, 1975) at T2. The ANOVA analysis revealed that the mothers' CC ratings were significantly different for the four outcome categories ( $F_{3,35} = 4.47$ ,  $p = .009$ ; see Table 4.E.1.). A comparison was made between the poor outcome and the rest of the outcome categories by using contrast (with coefficient -1, -1, -1, 3). The difference between poor progressed group and those who rated as either improved, significantly improved, or recovered at T2 was statistically significant in terms of the mothers' CC



Figure 4.D.5. Distribution of the SOBS  
for Low/High EE Parents of ED Patients



SOBS= Self- and Other-Blame Scales; EE= Expressed Emotion; ED= Eating Disorder; SB= Self-Blame; OB= Other-Blame

ratings ( $t= 3.36$ ,  $df= 35.0$ ,  $p= .002$ ).

Figures 4.E.1. demonstrates the distribution of the parental CC for outcome categories.

**Table 4.E.1. Distribution of family EE scales at T1 for general outcome categories at T2**

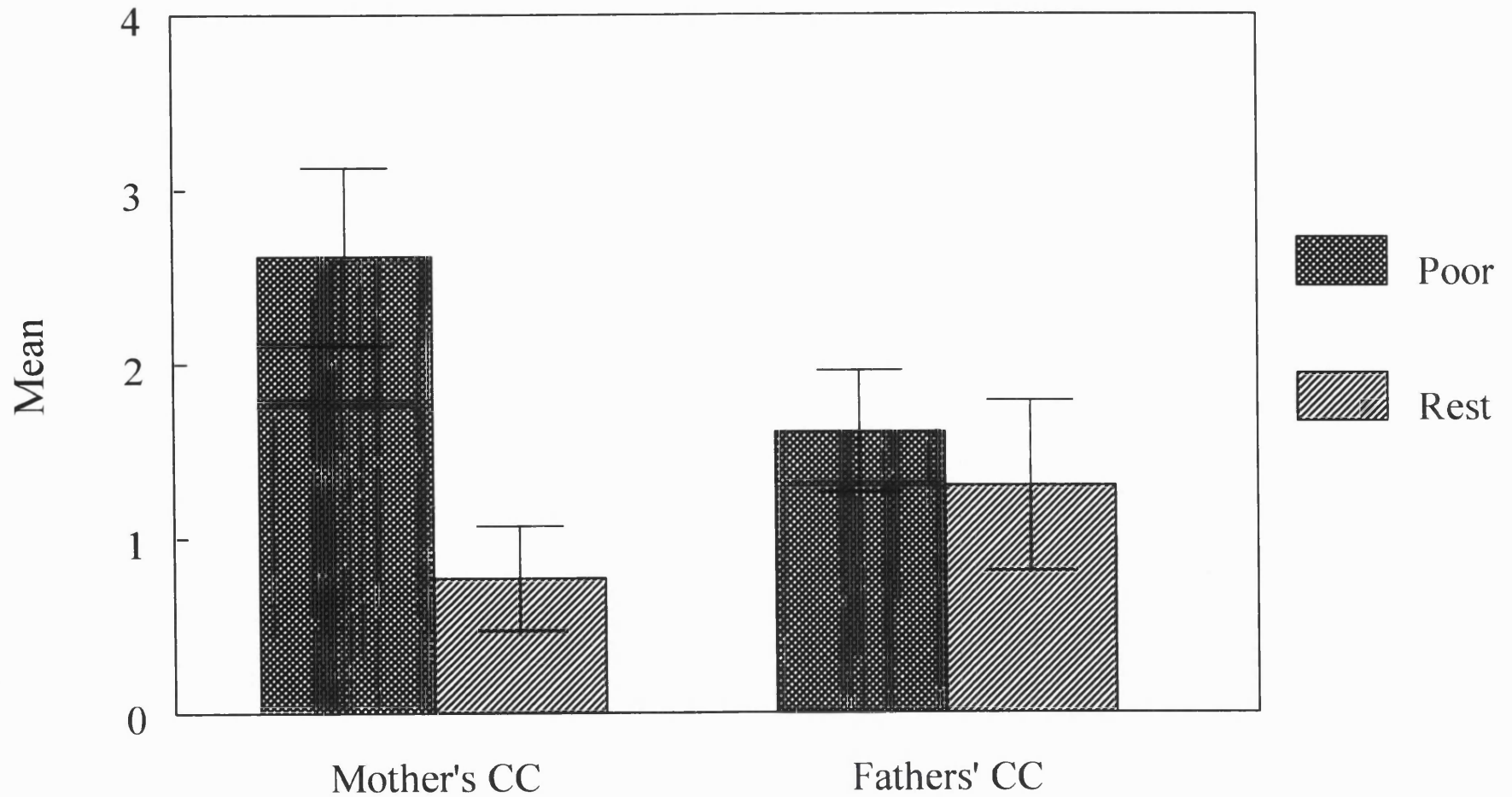
	General Outcome Category (No. of patients)				
EE Scales at T1	1(M/s.d.)	2(M/s.d.)	3(M/s.d.)	4(M/s.d.)	P*
<u>Mothers(N= 45)</u>					
CC	.20(.45)	1.43(1.90)	.55(1.21)	2.63(2.06)	.009
HOS	.00(.00)	.14(.38)	.09(.30)	.13(.50)	.92
EOI	.80(.45)	2.14(1.35)	1.73(1.68)	2.00(1.10)	.28
W	4.00(.71)	4.00(.58)	4.09(.70)	3.38(.89)	.08
PR	1.80(.84)	1.29(1.38)	1.18(1.25)	1.00(.89)	.56
<u>Fathers(N= 31)</u>					
CC	.75(1.50)	1.67(1.53)	1.29(2.14)	1.62(1.26)	.79
HOS	.00(.00)	.00(.00)	.14(.38)	.00(.00)	.43
EOI	.50(1.00)	1.33(1.58)	1.14(1.07)	.54(.97)	.39
W	3.50(1.00)	3.00(.00)	3.71(.95)	3.15(.80)	.45
PR	1.00(.82)	.33(.58)	.86(.69)	.92(.76)	.62
<u>Husbands(N= 12)</u>					
CC	1.00(.)	.(.)	.00(.)	3.29(3.30)	.59
HOS	.00(.)	.(.)	.00(.)	.43(1.13)	.89
EOI	.00(.)	.(.)	1.00(.)	.71(.76)	.62
W	4.00(.)	.(.)	5.00(.)	2.71(1.38)	.30
PR	1.00(.)	.(.)	1.00(.)	.29(.76)	.53

\* Oneway ANOVA; EE= Expressed Emotion; CC= Critical Comments; HOS= Hostility; EOI= Emotional Over-Involvement; W= Warmth; PR= Positive Remarks; T1= Intake; T2= Termination of Treatment; 1= Recovered (N= 5); 2= Significantly Improved (N= 7); 3= Improved (N= 11); 4= Poor (N= 16)

No statistically significant association was found between fathers' and husbands' EE ratings with general outcome categories.

Further , to investigate the association of family EE levels (high/low) with general outcome categories, Chi-square statistics were performed. In a comparison of the poor outcome group vs. the rest of the outcome categories, family EE levels at T1 were significantly associated with outcome (see Table 4.E.2.). Fifty per cent (8/16) of mothers of the poor outcome group were

Figure 4.E.1. Distribution of Parents' CC for Outcome Categories (Poor vs Rest)



CC= Critical Comments; Outcome= Poor vs Rest (Recovered, Significantly Improved, Improved)

rated as high EE at T1 whereas 13% (3/23) of mothers of the rest of the treatment outcome were rated as high at T1; a statistically significant difference (Fisher's Exact  $p = .02$ ). No significant association was found between fathers' and husbands' EE levels with general outcome categories.

**Table 4.E.2. Distribution of family EE levels at T1 for general outcome categories at T2**

EE Levels at T1	General Outcome Category (No. of patients)			
	Recovered	Sign. Improved	Improved	Poor
<b><u>Mothers(N= 45)</u></b>				
Low EE	5	5	10	8
High EE	0	2	1	8
Two-Tail Fisher's Exact (Poor vs Rest) $P = .02$				
<b><u>Fathers(N= 31)</u></b>				
Low EE	3	1	6	9
High EE	1	2	1	4
Two-Tail Fisher's Exact (Poor vs Rest) $P = 1.00$				
<b><u>Husbands(N= 12)</u></b>				
Low EE	1	0	1	3
High EE	0	0	3	4
Two-Tail Fisher's Exact (Poor vs Rest) $P = .44$				

EE= Expressed Emotion; T1= Intake; T2= Termination of Treatment

Next, to examine the relationship between the relatives' CC rating, type of therapy and treatment outcome, similar analyses were performed. No significant relationships were found between relatives' CC and outcome categories for any of the therapy types, when the poor outcome group was compared to the rest of the outcome categories.

#### **4.E.2. Prognostic Value of Personality Disorders**

In the first analysis concerning the association of personality disorder and progress in treatment as measured by the GOS, a comparison was made between the personality disorder (PD) group and non-personality disorder (NPD) group. Overall, the NPD group showed significantly more

improvement than did the PD group (Two-Tail Fisher's Exact  $p = .006$ ).

**Table 4.E.3. Distribution of the PAS personality diagnoses for general outcome categories**

PAS Personality Diagnoses	General Outcome Category (No. of patients)			
	Recovered	Sign. Improved	Improved	Poor
No Personality Disorder	5	5	3	8
Personality Disorder	1	1	11	17
Two-Tail Fisher's Exact $p = .006$				

**PAS= Personality Assessment Schedule**

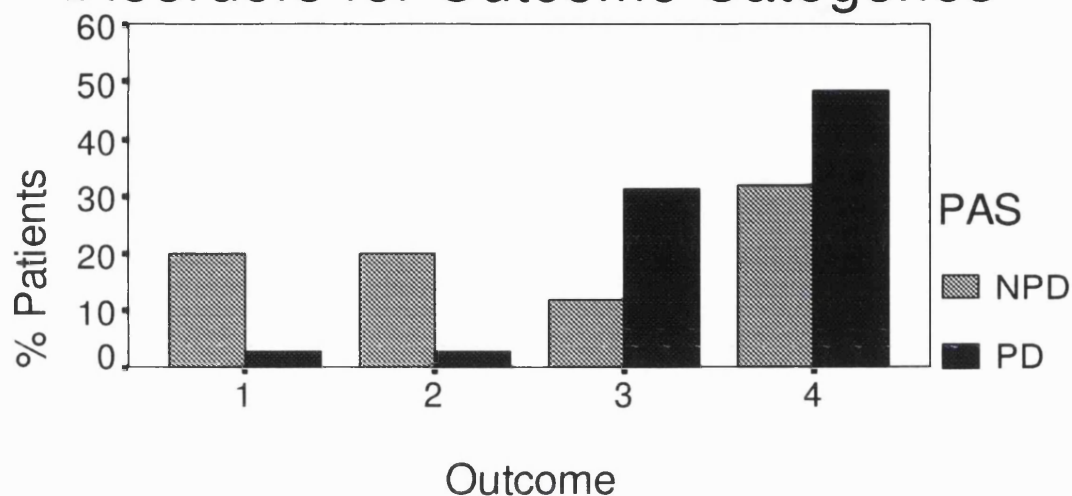
To examine the relationship between personality diagnoses, type of therapy and treatment outcome, similar analyses were performed. No significant relationships were found between personality diagnoses and outcome categories for any of the therapy types, when the poor outcome group was compared to the rest of the outcome categories. Figure 4.E.2. shows the distribution of the PAS personality disorders for outcome categories.

#### **4.E.3. Prognostic Value of the PRSS**

To investigate the prognostic value of the PRSS, associations were performed between the PRSS scales at T1 and general outcome categories at T2. Table 4.E.4. presents the distribution of the PRSS scales at T1 for general outcome categories. The ANOVA analyses revealed that the means of SD and EEn ratings were significantly different for the four outcome categories (SD:  $F(3,56) = 7.67$ ,  $p = .0002$ ; EEn:  $F(3,56) = 5.91$ ,  $p = .001$ ). A comparison was made between the poor outcome and the rest of the outcome categories by using contrast (with coefficient -1, -1, -1, 3). The difference between poor outcome group and those who were rated as either improved, significantly improved, or recovered at the end of therapy was statistically significant in terms of

**Figure 4.E.2. Distribution of the PAS Personality Disorders for Outcome Categories**

## 1. Distribution of the PAS Personality Disorders for Outcome Categories



**PAS= Personality Assessment Schedule; NPD= No Personality Disorder; PD= Personality Disorder; 1= Recovered; 2= Significantly Improved; 3= Improved; 4= Poor**

both SD ( $t = -4.49$ ,  $df = 56.0$ ,  $p < .001$ ) and EEn ( $t = -4.02$ ,  $df = 56.0$ ,  $p < .001$ ). A higher rating of the patients' SD and EEn was associated with better progress in therapy at T2. Figure 4.E.3.1. demonstrates the PRSS scales for outcome categories.

**Table 4.E.4. Distribution of the PRSS Scales at T1 for General Outcome Categories at T2**

PRSS Scales	General Outcome Category				
	Recovered	Sign. Improved	Improved	Poor	P*
<b>SD</b>	<b>4.00(1.26)</b>	<b>4.71(.49)</b>	<b>3.87(.83)</b>	<b>3.09(.93)</b>	<b>.001</b>
<b>EEn</b>	<b>3.67(1.37)</b>	<b>4.29(.49)</b>	<b>3.60(1.18)</b>	<b>2.66(1.10)</b>	<b>.001</b>

\* Oneway ANOVA; PRSS= Patient Response Style Scale; SD= Self-Disclosure; EEn= Emotional Engagement; M(s.d.)

Correlations were also computed between the PRSS levels at T1 and general outcome categories at T2 (see Table 4.E.5.). A majority of 81.8% (18/22) of the high-SD/EEn patients were rated as being either improved, significantly improved, or recovered whereas only 42.8% (3/7) of the moderate-SD/EEn patients, 10% (1/10) of the low-SD/EEn patients, and 28.6% (6/21) of the mixed-SD/EEn patients did so; a statistically significant difference (Fisher's Exact  $p = .0004$ )

Patients who showed a high level of SD/EEn to therapist and therapy did better in psychotherapy than those who showed either low-, mixed-, or moderate-SD/EEn. Figure 4.E.3.2. presents the PRSS levels for outcome categories.

**Table 4.E.5. Distribution of the PRSS Levels at T1 for General Outcome Categories at T2**

<b>PRSS Levels</b>	<b>General Outcome Category (No. Of patients)</b>			
	<b>Recovered</b>	<b>Sign. Improved</b>	<b>Improved</b>	<b>Poor</b>
<b>High-SD/EEen</b>	<b>4</b>	<b>7</b>	<b>7</b>	<b>4</b>
<b>Low-SD/EEen</b>	<b>1</b>	<b>0</b>	<b>0</b>	<b>9</b>
<b>Moderate-SD/EEen</b>	<b>0</b>	<b>0</b>	<b>3</b>	<b>4</b>
<b>Mixed-SD/EEen</b>	<b>1</b>	<b>0</b>	<b>5</b>	<b>15</b>
<b>Two-Tail Fisher's Exact (Poor vs Rest) P = .0004</b>				

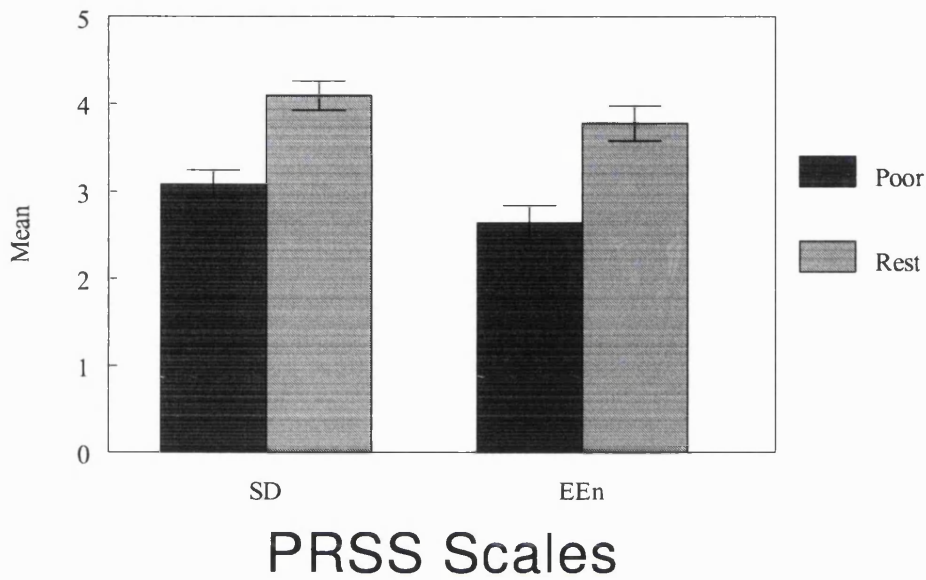
**PRSS= Patient Response Style Scale; SD= Self-Disclosure; EEen= Emotional Engagement**

Next, to investigate the relationship between PRSS, type of therapy and treatment outcome, similar analyses were performed. The PRSS was found to be significantly associated with general outcome categories for focal analytic psychotherapy ( $p < .009$ ), but not for the other therapy types, when comparing the poor outcome to the other outcome categories. High-SD/EEen at T1 were related to progress in focal analytic therapy at T2.

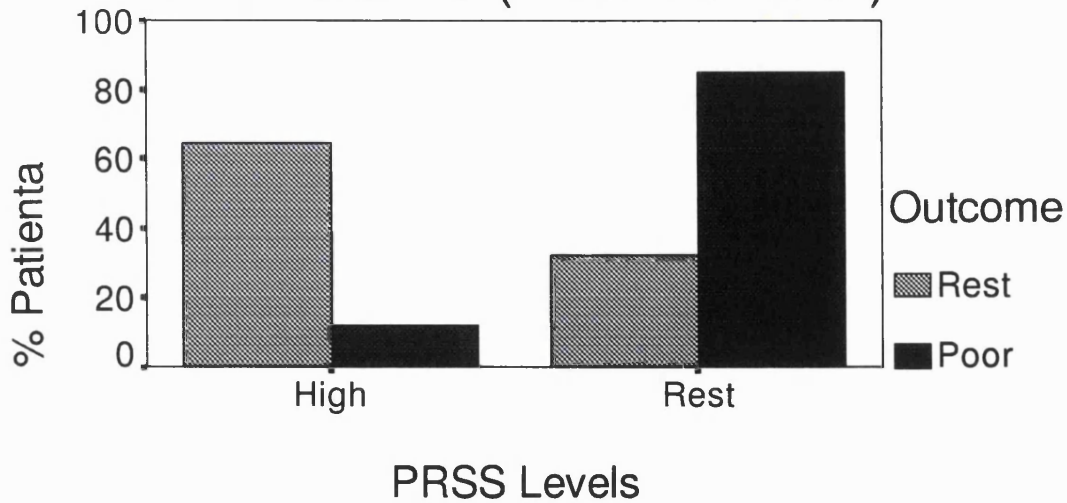


**Figure 4.E.3. Distribution of the PRSS Variables for Outcome Categories**

**1. Distribution of The PRSS Scales  
for Outcome (Poor vs Rest)**



**2. Distribution of the PRSS Levels  
for Outcome (Poor vs Rest)**



**PRSS= Patient Response Style Scale; SD= Self-Disclosure; EEn= Emotional Engagement; Outcome= Poor vs Rest (Recovered, Significantly Improved, Improved)  
PRSS Levels= High vs Rest (Low, Moderate, Mixed)**

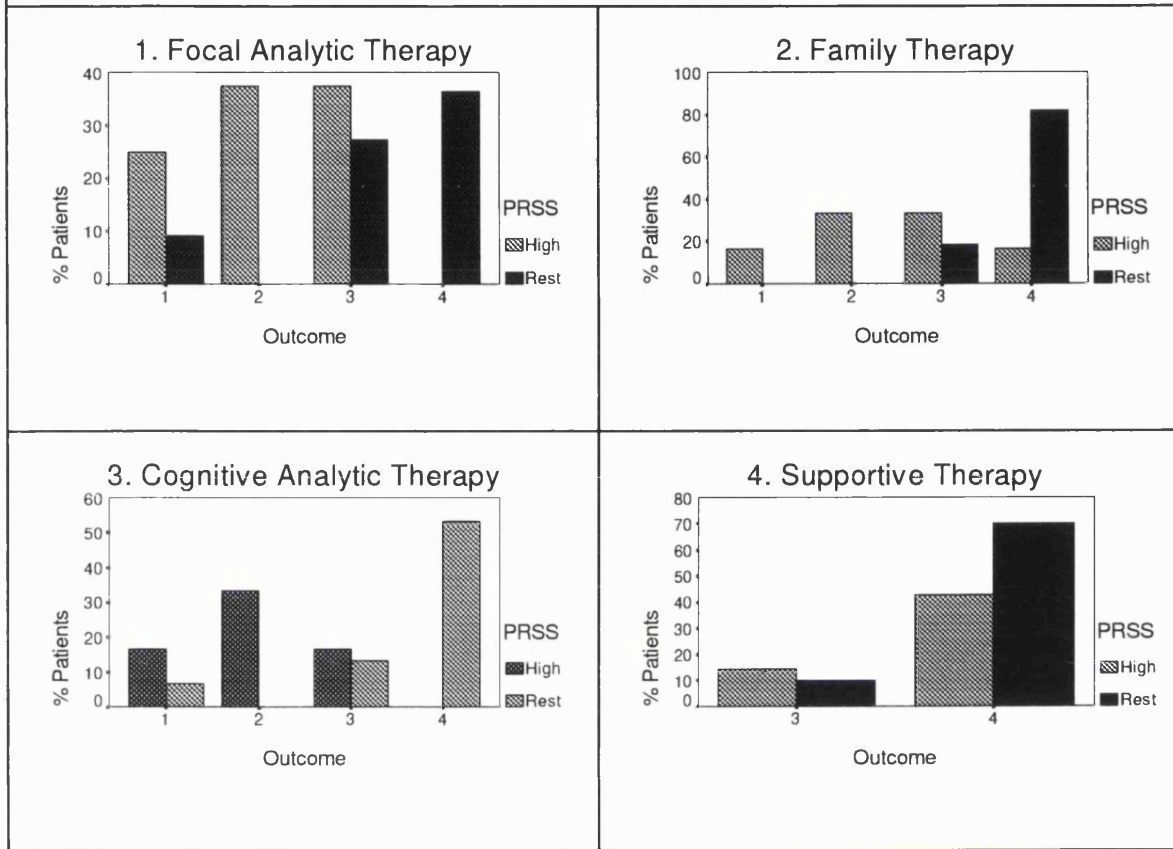
A comparison was performed between high-SD/EEEn levels and the other levels including low-, moderate, and mixed-SD/EEEn. High level of PRSS were found to be significantly related to progress in different psychotherapies but not the control group (see Table 4.E.6.). Figure 4.E.4. demonstrates the PRSS levels for the outcome categories of different types of therapy.

**Table 4.E.6. Distribution of the PRSS and therapy types for general outcome categories**

Therapy	Outcome Category (Poor vs Rest)			
	Recovered	Sign. Improved	Improved	Poor
<b><u>Focal</u></b>				
High-SD/EEEn	2	3	3	0
Low-SD/EEEn	0	0	0	2
Moderate-SD/EEEn	1	0	0	0
Mixed-SD/EEEn	0	0	2	2
Two-Tail Fisher's Exact (High vs Rest) P = .025				
<b><u>Family</u></b>				
High-SD/EEEn	1	2	2	1
Low-SD/EEEn	0	0	0	2
Moderate-SD/EEEn	0	0	1	2
Mixed-SD/EEEn	0	0	1	5
Two-Tail Fisher's Exact (High vs Rest) P = .034				
<b><u>Cognitive</u></b>				
High-SD/EEEn	1	2	1	0
Low-SD/EEEn	1	0	0	4
Moderate-SD/EEEn	0	0	1	1
Mixed-SD/EEEn	0	0	1	3
Two-Tail Fisher's Exact (High vs Rest) P = .025				
<b><u>Supportive</u></b>				
High-SD/EEEn	0	0	1	3
Low-SD/EEEn	0	0	0	1
Moderate-SD/EEEn	0	0	0	1
Mixed-SD/EEEn	0	0	1	5
Two-Tail Fisher's Exact (High vs Rest) P = 1.00				

PRSS= Patient Response Style Scale; SD= Self-Disclosure; EEn= Emotional Engagement

**Figure 4.E.4. Distribution of the PRSS levels for Outcome Categories by Different Types of Therapy**



**PRSS= Patient Response Style Scale; PRSS Levels= High vs Rest (Low, Moderate, Mixed); 1= Recovered; 2= Significantly Improved; 3= Improved; 4= Poor**

#### 4.E.4. Prognostic Value of the SOBS

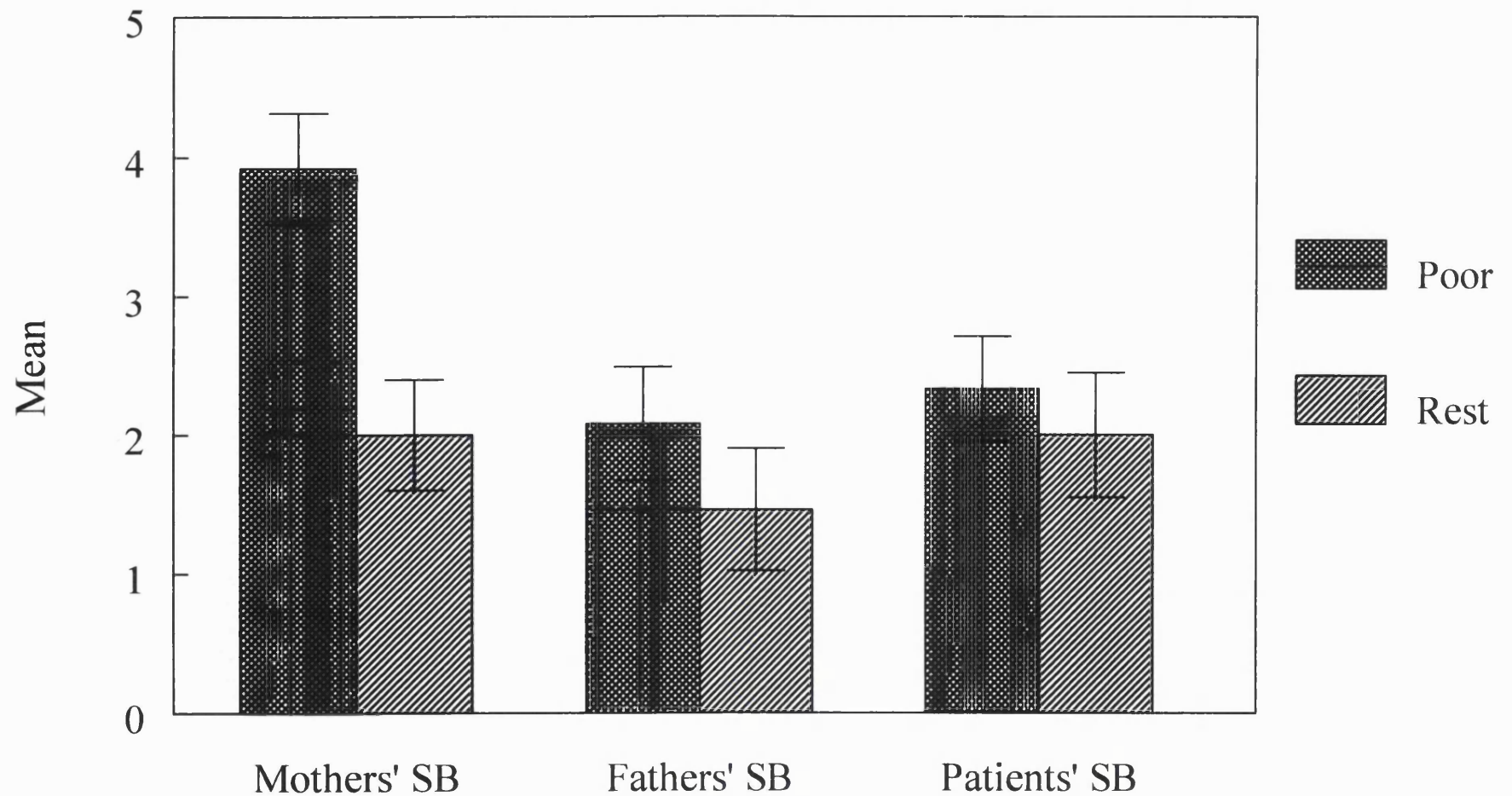
In order to investigate the SOBS predictive value, a comparison was performed between the SOBS measures at T1 and the general outcome categories at T2. The ANOVA analyses revealed that the mean of the mother's SB ratings was significantly different for the four outcome categories ( $F_{3,31} = 6.37, p = .001$ ). A comparison was made between the poor outcome and the rest of the outcome categories by using contrast (with coefficient -1, -1, -1, 3). The difference between poor outcome group and those who were rated as either improved, significantly improved, or recovered at the end of therapy was statistically significant in terms of mothers' self-blame ( $t = 3.60, df = 31.0, p = .001$ ), and patient blaming mother ( $t = 2.42, df = 30.0, p = .02$ ). Poor progress in therapy was related to both mothers' self blaming attitudes and patients blaming mother (Table 4.E.7.). The SB scores for outcome groups are presented in Figure 4.E.5.

**Table 4.E.7. Distribution of the SOBS Measures at T1 for General Outcome Categories at T2**

SOBS Measures	General Outcome Categories				P*
	1(M/s.d.)	2(M/s.d.)	3(M/s.d.)	4(M/s.d.)	
<b>Mother SB</b>	.80(.84)	2.14(1.57)	1.80(1.93)	3.92(1.44)	.001
<b>Father SB</b>	.50(1.00)	2.33(1.15)	1.43(1.90)	2.08(1.44)	.28
<b>Husband SB</b>	2.00(--)	--(--)	--(--)	3.25(.50)	.11
<b>Mother Blaming Patient</b>	.40(.89)	.43(1.13)	.40(1.26)	.43(.94)	.99
<b>Father Blaming Patient</b>	.25(.50)	1.67(2.89)	.57(1.51)	.67(1.50)	.69
<b>Husband Blaming Patient</b>	2.00(--)	--(--)	--(--)	1.75(2.06)	.92
<b>Patient SB</b>	1.17(1.60)	1.67(1.37)	2.90(2.51)	2.33(1.64)	.29
<b>Patient Blaming Mother</b>	.00(.00)	.57(1.13)	.50(1.58)	1.75(2.01)	.13
<b>Patient Blaming Father</b>	.00(.00)	2.00(2.65)	.71(1.89)	1.75(2.26)	.39
<b>Patient Blaming Husband</b>	.00(--)	--(--)	--(--)	1.00(2.24)	.70

\* Oneway ANOVA; SOBS= Self- and Other-Blame Scale; SB= Self Blame; 1= Recovered; 2= Significantly Improved; 3= Improved; 4= Poor

Figure 4.E.5. Distribution of Self-Blame  
for Outcome (Poor vs Rest)



SB= Self-Blame; Outcome= Poor vs Rest (Recovered, Significantly Improved, Improved)

Small sample size did not allow further analyses to examine the relationship between the SOBS, type of therapy and treatment outcome.

#### **4.E.5. Multivariate Evaluation of the Prognostic Factors**

Finally, to examine the prognostic value of the family EE, PAS, PRSS and SOBS in a multivariate analysis level, forward logistic regression analyses were performed. When the poor outcome group vs the rest of the outcome categories was introduced as dependent variable, the PD and patient blaming mother were no longer statistically significant while the association of the other variables with treatment outcome were still significant. To control the effect of each of these variables on outcome by the others, they were introduced into the equation of the logistic model in pairs. Regression analyses revealed that mothers' CC and SB, and patients' SD and EEn were all significantly associated with outcome (see Table 4.E.8.). The patients' progress in treatment at T2 could be predicted by the mothers' CC and SB, and the patients' SD and EEn ratings at T1. The mothers' CC and SB ratings were correlated with poor outcome at T2, whereas the patients' SD and EEn ratings were associated with the patients' progress in therapy at T2.

**Table 4.E.8. Forward logistic regression analysis on GOC at T2, mothers' CC and SB, and patients' SD and EEn at T1**

<b>Variable</b>	<b>B</b>	<b>S.E.</b>	<b>Wald</b>	<b>df</b>	<b>Sig</b>	<b>Exp (B)</b>
<b>Mothers' CC</b>	<b>.72</b>	<b>.30</b>	<b>5.78</b>	<b>1</b>	<b>.01</b>	<b>2.06</b>
<b>SD</b>	<b>-1.34</b>	<b>.53</b>	<b>6.22</b>	<b>1</b>	<b>.01</b>	<b>.26</b>
<b>Constant</b>	<b>3.25</b>	<b>1.81</b>	<b>3.20</b>	<b>1</b>	<b>.07</b>	
<b>Mothers' CC</b>	<b>.81</b>	<b>.33</b>	<b>5.99</b>	<b>1</b>	<b>.01</b>	<b>2.25</b>
<b>EEn</b>	<b>-1.42</b>	<b>.50</b>	<b>8.02</b>	<b>1</b>	<b>.004</b>	<b>.24</b>
<b>Constant</b>	<b>2.57</b>	<b>1.35</b>	<b>3.58</b>	<b>1</b>	<b>.05</b>	
<b>Mothers' CC</b>	<b>.50</b>	<b>.26</b>	<b>3.65</b>	<b>1</b>	<b>.05</b>	<b>1.66</b>
<b>Mothers' SB</b>	<b>.55</b>	<b>.27</b>	<b>4.04</b>	<b>1</b>	<b>.04</b>	<b>1.73</b>
<b>Constant</b>	<b>-3.04</b>	<b>1.07</b>	<b>7.97</b>	<b>1</b>	<b>.004</b>	
<b>Mothers' SB</b>	<b>.89</b>	<b>.32</b>	<b>7.38</b>	<b>1</b>	<b>.006</b>	<b>2.44</b>
<b>SD</b>	<b>-1.68</b>	<b>.64</b>	<b>6.90</b>	<b>1</b>	<b>.008</b>	<b>.18</b>
<b>Constant</b>	<b>2.66</b>	<b>2.07</b>	<b>1.65</b>	<b>1</b>	<b>.198</b>	
<b>Mothers' SB</b>	<b>.69</b>	<b>.29</b>	<b>5.73</b>	<b>1</b>	<b>.01</b>	<b>2.00</b>
<b>EEn</b>	<b>-1.33</b>	<b>.50</b>	<b>7.05</b>	<b>1</b>	<b>.007</b>	<b>.26</b>
<b>Constant</b>	<b>1.34</b>	<b>1.64</b>	<b>.66</b>	<b>1</b>	<b>.413</b>	

**GOC= General Outcome Categories; SD= Self-Disclosure; EEn= Emotional Engagement; CC= Critical Comments; SB= Self-Blame**

Forward logistic regression also excluded the patients' EEn from the equation when it was controlled by the patients' SD (SD:  $p = .0007$ ; EEn:  $p = .27$ ). Therefore, the three significant variables i.e., the mothers' CC and SB, and the patients' SD, remained to be tested for their predictive value at the same time. The same analysis was performed. When the poor outcome group vs the rest of the outcome categories was introduced as dependent variable, the difference between the two outcome groups remained significant for the mothers' SB ratings ( $p = .02$ ) and the patients' SD ratings ( $p = .01$ ), but disappeared for the mothers' CC ratings ( $p = .11$ ). Both mothers' SB and patients' SD continued to predict outcome. A higher rating of mothers' SB was correlated with a poorer outcome, and a higher rating of patients' SD was associated with the

patients' progress in therapy at T2 (see Table 4.E.9.)

**Table 4.E.9. Forward logistic regression analysis on GOC at T2, mothers' CC and SB, and patients' SD at T1**

<b>Variable</b>	<b>B</b>	<b>S.E.</b>	<b>Wald</b>	<b>df</b>	<b>Sig</b>	<b>Exp (B)</b>
<b>Patients' SD</b>	<b>-1.86</b>	<b>.77</b>	<b>5.79</b>	<b>1</b>	<b>.01</b>	<b>.15</b>
<b>Mothers' SB</b>	<b>.77</b>	<b>.33</b>	<b>5.27</b>	<b>1</b>	<b>.02</b>	<b>2.17</b>
<b>Mothers' CC</b>	<b>.65</b>	<b>.41</b>	<b>2.51</b>	<b>1</b>	<b>.11</b>	<b>1.92</b>
<b>Constant</b>	<b>2.64</b>	<b>2.27</b>	<b>1.35</b>	<b>1</b>	<b>.24</b>	

**GOC= General Outcome Categories; SD= Self-Disclosure; SB= Self-Blame; CC= Critical Comments; T1= Intake; T2= Termination of treatment**



## **CHAPTER 5**

### **DISCUSSION OF THE RESULTS**

The results of this study will be briefly summarized in the first section of this chapter. The second section addresses the limitations of the study. In the third section, the principal hypotheses and questions of the study will be presented. This discussion presents the empirical findings in five sections. Section A addresses four issues: 1) the distribution of family EE in eating disordered patients; 2) the changes of family EE during the course of therapy; 3) clinical factors associated with the diagnostic groups; 4) the distribution of treatment outcome for AN and BN patients. Section B presents two issues: 1) the distribution of personality disorder diagnoses in eating disordered patients; 2) clinical features of the patients and family factors associated with personality disorders. The following two sections discuss the results concerning the patient's response style to therapist and therapy, and the self- and other-blame, respectively. In section C, three issues are presented: 1) reliability of the PRSS; 2) the distribution of the PRSS in families with an eating disorder patient; 3) clinical features of the patients and family factors associated with the PRSS. Section D presents three issues in a similar way: 1) reliability of the SOBS; 2) the distribution of the SOBS in families with an eating disorder patient; 3) clinical and family factors associated with the SOBS. The final section, section E, reports prognostic values of the EE, PD, PRSS, and SOBS, respectively.

#### **5.1. SUMMARY OF THE RESULTS**

This thesis has explored four issues: (1) family expressed emotion, (2) personality disorders, (3) patients response style to therapist and therapy, and (4) self- and other-blaming feelings in a sample of families with an eating disorder patient. Chapter 4 presented the results of the study into

five sections. The results will be briefly summarized here.

In section A, the distribution of family expressed emotion was reported , with rating based on the Standardized Clinical Family Interview (SCFI), both at intake and at the termination of treatment. At intake, the levels of critical comments, hostility, emotional over-involvement, and positive remarks were rated low. The mean number of critical comments for the fathers was 1.29 and for the mothers 1.60. Mothers of bulimic patients scored higher on critical comments and emotional over-involvement than did mothers of anorexic patients. Levels of critical comments in single-parent families was significantly higher than those of two-parent families.

The mean number of critical comments and emotional over-involvement had significantly decreased over the treatment period for both fathers and mothers, whereas the mean number of parents' warmth had significantly increased. Nearly half of the high EE families, 45.50%, at intake had become low EE in the course of treatment. In three families in which patients had made poor progress at the end of treatment period, family EE levels had changed from low to high. The AN patients showed significantly more improvement during the course of therapy than did BN patients.

Section B presented the distribution of personality disorders based on the Personality Assessment Schedule (PAS). Overall, 39.7% of the patients had no personality disturbances and 46.9% of the patients met the PAS criteria for at least one personality disorder. The BN patients tended to fulfill the criteria more than the AN patients. The most common diagnoses were “anxious” and “histrionic” personality disorder in the AN and BN group, respectively. The association between family EE scales and personality disorders was detected. Mothers of the patients with personality

disorder tended to show more critical comments to the patients than did mothers of the patients without personality disorder. Over the treatment period the family EE levels of patients without personality disorder had reduced, whereas the family EE levels of the patients with personality disorder had remained the same.

Section C reported the distribution of the patient's response style to therapist and therapy as measured by the Patients Response Style Scale (PRSS). This is a new measure developed specifically for the purpose of this study. It was shown to have good interrater reliability and a robust predictor of outcome. Bulimics tended to reveal more mixed disclosing style which implies more discrepancy between the levels of verbal and non-verbal measures in this group of patients than anorexics. Anorexics also demonstrated a greater significant correlation between verbal and nonverbal aspects of their response style to therapist and therapy than did bulimics.

Patients without personality disorder tended to show more Self-Disclosure (SD) and Emotional Engagement (EEn) than did patients with personality disorder.

Section D presented the distribution of self- and other-blame feelings among families with an eating disorder patient based on the Self- and Other-Blame Scale (SOBS) ratings. This is a new measure developed specifically for the purpose of this study. It was shown to have good interrater reliability and a robust predictor of outcome.

Mothers blamed themselves for the patient's eating problems significantly more than did fathers. Patients expressed significantly more other-blaming attitudes towards their parents than did parents to the patients. Families of bulimic patients tended to express more self- and other-blaming attitudes than families of anorexic patients. Bulimics were more likely to blame their parents specifically the mothers, whereas anorexics tended to blame themselves rather than their

parents. Families of the patients with personality disorder blamed themselves more than did families of the patients without personality disorder. Eating disorder patients with additional PD also blamed themselves, as well as their parents more than did those without PD. Patients who blamed themselves tended to express higher levels of SD/EE<sub>n</sub> whereas patients who blamed their parents (particularly mothers) tended to reveal lower levels of SD/EE<sub>n</sub>.

The investigation of the association between the SOBS and family EE scales revealed that family (both mothers and fathers) other-blaming feelings were positively and significantly correlated with family critical comments and family hostility, and negatively correlated with family warmth. High EE families showed significantly more self-blame and also other-blaming attitudes than did low EE families.

Section E reported the prognostic factors. It was shown that patients' progress in treatment at the termination of treatment, as assessed by the general outcome scores of the Morgan-Russell scales, could be predicted by 1) the mothers' critical comments at intake: Higher critical comments at intake was correlated with poorer outcome at the end of treatment period; 2) the patients' PD: Patients without personality disorder showed significantly more improvement in treatment at the termination than did the patients with personality disorder; 3) the PRSS: Examination of the predictive power of the PRSS revealed that patients who showed higher levels of SD/EE<sub>n</sub> to the therapist and therapy did significantly better in psychotherapy than those who showed lower levels of SD/EE<sub>n</sub>. The PRSS was also shown to be a particularly powerful predictor of outcome in focal psychoanalytic therapy and to a lesser degree predicted outcome in family therapy and cognitive analytic therapy; 4) the SOBS: Concerning prognostic value of the SOBS, poor outcome in therapy was related to both the mothers' SB and the patients blaming mothers.

Finally, multivariate analyses revealed that the patients' SD ratings, as well as the mothers' SB scores, could be considered as the most important predictive factors in the present study.

## **5.2. LIMITATIONS OF THE STUDY**

The present research has several limitations which should be kept in mind when interpreting the findings and addressed in future studies. Several methodological issues require close attention.

First, it has been shown (e.g., the present study; Van Furth, 1991; Valone et al., 1983) that dual high-EE families are distinct from the mixed and dual low-EE families both in how much total criticism they express toward the patient in direct interactions, as well as in the specific kind of criticism expressed. The frequency of two-parent families in which both parents are interviewed is indicative of the percentage of possible false negative low-EE families in a sample. When only one parent of a two-parent family is assessed and classified as low-EE, the family could be wrongly classified as low-EE when the other partner is in fact high EE.

Second, in the study of personality disorders in eating disorders which are relevant to the present investigation and to the other studies cited previously, most reports are of limited value since it is very difficult to separate the psychopathological characteristics that are possible precursors of the disorder from those that are byproducts of a serious illness or are secondary to starvation. Over-diagnosis of personality disorders using different diagnostic schemes could occur as well, owing to the presence of an affective illness. Therefore, rates of major affective illness in the subject groups should always be measured. Important details regarding the clinical samples should be included, since different results are obtained with different patient groups. Researcher biases always exist and are hard to assess. The use of psychiatric and nonpsychiatric control groups is

important in trying to clarify this issue.

Third, although the initial individual assessment interview had enough and appropriate probes to evaluate the patient's response style to therapist and therapy, the interview was not designed specifically to yield self-disclosure and emotional engagement. More complete information concerning the PRSS subscales could be obtained in future studies by devising an assessment procedure that is exclusively designed to assess the various parameters of the scale.

Further, self-disclosure and emotional engagement need to be examined on a more case-specific basis, taking contextual cues into account to provide information about the clinical significance of disclosures and how they function in different cases. Perhaps the nature of the problem, the background of the patient, or the interpersonal dynamics, such as family self-disclosure during a family assessment interview, could be used to provide greater understanding of the differential use and helpfulness of both verbal and nonverbal aspects of disclosure within cases.

Fourth, one issue concerning the SOBS is how the relation between self/other-blame and outcome changes over the course of psychotherapy. The present study was not designed to evaluate this. However, only longitudinal investigations can address this issue directly. A second issue that is not addressed by our model is the clarification of the SOBS construct, causality and responsibility, and how each may have unique implications for outcome. Most researchers to date have not attempted to conceptualize very precisely how self/other-blame and adjustment are linked. Part of the problem may lie in the fact that feelings of guilt or blame are somewhat difficult to assess. Nevertheless, most operationalizations include a focus on affect which is negatively toned and which includes a sense of responsibility or causality. Although these elements can be distinguished

(one can feel responsible without guilt, or accept that one has caused an effect without feeling negative about it), they often co-occur when people blame themselves for negative outcomes (Shaver, 1985). In the absence of careful operationalization of the responsibility construct in empirical analyses, one is likely to impose idiosyncratic meanings on the term when it is encountered. One solution to this problem is to ask separate and distinct questions about causality, responsibility, and blameworthiness.

Fifth, although both the PRSS and the SOBS have been shown to be powerful predictors of outcome and the results of the present study have provided evidence for reliability and validity of these newly developed measures, these data link the PRSS and the SOBS scores with outcome only. While this relation is a necessary element to claim validity, it alone may not be sufficient to link these instruments to the theoretical construct they wish to measure. More research is needed to demonstrate directly the content of these measures adequately and without bias to ensure that they represent the theoretical construct they contend to measure.

Together with methodological limitations, composition of the sample limits the generalizability of the results of this study in some aspects. The Maudsley Hospital is a tertiary referral centre for patients with eating disorders, and most patients have failed in treatment elsewhere before seeking therapy at the Maudsley. As a result, this sample represents the more severely impaired end of the eating disorders spectrum, where one might expect personality disorders or previous treatment to be disproportionately represented. To illustrate this point, 74% of the patients in this study have had previous treatment for eating disorders, at other centres. This factor might have caused the treatment sample to be biased in favour of seriously ill and poor prognostic cases. Caution must be exercised in generalizing the results of this study. A different pattern of outcome would

be likely in a centre serving a different population of patients.

It should be noted that in this study I was unable to test several potential variables that have been considered to moderate the effect of either family-related, patient-related, or therapy-related factors on outcome. Consequently the conclusions that could be drawn from the study were reduced. This was mainly because of small sample size involved in different therapeutic groups or of those who filled in self-report questionnaires.

The high number of drop-out of patients (38%) also led to a decrease in sample size and this may limit generalizability of the results too.

This investigation did not include a normal control group, or male subjects, for comparison. The BN sample was smaller than AN sample, which made some comparisons difficult.

The diversity of the kinds of treatment and the differences in therapists may also have influenced patient outcome and parental EE levels.

Finally, this study has not addressed the issue of whether the benefit of the different treatment modalities, as well as changes in patient- and family-related factors, seen at 1-year follow-up will also be evident in the longer-term outcome, for example, at 4 years or later.



## **5.3. DISCUSSION OF THE RESULTS**

### **5.3.A. EXPRESSED EMOTION**

#### **5.3.A.1. The Distribution of Family Expressed Emotion**

The family EE ratings reported in this study are low on CC, HOS, EOI, and PR; and moderate on W. These levels of family EE are comparable to the low levels of EE reported by Le Grange et al. (1992a) and Dare et al. (1994), who studied family EE in a younger eating disorder sample using the same instrument (SCFI); and by Van Furth (1991) and Van Furth et al. (1996), who investigated family EE in a younger eating disorder sample, but using a standard individual interview (CFI). Using the CFI, however, Szmukler et al. (1985) found higher levels of parental EE, especially for mothers (mean score of mothers' CC= 5.33) in a sample of eating disorder patients. Methodological-, cultural-, and family-related factors could be considered as possible explanations for similarities and differences between these findings.

In a family interview like SCFI, each family member has less time to express his/her emotion (Dare et al, 1994; Le Grange et al, 1992a). A comparison between single- and two-parent families showed that single-parent families scored significantly higher on CC than did two-parent families. This finding suggest that the variables involved in measuring EE may well be influenced by the situation in which they are assessed.

In spite of using different instruments, comparability of family EE levels of the current British study to the Dutch (Van Furth, 1991; Van Furth et al., 1996) studies could be explained by interaction of methodological and cultural factors. Using the same interview (CFI), Van Furth (1991) found that the levels of the Dutch mothers' CC was significantly lower than the ratings in the British sample (Szmukler et al., 1985). The same results were obtained for the Dutch fathers,

but the difference was not statistically significant. If the method of assessment was the only reason, family EE scores in the present study would have been significantly lower than of those in the Dutch studies. Therefore, interaction of methodological and cultural factors seems to be responsible for the comparability of family EE ratings in the two settings.

Minuchin's et al. (1975, 1978) psychosomatic family model proposes four qualities for families containing an eating disorder patient, enmeshment, overprotectiveness, rigidity, and the lack of conflict resolution. Based on Minuchin's et al. (1975, 1978) definition of these constructs, one might predict higher levels of EOI and lower scores of criticism as rated on EE (Dare et al., 1994). Consistent with Dare et al. (1994), however, the results of the present study do not confirm these predictions. The low level of family EOI in this eating disorder sample either as expressing exaggerated emotional responses which is the most closely related to enmeshment, or as overprotectiveness which is the most closely to Minuchin's et al. (1978) description of overprotectiveness, is in contrast with the psychosomatic model. As "a family with a problem" rather than "dysfunctional family" (Frude, 1990), in the case of higher level of EOI, it could be explained as having a high level of "commitment" (Dare et al., 1994) or "concern" (Hooley, 1985) to each other especially toward the patient. However, as Dare et al. (1994) point out, the lower level of EOI limits channels of communication and this can give some support to the Minuchin's et al. account of rigidity (1975, 1978). And, as can be seen later, interactional dynamics of some family- and patient-related factors of high EE relatives might be explained in terms of "enmeshment" and "overprotectiveness" of this group of families.

The low levels of criticism and hostility in relatives of eating disorder patients might indicate a process of avoiding the expression of differences between family members (Goldstein, 1981; Kog

& Vandereyken, 1989; Minuchin et al., 1978) and a pattern of low levels of conflict (Dare et al., 1994). However, it is equally possible that the “lack of conflict resolution” provokes more conflict and criticism between the family members. Findings of self- and other-blaming attitudes in the present study provides some support for this assumption.

Mothers tended to be more positive and showed more emotions and warmth to the patients than did fathers. This is consistent with the finding of Dare et al (1994) and may provide empirical confirmation for the view often put forward by family therapists that problems often arise in families when there is an overclose relationship between mother and child and the father is peripheral.

#### **5.3.A.2. Changes in Family Expressed Emotion During the Course of Therapy**

Several studies (e.g., Dulz & Hand, 1986; Hogarty et al, 1986; Leff et al., 1982; Tarrier et al., 1988) have shown the variability of family EE scales in families with a schizophrenic patient over time. An overall change in level of CC and EOI during the course of therapy was reported in families containing an eating disorder patient (Le Grange et al., 1992a, 1992b; Van Furth, 1991; Van Furth et al., 1996). In the present study, changes in family EE scales were significant for CC, EOI, and W over the treatment period. Regardless of treatment outcome, families tended to show less CC and EOI, and more W to the patients over time. These changes in family EE scales, thus, can be considered as adaptive coping strategies (Hubschmid & Zemp, 1989; Kavanagh, 1992). Changes in family EE, however, were significantly greater in the patients who had made at least some progress than those who made a poor progress. As predicted, this may indicate that changes in the EE variables are associated with the symptomatic changes in the patients. Changing from low to high EE in three families in which the patients had made poor outcome, further supports

the notion that symptomatic improvement may reduce the levels of EE.

Several interactional patterns of the SOBS and EE variables between family members (particularly mother-patient) may be considered as underlying mechanisms for the association between symptomatic change and changes in parental EE. One possibility is that each member of the family may feel guilty and responsible for their contribution to either the problem or the resolution of the problem. Based on this explanation, SB reinforces the sense of responsibility and encourages members of the family to make continued effort to resolve the problem. The association found between the patient's SB and progress during the course of therapy (most likely through increasing SD, see later) gives support to this explanation. The mothers' SB, however, is difficult to be explained. It is possible that the positive function of SB on the part of the patient is responsible for changes in the parental EE levels. This is demonstrated by decrease of their CC and EOI, and increase in their warmth.

In contrast, other-blaming attitudes seem to fire conflicts between family members and consequently affect both the family EE levels and the patient's progress in therapy. This explanation is further supported by the association between patients' blaming mother and poor progress in therapy, as well as high correlation between parent's CC and blaming patient.

### **5.3.A.3. Factors Associated with the Diagnostic Groups**

The patients' eating problems, AN and BN, were significantly associated with some of the other clinical factors. As would be expected, the BN patients had significantly higher scores in the bulimic measures (BITE, EDI subscales). No significant association was found between anorexics and bulimics in terms of: age at presentation, age at onset of eating disorder, duration of illness,

BMI, BDI, average score of the Morgan-Russel scales, IIP, and MOCI ratings.

Mothers of bulimic patients tended to be more critical and overinvolved than mothers of anorexics. This may indicate that families with a bulimic patient engage in conflicts more than families with an anorexic patient. It may also indicate that the bulimia symptom on the one hand provokes and on the other hand is maintained by more emotional expression in these families (Dare et al., 1994; Dolan et al., 1990; Garner et al., 1985; Goldstein, 1981; Johnson & Flach, 1985; Strober, 1981; Szmukler et al., 1985; Van Furth, 1991).

#### **5.3.A.4. The Differences in Treatment Outcome for AN and BN Patients**

Anorexic patients showed significantly more progress during the course of therapy than did bulimic patients. Several possibilities could account for this finding. The first possibility is that families of BN patients are more conflictual in their relationships than families of AN patients as measured by EE. This conflictual patterns of relationship could be responsible for the BN patients and their families being more difficult to engage in effective treatment (Szmukler et al., 1985).

Negative function of OB, as well as positive function of SB could be considered as a second explanation. Bulimic patients were identified to blame their parents more whereas AN patients showed more self-blaming attitudes. Associations found between OB and poor progress in therapy on one hand and between SB and progress in therapy on the other lend support to this explanation.

Different patterns of PRSS could also explain this finding. Bulimic patients demonstrated more discrepancy on the PRSS scales (SD, EEn) than AN patients did. Bulimic patients also tended to be less disclosive to the therapist than AN patients. The association found between the PRSS

variables and treatment outcome gives support to this explanation. This explanation is further supported by the interactional function of the PRSS and EE variables. Mothers of patients with lower levels of SD/EE<sub>n</sub> revealed more CC and EOI than mothers of patients who showed higher levels of SD/EE<sub>n</sub>.

### **5.3.B PERSONALITY DISORDERS**

#### **5.3.B.1. The Distribution of Personality Disorder Diagnoses**

The results of this study demonstrate that nearly half the patients with eating disorders met criteria for at least one personality disorder. A prevalence rate of 46.5% among patients with eating disorders in this study closely approximates prevalence rates of personality disorders in subjects with eating disorders shown by McClelland et al. (1991) - 52% - and Fahy et al. (1993) - 39%. These personality disorder rates are also in keeping with those found in other diagnostic groups by Casey (1988) - 40% and Tyrer et al. (1983) - 39.9%, using the same instrument, the PAS. Consistent with previous studies (e.g., Piran et al., 1988; Wonderlich et al., 1990), the most commonly assigned PAS personality diagnoses were histrionic personality disorder (26.7%) in the BN patients, similar to the rate found by Fahy et al. (1993) - 22%, and anxious personality disorder (16.3%) in the AN patients. The results support our prediction that a histrionic PD would be more common in bulimic patients, whereas anorexics would be characterized more by anxious PD.

Further, consistent with previous findings (Garfinkel & Garner, 1982; Strober, 1985), this result provides support for the hypothesis of pathoplastic influences of personality on the clinical expression and course of illness, in which histrionic characteristics seems to influence bulimic symptoms, whereas anxious characteristics seem to influence anorexic symptoms.

Consistent with previous findings (Bruch, 1973; Crisp, Hsu & Stonehill, 1979; Gomes & Dally, 1980; Solyom, Freeman & Miles, 1987; Stonehill & Crisp, 1977), the findings of this study also give support to the predisposing hypothesis which considers personality to provide a “characterological” predisposition to the development of eating disorders. However, this hypothesis could be best proved when patients are assessed both at admission and at the termination of treatment when there has been symptomatic recovery.

A secondary diagnosis of histrionic PD in eating disorder patients is of particular clinical significance, as it is associated with poor therapeutic outcome (all four histrionic PD patients had a poor outcome at the end of treatment programme), whereas the association between a secondary diagnosis of anxious PD in eating disorder patients has limited clinical significance since 40% (2/5) of the patients had a poor outcome and 60% (3/5) were rated as recovered or significantly improved. However, interpretation of the results must be conducted with caution because of the small sample sizes.

As predicted based on the earlier studies, the finding of 50% prevalence of the dependent PAS cluster in BN patients and 40% prevalence of the inhibited PAS cluster closely parallel the association of BN with the dramatic-eratic DSM-III-R personality cluster and AN with the anxious-fearful personality cluster (e.g., Piran et al., 1988; Wonderlich et al., 1990), respectively. Other studies, however, do not support these associations (Gartner et al., 1989; Pope et al., 1987; Steiger et al., 1991). The differential distribution of the PAS personality disorder diagnoses among eating disorder subtypes suggests the employment of different interventions across different personality types. Further studies are required to examine this issue.

### **5.3.B.2. Clinical Features of the Patients and Family Factors Associated with Personality Disorders**

When compared with the group without personality disorders, those with personality disorders tended to have a younger age of onset of eating disorder, a longer duration of illness, and a lower BMI. These results are consistent with the findings of a previous study (McClelland et al., 1991) using the same instrument. It was found that patients with personality disorder evidenced more psychiatric symptoms than patients with eating disorders who did not have the diagnosis of personality disorder. Several possibilities could account for this finding which is also consistent with Cooper's et al. (1988) findings in BN patients. One possibility is that psychopathological characteristics in patients with PD are merely artifacts of prolonged starvation and ongoing emotional distress in the patients (Crisp et al., 1979; Gomes & Dally, 1980; Stonehill & Crisp, 1977; Strober, 1980), other investigations, however, have found that PD can not be explained as starvation-induced artefacts (Gartner et al., 1989; Piran et al., 1988). Longitudinal studies which assess eating disorder patients at presentation and following treatment in which symptom reduction has occurred would represent a first step in clarifying this important issue.

Two other possibilities which should be considered are the presence of a major affective disorder and the effect of family factors. Major affective disorders have been found to increase the diagnoses of PD (Hirschfeld et al., 1983; Pope et al, 1987). Other findings (Piran et al., 1988), however, do not support this association. It is also speculated (Fahy et al., 1993) that the converse, i.e. that a PD increases the risk of affective disturbance, may be true. Previous studies have typically not examined the influence of concurrent eating pathology or psychopathology on personality tendencies and vice versa. Further studies, employing different patient groups, are needed to clarify this important issue and to examine the way in which personality function can



be separated from clinical symptoms.

As predicted, families of those patients who met criteria for PD were more critical to the patients than families of patients without a PD. This finding gives limited support to the potential associative role of family dysfunction, that has been noted by a number of researchers and clinicians (e.g., Humphrey, 1989; Kog & Vandereyken, 1989; Walles et al., 1989; Wonderlich & Swift, 1990). For example, Wonderlich and Swift (1990) have shown that the borderline personality disorder (BPD) subgroup of anorexics and bulimics perceive their parents as abnormally hostile, while Johnson et al. (1990) report that this BPD subgroup has a family history of psychiatric disturbance.

Therefore, family CC could possibly be responsible for psychopathological impairments of this group of patients. Again it could be possible that family CC is influenced by patient-related factors. The interactional relationship between family CC and patients' symptomatology was discussed in section A (5.3.A.2.) of this chapter. Study of both patient-related factors including patients' mental state and personality, and family-related factors will lead to better assessment and understanding, if such an interactional orientation is employed.

### **5.3.C. PATIENT'S RESPONSE STYLE TO THERAPIST AND THERAPY**

#### **5.3.C.1. Reliability of the PRSS**

The results of the present study suggests that the PRSS is a reliable assessment procedure for measuring the patient's self-disclosure and emotional engagement in the course of a psychotherapy assessment interview. The results further indicate the construct validity of the PRSS and provide some evidence for its predictive validity as well. Needless to say, more

empirical work will be needed to provide further evidence for the validity of the measure and its underlying constructs. Continued use of the PRSS in investigations of therapeutic interactions of patients from different clinical populations with therapists from different orientations using different styles of intervention will help to establish whether or not PRSS can identify varying styles of patient's response to therapist and therapy across diverse therapeutic context. Ultimately, however, the utility of this measure will depend upon future research that will demonstrate how well its components relate to consequences of therapy. While the preliminary evidence is encouraging in this regard, more extensive investigations of the relationship between patient's response style to therapist and therapy and various psychotherapy outcome criteria will be needed to determine the potential of PRSS as a predictor of therapeutic outcome.

#### **5.3.C.2. The Distribution of the PRSS in Families with an Eating Disorder Patient**

In this study, subjects were classified as either high, moderate, low, or mixed for both SD and EEn based on the PRSS measures. Presenting more mixed-SD/EEn response style, as well as less positive correlation between the PRSS scales, BN patients demonstrated more discrepancy between verbal/nonverbal aspects of SD. This discrepancy may establish support for the previous findings (Dare et al., 1994; Dolan et al., 1990; Garner et al., 1985; Goldstein, 1981; Johnson & Flach, 1985; Strober, 1981; Szmukler et al., 1985; Van Furth, 1991) which indicate a greater degree of interpersonal and emotional conflict in bulimics than anorexics.

It was also found that AN patients showed more SD than bulimics. Two possible explanations for this finding must be considered. The first explanation is that bulimic patients themselves, as well as their families, are suffering from interpersonal conflicts more than anorexic patients. The bulimic's ability/willingness to SD, is then affected by this interpersonal quality which makes them

less able or willing to disclose themselves to others, in this case the therapist.

The nature of information which the PRSS rating is based on, could be considered as a second explanation. Having more conflictual family relationships, bulimic patients are more likely to experience difficulty in disclosing information about their family than anorexics. Further research should examine the association between discrepancy of verbal/nonverbal aspects of SD and symptomatology.

### **5.3.C.3. Clinical Features of the Patients and Family Factors Associated with the PRSS**

Comparing different levels of PRSS in terms of clinical and family factors resulted in those with low- and mixed-SD/EEEn ratings being more likely to report symptoms of depression and psychological impairments than those with high-SD/EEEn scores.

Consistent with previous studies (Cozby, 1973; Jourard, 1964; Mowrer, 1961; Rogers, 1961), this finding may indicate that the PRSS levels and scales are influenced by the patient's mental state. This was further supported by a positive association between the PRSS scales and the patients' physical (e.g., BMI) and psychological (measured by the Morgan-Russel scales) well-being.

Typically, patients have multiple behavioural problems that impair their response style (SD, EEEn). Clinical assessment needs to establish which patient behaviours covary, if any, and the direction of relationships among behaviours. For example, the level of depression may affect or be affected by the level of SD/EEEn.

Association between the PRSS and EE variables revealed that mothers of patients with lower levels of SD/EEEn tended to be more critical and emotionally overinvolved with patients, than

mothers of patients who showed higher levels of SD/EE<sub>n</sub>. Situations with a high demand for disclosure are likely to reactivate the conflictual and critical pattern of the mother-patient relationship, reducing the patient's ability to disclose information.

There is a strong indication that low SD/EE<sub>n</sub>, either because of negative and defensive behaviour or simply an absence of the SD/EE<sub>n</sub> qualities, can serve as a mechanism to avoid intimacy. Fear of intimacy can include fear of being hurt and loss of self.

Another explanation is that intrafamilial patterns of SD determine to a large extent a family's stance toward disclosure to those outside the family. From a systemic perspective, the family seeks to maintain its present balance of power and functions so as not to upset the equilibrium that has been established. This pressure toward homeostasis tends to maintain the existing SD patterns. Members of an emotionally overinvolved family with diffuse boundaries within the unit are likely to pull a rigid boundary around the total family structure. The intensity of interaction within the family satisfies the members' need for communication and intimacy. Attempts by family members to move toward people outside the family are highly threatening to family members and are met by resistance from within. Any moves toward independence are met by drawing the net of family involvement even tighter.

In contrast, in a family with low levels of CC and EOI in which boundaries within the family unit are more clear, relationships with people outside the family are allowed. These families not only offer members gratification within the context of family relationship, but also encourage them to enjoy outside relationships. Significant association of mothers' W and PR with the patients' level of SD/EE<sub>n</sub> further supports this explanation.

Results of the analysis for PD vs NPD groups showed that eating disorder patients who demonstrated lower SD and EEn to the therapist and therapy had more personality disturbances than did those who reported higher SD and EEn. This result is consistent with previous findings (Jourard, 1964; Mowerer, 1961; Rogers, 1961) that SD is a sign of a healthy personality. This notion is further supported by the correlations of PD with both interpersonal problems (measured by IIP) and interpersonal distrust (measured by EDI). However, the complex interaction between personality and situational factors does not allow us to draw a simple direct line between personality and SD. Clinical observations, as well as hypothetical impressions, show that what is called a healthy personality can be affected by a particular situational factor resulting to a low level of SD. It is also likely that an appropriate situational factor facilitates the process of disclosure in an individual suffering from personality disturbances.

#### **5.3.D. SELF- AND OTHER-BLAME**

##### **5.3.D.1. Reliability of the SOBS**

Overall, the results of the present study suggest that the SOBS is a reliable assessment procedure for measuring self- and other-blaming attributions experienced by patients and their families in the course of a psychotherapy assessment interview. The results further provide some evidence for the construct validity, as well as predictive validity of the SOBS. Needless to say, more empirical work will be needed to provide further evidence for the validity of the measure and its underlying constructs.

##### **5.3.D.2. The Distribution of the SOBS in Families with an Eating Disorder Patient**

More than two thirds of the sample including both patients and relatives reported feelings of self-blame (SB) for the patients' eating problems, whereas less than one third of the sample showed

feelings of blaming someone else for the same reason.

Mothers expressed self-blaming feelings the most and other-blaming feelings the least. High levels of feeling of self-blame among relatives of eating disorder patients are consistent with clinical observations in this population (Dare et al., 1990; Dare et al., 1994; Le Grange et al., 1992a; Szmukler et al., 1985).

Several possible explanations for this finding must be considered. The first explanation is that families with an eating disorder patient are characterized by a lack of conflict resolution (Minuchin et al., 1975, 1978) which, in turn, leads to feelings of guilt and self-blame (Dare et al., 1990; Dare et al., 1994; Le Grange et al., 1992a). In this regard, self-blame is considered as a mechanism to avoid conflict and therefore bypass problems of conflict resolution. This mechanism, however, was not found in high EE families characterized by high level of criticism and interpersonal conflict.

The second possibility is that feelings of SB play an adaptive role (e.g., Afflack, McGrade et al., 1985; Bulman & Wortman, 1977; Janoff-Bulman, 1979; Timko & Janoff-Bulman, 1985; Tennen et al., 1984) among families with an eating disorder patient. This explanation, however, is weakened by those findings which showed either no association between SB and adaptation (Affleck, Allen et al., 1985; Taylor et al., 1989; Witenberg et al., 1983) or association between SB and poor adaptation (Frey et al., 1985; Healy et al., 1993; Meyer & Taylor, 1986; Nielson & McDonald, 1988).

In line with systemic thinking, the last possible explanation is that each member of the family may plausibly feel either that he/she contributed to the problem or that he/she should contribute to the

resolution of the problem. Self-blame may then reinforce both:

a) positive family relationship patterns which encourage members of the family to make continued efforts to resolve their conflicts and problems including the patient's eating problems.

b) dysfunctional relationship patterns between family members (particularly mother-patient). An inappropriate sense of SB may result in maintenance of unhealthy interaction patterns and pointless efforts to change the situation. Continued efforts to resolve the problem may not only preclude the patient's progress in treatment (as can be seen later), but may also annoy parents who recognize the problem as unresolvable. Perhaps the latter explanation is more likely in terms of findings of the present study.

Whether SB is adaptive as noted above, or not either simply as conflict avoidant function or rather poor adaptation, requires further investigation. Future research should be aimed at identifying precisely how SB compromises adjustment in this situation, as well as clarifying the kinds of situation in which this relationship holds.

Mothers tended to show more self-blaming feelings for the patients' eating problems than did fathers. One possible explanation for this finding which is consistent with Szmukler's et al. (1985), is that mothers are more emotionally involved with their eating disorder offsprings than fathers. It seems that mothers' SB will be increased by mothers' EOI. Whatever the manifestation of EOI (overcontrol/protection, self-sacrificing, emotional identification), this closed system seems too "enmeshed" and "rigid" in its boundaries and functions to be able to psychologically differentiate between subsystems. This mechanism will be employed to explain interactional patterns of SOBS and EE variables and the way they alter in relation to one another.

Consistent with Humphrey (1988), families of bulimic patients tended to show both self- and other-blaming feelings slightly more than did families of anorexic patients. Lending support to earlier explanation of families of bulimics being more critical and more overinvolved than families of anorexics, this finding may also indicate that families with a bulimic patient engage in conflicts more than families with an anorexic patient (Dare et al., 1994; Dolan et al., 1990; Garner et al., 1985; Goldstein, 1981; Johnson & Flach, 1985; Strober, 1981; Szmukler et al., 1985; Van Furth, 1991). This may simply reflect the interactional functioning (attack-counterattack communication style- see later) of the other-blaming attitudes in which parents blaming patients is followed by patient blaming parents in families with a bulimic patient, as well as high EE families. This explanation is further supported by the finding that bulimic patients tended to blame their parents (specifically mothers) whereas anorexic patients tended to blame themselves rather than their parents.

Intercorrelational patterns of the SOBS scales revealed a minor association between self- and other-blaming feelings among the patients and relatives indicating two different attitudes. However, they can easily affect one another and alter SB feelings to OB feelings and vice versa (Dare et al., 1994).

In spite of the lack of intercorrelational association of SB and OB, significant positive correlation between fathers' and mothers' scores on the components scales of the SOBS reveals a similar attitude of parents in their relationship with their daughters. This is further supported by the same positive correlation between fathers' and mothers' scores on the EE scales, which suggest that the similarity of attitude between the parents is not just a measure of their personality attributes, but a reflection of their relationships with their daughters, as well as possibly between themselves



(Szmukler et al, 1985).

### **5.3.D.3. Clinical Features of the Patients and Family Factors Associated with the SOBS**

Family self- and other-blaming attitudes concerning the patients' eating problems were significantly associated with some of the patients' clinical factors. Mothers' feelings of SB were negatively correlated with the patients' BMI. Mothers' feelings of OB were also negatively associated with the patients' BMI.

Patients' feelings of SB were negatively correlated with their age at onset of eating disorder. The association between blaming others and psychological impairment among families with an eating disorder patient is consistent with many other reports on different diagnostic populations (Affleck et al., 1982c; Bulman & Wortman, 1977; Taylor et al., 1984; Timko & Janoff-Bulman, 1982).

Eating disorder patients who met the PAS criteria for at least one personality disorder showed significantly more self-blaming feelings than did those without personality disorder. They were also more likely to show other-blaming feelings than did those without PD.

The potential role of personality factors could be considered as general propensities to internalize/externalize blame. For example, paranoid dynamism leads to the transference of blame to others. Transfer of blame may also be an attempt by the patient to have someone else experience the blameworthiness that she fears. Blaming others for one's problem might also reflect the use of defensive projection.

Personality-related explanation does not mean that individuals with an internalizing style will

always blame themselves or that externalizing will always blame others. Rather, they will give prominence to different aspects of the situation. Once someone does blame another person, whether because of personal inclination, situational factors, or their interaction, distress and dysfunction may be produced at two levels, the intraindividual and the interpersonal. The role of personality factors in attribution of self- and other-blame further supports the clinical usefulness of “characterological/behavioural” classification of self-blame (Janoff-Bulman, 1979).

In accordance with findings concerning the association between OB and poor adjustment (see Tennen & Affleck, 1990), greater feelings of OB among PD patients could be considered as a result of their personality problem. It seems reasonable that personal characteristics influence other-blaming attitudes as they were shown to have more interpersonal distrust and problems.

Relatives of PD patients did not differ in terms of OB, while they did differ in terms of SB; that is, they blamed themselves more than did relatives of patients without PD. This finding provides support to a potential role for attributional search in the SB/perceived-severity link (Wong & Weiner, 1981). One possible explanation of this finding is that families who perceived their daughter’s eating problem coexisting with personality problems as more severe became further distressed which, subsequently, led to more extensive causal searches. A more extensive search could produce some form of SB.

The same explanation could be introduced for SB feelings among PD patients themselves. Patients who perceive their eating disorder in conjunction with PD as more severe become further distressed. Their distress, in turn, leads to more extensive causal searches which can provide SB feelings. Our data, however, provide limited support for the latter interpretation.

Interactional patterns of the SOBS and PRSS variables revealed that patients who blamed themselves tended to be more disclosing than those who blamed others. It is most likely that the former group of patients feel more responsible for their problems. Taking responsibility, consequently, affects their response style to therapist and therapy and leads to more SD and EEn. This finding explains the positive function of SB.

In contrast, those who blamed others (probably through bypassing the responsibility) showed difficulty to disclose themselves and engage in therapy. This finding explains the negative function of OB. The strong associations of both OB and SD to outcome, further support this negative function.

The results clarify the relationship between the SOBS scales and family EE scales. As hypothesized, family (mothers and fathers) blaming patients was positively associated with family critical comments and hostility, and negatively related to family warmth.

Patients blaming mothers correlated with mothers' CC and EOI, while patients' blaming fathers associated with fathers' CC, but not with fathers' EOI. The association between patients blaming parents and parents' CC could be explained by an interactional function of OB-CC. It is possible that parents respond to the patients' OB by blaming the patients. This is also consistent with previous findings (see Tennen & Affleck, 1990) in which OB was found to be associated with poor adjustment.

The association between patients blaming mother and mothers' EOI reflects a quality of mother-daughter relationship in which an overinvolved relationship between mother and daughter with

eating disorder (presented by EOI) possibly plays a key role in the development of both mothers' SB and patients' blaming mother.

The main characteristic of an overclose system, a lack of individuation and clear boundaries between subsystems, would explain the way SB alters to OB and vice versa, as well as their relation to CC. Accordingly, each part of the system blames him/herself as well as the other part. Other-blame increases feelings of guilt and SB on the one hand, and provokes more OB and CC on the other. Each of these factors interactionally affect one another. For example, mother's SB alters to mother blaming patient either because of the emotionally overinvolved quality of the relationship in general, or more specifically overprotectiveness (in high EE families) which has been broken by the patient's control over her eating behaviour (a sign of separation-individuation). Subsequently, the mother feels more guilty and again blames herself while receiving some blame from the patient. Criticism is the typical presentation of this interactional OB.

It was also shown that high EE relatives, especially mothers, express more self- and other-blaming attitudes than do low EE relatives. One can infer that the interactional SOBS pattern of high EE families is characterized by attack-counterattack communication style (e.g., patient blaming mother is followed by mother blaming patient), whereas that of low EE families is identified by an attack-defensive communication style (e.g., patient is blaming while mother is excusing herself).

A more systemic description of this finding to explain the interactional patterns of EE (EOI, CC) and SOBS (SB, OB) variables in high EE families is a "power struggle" in the relationship. Being "the property of her parent" (Bruch, 1979, p. 107), the patient has no self-control. In such cases,

the mother rewards compliance to her wishes, is overprotective, and is unable to allow separation from the child. She needs to have a submissive, perfect child as her own fulfilment. The issue of separation-individuation tends to threaten this relationship. The patient's control over her eating frustrates the mother who can not tolerate independence in her child. Lacking the ability to resolve the conflict, criticism and blaming the patient are most likely reactions. In response to these emotional reactions and to avoid rejection and loss of self, the frustrated patient steps back. The scenario will be dramatized by the mother, she feels guilty and blames herself which in turn, makes the patient feel guilty as well. As time goes by, this blaming dance which is entered around the power struggle can set off a vicious cycle presenting interactions between the SOBS and EE variables.

This interactional pattern further supports the association between attitudes and overt interactional behaviour of the relatives as an underlying assumption of both the SOBS and EE constructs.

### **5.3.E. PROGNOSTIC FACTORS**

#### **5.3.E.1. Prognostic Value of Family Expressed Emotion**

It has been shown that treatment outcome could be predicted by parental CC in a sample of eating disorder patients (Le Grange et al., 1992a; Szmukler et al., 1985; Van Furth et al., 1996). The prognostic value of family EE is further supported by the findings in this study. Levels of the mothers's CC in the poor outcome group were significantly higher than those of the rest of the outcome categories. A comparison between the two groups revealed significant differences in terms of the mothers' CC ratings for the two groups. Consistent with the data regarding the predictive value of EE in other diagnostic groups (e.g., Brown et al., 1962, 1972; Hooley et al., 1986; Leff & Vaughn, 1981; Vaughn & Leff, 1976a; Vaughn et al., 1984), this finding indicates

the direction of the relationship between family EE and treatment outcome.

EE has consistently and reliably been demonstrated as an important predictor of symptomatic relapse in patients diagnosed as schizophrenic and probably in patients with depression (Brown et al., 1972; Leff et al., 1982; Leff & Vaughn, 1980; Vaughn & Leff, 1976a). From this it would appear that looking at EE over time in relation to symptomatic relapse in a sample of eating disorder patients is an obvious subject for future research.

### **5.3.E.2. Prognostic Value of Personality Disorders**

It has been shown that negative outcome of treatment could be predicted by personality disorders in different clinical conditions including depression (Black et al., 1988; Charney et al., 1981; Frank et al., 1987; Pfohl et al., 1984, 1987; Pilkonas & Frank, 1988; Zuckerman et al., 1980; Weissman et al., 1978), anxiety (Favarelli & Albanesi, 1987; Mavissakalian & Hamman, 1987; Reich, 1988; Turner, 1987), neurotic disorders (Tyrer et al., 1983), drinking problems (Griggs & Tyrer, 1981), and eating disorders (Deter et al., 1992; Johnson et al., 1990; Rossiter et al., 1993 Sansone & Fine, 1992). The prognostic value of PD is further supported by the findings in this study. Patients who had not PD showed more progress during the course of therapy than those who suffered from PD. Using the same instrument, Fahy et al. (1993) found that PD did not predict poor response to treatment in a sample of bulimic patients. Small sample size did not allow us to control the eating disorder subgroups (AN, BN) and test the predictive value of PDs in BN patients. The discrepancy between our present findings and that of Fahy et al. (1993), however, could be explained in several ways. The first is that the duration of eating disorders in patients with PD in our sample is, on average, 3 years longer than that of patients with PD in Fahy's et al. (1993) study. The longer duration of eating disorders possibly through increasing PD

symptomatology could partly explain the difference between the results of the two studies. The second explanation is that type of therapies conducted in the studies are different. It has been shown that different eating disorder patients respond best to different types of therapies (e.g., Dare & Eisler, 1995; Russell et al., 1987).

There is an impression that eating disorder patients with different types of PD would probably respond better to different kinds of interventions. For example, an eating disorder patient with an internalizing type of personality would possibly benefit more from family therapy whereas a patient with externalizing personality might benefit best from Intensive individual psychotherapy. Our small sample size did not allow us to test the best treatment for each personality type. The question of what type of therapy is best for what type of PD remains to be investigated.

Closer examination of the data suggests that the relationship between PD and outcome should not be regarded as a simply direct relationship between the two factors. It is more likely that personality is just one of the predisposing factors that influence the individual's response to treatment. Family factors, personality function and mental state should be considered as three components influencing treatment outcome. High EE families, greater personality disturbances and greater mental state pathology are three components to the poor outcome. Further studies should address whether PDs have predictive value in the long-term course and outcome of eating disorders and whether there are important differences in the subcategories of personality disorders in determining outcome.

### **5.3.E.3. Prognostic Value of the PRSS**

The pattern of results indicates significant differences between the two outcome groups, poor vs.

the rest, both in terms of the PRSS levels and scales (SD and EEn). In both cases differences were significantly in the predicted direction. Patients with higher SD and EEn showed greater improvement in psychotherapy than did patients with lower SD and EEn. This is consistent with earlier findings (e.g., Staples et al., 1976), which found the level of self-disclosure was a significant predictor of subsequent success in therapy. The PRSS patterns reflect and possibly facilitate the development of therapeutic engagement during the course of psychotherapy.

Self-disclosure is thought to be instrumental in achieving a sense of identity. Through SD, the patient presumably attains and maintains the assumption or reassumption of responsibility for past and present behaviour. Taking responsibility in therapy will facilitate the patient's progress and directly affect the outcome of treatment. Our results showed that high levels of PRSS (SD, EEn) are predictive of outcome for different therapy types.

This is consistent with previous studies (e.g., Sloane et al., 1975a; Staples et al., 1976; Stiles & Sultan, 1979), which found successful treatment appears to be more a function of the patient rather than therapeutic interventions.

In contrast to this apparent equivalence of outcome, psychotherapists' interventions vary greatly and systematically depending on their theoretical orientations. There really are different ingredients in the different psychotherapies. This paradox has been very troubling because it suggests that it does not matter what the therapist does, an unpleasant conclusion for professional psychotherapists. One possible resolution is that patient disclosure represents a major restorative process that is common across psychotherapies, regardless of the therapist's techniques. The outcome equivalence may, in part, reflect this common core process. This resolution rests on the



observation that, despite the great variation in therapists' behaviour, patients' response styles are strikingly consistent across therapeutic styles. Thus patient disclosure is a prominent common ingredient of different psychotherapies.

The purpose of the study, however, was to investigate the patient-related factors, SD and EEn, associated with success in treatment than the technique of the therapist. We have, of course, no assurance that therapist-related factors and some other process factors, not measured may be critical for success.

Closer examination to investigate differential effectiveness of different psychotherapies showed that SD and EEn might be the best predictors of the patients' progress in focal psychodynamic therapy. These results suggest that those individuals who are able to disclose themselves to the therapist and to engage more in the process of therapy might benefit most from this form of therapy that emphasizes the patient-therapist relationship and focuses on what the patient brings in therapy. Within the limits of this study, therefore, it is safe to conclude that the outcome of treatment is influenced by interaction of both patient-related (SD) and therapy-related (type of therapy) factors.

Having said this, it is also important to note that the data must be interpreted cautiously. Further research is needed to determine whether this distinction is clinically useful in designing effective treatment programs.

#### **5.3.E.4. Prognostic Value of the SOBS**

In a comprehensive review, Tennen and Affleck (1990), concluded that there is a reliable association between blaming another person and poor adjustment. The association between the

SOBS scales and poor outcome in psychotherapy is further supported by the findings in this study.

Across patients, blaming mothers was associated with poor outcome in psychotherapy, whereas across relatives specifically mothers, a sense of feeling self-blame for the patients' eating problem did so. Phillips's (1968) conception of turning against others should be considered as a possible psychodynamic explanation for the relationship between patients' blaming mothers and poor outcome in therapy. Phillips and Zigler (1964) and Phillips (1966) found that female patients whose emotional symptoms were characterized by turning against others required longer psychiatric hospitalization than those who symptomatically turned against themselves.

The next explanation, from an interactional point of view, is that blaming others is not only faced with an absence of validation from others, but is also followed by a similar reaction which subsequently, leads to more serious distortion of relationship. For example, when the patient blames the mother for her eating problems it may be difficult for the mother to be very supportive in the face of this attribution. However, even if the mother offers support, the patient may not be able to see it as support as long as she continues to view the mother as the cause of her problems. It is the patient's perception of the mother's behaviour, rather than her intent, that will best predict the patient's progress during the course of therapy. Thus OB may significantly comprise the potential benefits of support by decreasing the likelihood of: a) the other's support and/or b) the individual's ability to accept the support.

Finally, blaming others for an individual's eating problem can most likely be explained by avoidance in taking responsibility for the problem. When the patient does not accept responsibility for her problem, she may not be willing to participate in the process of psychotherapeutic change

as well.

The findings also revealed that AN patients, who blamed themselves more than blaming their parents, presented significantly more progress during the course of treatment than did BN patients who blamed their parents rather than blaming themselves. These findings give further support to the responsibility explanation.

Several possible explanations should be considered for the relationship between mothers' self-blame and patients' poor outcome in therapy. Clinical observations provide a useful explanation in which the mother's SB is associated with the patient's feelings of guilt and blame. When the mother blames herself because of the patient's eating problem, it is likely that the patient feels guilty. The patient's attempt to improve the situation is defeated in advance by the overinvolved quality of the mother-patient relationship which does not allow separation in the patient.

The dynamics of SB in an overclose relationship were discussed earlier. Here, another possibility is that the mother's SB may alter to blaming and criticising the patient which consequently, leads to more relationship conflicts. These relationship conflicts together with their psychological counterparts, feelings of guilt and blame, are obviously against therapeutic interventions and result in poor outcome.

The difference between mother's SB and patient's SB feelings in terms of their clinical presentation and predictive value also deserves comment. Mother's expression of SB seemed more genuine, spontaneous, negative, and emotionally charged whereas patient's SB seemed more reflective (to the mother's distress, hopelessness, and helplessness), and less negative and emotionally charged. As mentioned earlier, the mother's SB was also predictive of outcome

whereas the patient's SB was not.

The recognition that SB may be both predictive (mother's SB) and unpredictable (patient's SB) is a first step toward a conclusion that there are two different types of SB; one representing a spontaneous and self-initiated response, the other a reflective, other-initiated response - one might consider these to be primary and secondary SB.

The immediate clinical application of this new classification of SB has already been explained by its predictive power. Further work is obviously required to clarify the patterns that characterise the interaction between these two types of SB.

#### **5.3.E.5. The Relationship between Prognostic Factors**

Attention must be paid to the limitations of statistical analysis with regard to the nature of psychological mechanisms, particularly in relation to the interactional patterns of these mechanisms. For example, statistical exclusion of one variable from the analysis model by the other does not in itself demonstrate a complex psychological mechanism. It should be kept in mind that there is a degree of arbitrariness in statistical analyses of this kind.

Multivariate tests of predictive factors diminished the significant difference of the outcome categories for PD and patient blaming mother. It seems that these factors do not have either a strong or an independent effect on outcome. For this reason, caution is in order when addressing the question of whether personality and other blaming factors are predictive.

EEn was excluded from the equation by SD. Two possibilities should be considered. First, a high

correlation coefficient between SD and EEn could be responsible for the exclusion. When two factors are highly correlated, then it is most likely that including one of them excludes the other. A second but similar explanation is that the two factors measure one thing. This finding further gives support to the point that SD and EEn are two aspects of one underlying construct.

Mothers' CC was also excluded from the equation by the mothers' SB. Again the high correlation coefficient between the two factors may explain the exclusion. Similarly, it is also possible that the excluded factor does not have an independent effect on outcome.

The same explanations could be employed for decreasing the prognostic value of mothers' CC after it had entered the equation together with both SD and mothers' SB. It is plausible that the mothers' CC is associated to the outcome through the mothers' SB. Mediated by mother blaming patient, mothers' CC could be easily affected by high levels of feeling SB among mothers in this study. When SB is altered to OB then CC is its typical manifestation. The correlation between the mothers' CC and mothers' OB showed in the present sample, further supports this explanation.

## **CHAPTER 6**

### **IMPLICATIONS OF THE STUDY AND CONCLUDING STATEMENTS**

The results of this study of family-, personality-, and treatment-related factors in eating disorders using multiple measures and perspectives are quite compelling. Each of the therapeutic factors is meaningfully related to the quality of clinical outcome for eating disorder patients. This final chapter presents the clinical and theoretical implications of the results, followed by addressing the concluding statements drawn from the results.

#### **6.1. The Clinical Implications of the Study**

The results of the present study have several important clinical implications. The way individual and family factors interact and the way each of these factors affect the others indicated that there is a need for the clinician to carefully assess not only the eating behaviour of patients presenting for treatment, but also to consider more broadly the whole variety of individual and family factors.

Specific association between personality type and eating disorder subtypes may call for specific treatment modalities and have a modifying influence on the outcome of treatment.

In order to target evaluation and treatment more effectively, it is important to understand the links between specific family factors and specific personality characteristics in eating disorder patients. Findings of this study may help to explain the difficulties of therapeutic intervention with eating disorder patients who have a concurrent personality disorder (e.g., Deter et al., 1992; Johnson et al., 1990; Rossiter et al., 1993; Sansone & Fine, 1992).

Therapeutic intervention of those patients with personality disorder as well as high EE relatives may depend upon addressing specific family issues - criticism, hostility, self- and other-blame - in order to reduce the PD symptoms, either before or in conjunction with attempting to treat the eating disorder itself. The therapist is likely to need to use a variety of therapeutic strategies simultaneously (e.g., Stone, 1993; Westen, 1991) in order to deal with the different aspects of psychopathology most effectively.

Empirically, the analyses of the patient's response style to therapist and therapy provided further evidence that nonverbal, as well as verbal information are a valuable measure of therapeutic engagement and that correlation between Self-Disclosure (SD) and Emotional Engagement (EEn) as measured by the PRSS is an aspect of verbal/nonverbal consonance important for consistent therapeutic communication. Future research can investigate phases when there is a marked discrepancy between the levels of verbal and nonverbal measures. This may yield clinically useful information about when the therapist is distracted, uneasy with the intervention, or in some way in conflict.

The association of the PRSS to therapeutic outcome has been shown for the PRSS levels in different kinds of psychotherapy. On a practical level, SD and EEn can be taught or encouraged in the course of therapy. The more specially the intervention is targeted at the PRSS the stronger this association will be. The findings of this study indicate that the patient is moved toward her goal of successful therapy more effectively through her ability or willingness to SD and EEn. Regardless of the method used, the results indicated that patient's SD/EEn should be reinforced by the therapist to shape the patient's response style to more SD and EEn where appropriate.

Relatively little work has been done on the patient's response style to therapist and therapy in the last stages of treatment. Part of the reason for this may be the lack of interest in predictor variables near termination. Nonetheless, it seems quite possible that the quality of the relationship at this point may have important implications for the long-term effectiveness of the therapy. It is also possible that, over time, the patient's response style becomes more treatment specific; that is, some aspects of the patient's response style may be more critical for one form of therapy than another. According to this hypothesis, we may need to explore specific aspects of the patient's response style critical for the type of treatment, rather than looking at the overall patient's response style picture to determine quality of long-term outcome.

The analyses of the self- and other-blame patterns also revealed feelings of self- and other-blame to the same extent for both relatives and patients. This amplifies the necessity of inclusion of the patient in family care because it seems that she contributes in the same way to the style of interaction. However, it may well be that there are subtle differences in the way in which the relatives or the patient contribute to the SOBS pattern. During the treatment it is important that the therapist truly believes that neither the parents nor the patients are to blame or to held responsible for the psychiatric condition of the patient and any consequent interpersonal difficulties, in order to counter the feelings of guilt and self-blame, as well as other-blame.

However, SB can also have a positive function which reinforces a sense of self-responsibility and may increase the levels of SD/EE during the course of psychotherapy. On a practical level, therefore, the therapist can help a family to express such feeling if it is initiated by them.



## **6.2. The Theoretical Implications of the Study**

In terms of theoretical implications, the results of the present study raised a number of issues. Following the establishment of the role of family EE as a predictor of treatment outcome, the question arises whether it predicts the relapse in eating disorders. Since this study did not evaluate long term follow-up results, positive answer to this question remains tentative at this stage. Clarification of this issue requires longitudinal study of the stability of EE and its relationship to relapse in a large sample of eating disorder patients. The EE construct may prove to be an important organizing factor in conceptualizing the relapse process and studying the course of eating disorders.

We need to know how changes in EE relate to changes in the patient. This is the second theoretical consideration, the mechanism by which family EE and patients' symptoms affect one another in an interactional manner. It is demonstrated that high critical attitude is related to poor outcome. It is also showed that symptomatic improvement could reduce level of negative attitudes, as well as increase in positive attitudes like warmth, but further research with a large sample is required to clarify underlying mechanisms of changes in both family EE and patients' symptoms.

Although the role of family critical comments as predictive of treatment outcome is well established, the value of the other scales of EE is less well defined. When applied in a family setting it might be useful in the elaboration of 'hypotheses' about how particular families function. Expressed emotion may thus provide a much needed reliable basis for describing patterns of family interaction.

The next theoretical issue is concerned with the nature and assessment of EE. Most studies of EE have obtained information about families by interviewing individual family members. The assumption underlying the EE construct is that there is an association between attitudes and overt interactional behaviour of the relatives. Otherwise it would be very difficult to explain how high EE relatives constitute a form of increased social stress for vulnerable individuals. An appropriate approach is to analyse systematically the behaviour of high and low EE relatives when interacting with the patient. Using the SCFI, this study showed remarkable potential for tapping family interaction. The contributions family members make about the causes of eating disorder in the patient may help us understand more about the nature of EE, and provides a more reliable basis for the assessment of EE as a family and interactional quality. This method of measuring EE provides researchers with a reliable instrument that has specific guidelines, which can be easily replicated. Further, this study provided some evidence for the predictive validity of SCFI in measuring family EE in families with an eating disorder patient.

In line with systemic thinking, it was indicated that one important implication both for theory and practice is to consider personality, mental state and family factors from an interactional point of view. In research, specially based on the pathoplastic hypothesis, coexistence of eating disorder and personality disorder can also assist in determining whether AN and BN are discrete and independent disorders or different features of the same disorder.

It is clear that a good start has been made in sorting out the relationship between personality disorders and the eating disorders. Nevertheless, much work needs to be done in two particular realms. First, the directions of association between these two phenomena should be further detected. Second, emphasis should be placed on determining clinically relevant subtypes of AN

and BN based upon distinctive personality constellations.

The analyses of the PRSS results revealed that individual variations in response style to therapist and therapy can be variously manifested in self-disclosure and emotional engagement expressed during therapy sessions. Although the importance of nonverbal aspects of disclosure have been emphasized, both for the therapist and the patient (e.g., Altman & Taylor, 1973; Argyle & Dean, 1965; Montgomery, 1981, 1984; Patterson, 1990; Shapiro, Krauss, & Truax, 1969), most studies of self-disclosure have obtained information about the patient's response style to therapist and therapy from verbal disclosure alone. In this study, the PRSS showed exceptional potential for employing both verbal and nonverbal aspects of the patient's disclosure in the course of psychotherapy sessions. The inclusion of nonverbal, as well as verbal behaviour, considerably expands the concept of self-disclosure and the range of interpersonal activities to be observed.

An aspect of the psychotherapy process that can be valuable to assess is the degree of consonance between verbal and nonverbal measures. As Doster and Nesbitt (1979) note: "When variables are used conjointly, their congruence or incongruence may present valuable information for research and treatment (p. 222)." Thus, the use of multivariate assessment devices should allow researchers to make more meaningful statements regarding patient's response style to therapist and therapy. Measuring the patient's response style to therapist and therapy by the PRSS provides researchers with a reliable instrument that has specific guidelines, which can be easily replicated. This study also provided some evidence for both the construct and predictive validity of the PRSS in measuring the patient's response style to therapist and therapy in eating disorder patients.

Truax (1961) and Truax and Carkhuff (1964, 1965) have noted that both the patient's and the

therapist disclosures play important roles in the successful outcome of therapy. The PRSS would be entirely appropriate for assessing patient-related factors. Therapist-related factors need to be scientifically studied as well, perhaps by employing methods that involve direct observation of therapist's management and therapist-patient interactions during the course of therapy. It is to be hoped that such studies will help us better understand the prognostic factors and further refine our strategies for treatment.

It appears that the concept of patient's response style is a useful model of some important features of successful therapy and may, in the long term, assist us in better understanding the process of patient change. Although many different types of outcome were successfully predicted, SD and EEn may be more strongly associated with certain types of outcomes than others. The identification of similarities between certain kinds of the patient's response style and specific therapy results are potentially important areas of research.

In future research, it will be feasible to measure specific components of the PRSS scales in therapy sessions, before and after therapy, and correlate these measures with changes in both patient's symptomatology and family relationships.

SD within a family dynamics perspective, holds many interesting possibilities for future research. In future investigations of self-disclosure patterns and family dynamics, as suggested by Chelune et al. (1979), primary need seems to be for an application of research methods to family dynamics theories. Hypotheses about communication and self-disclosure in families need to be explored systematically with sound research. Several major areas appear to require examination. First, theoretical formulations about family boundaries and self-disclosure need to be tested empirically.

Specifically, studies need to identify well-functioning, disengaged, and enmeshed families and measure the amount, content, and style of actual self-disclosure in both verbal and nonverbal aspects by each type of family during a family discussion and interview. Videotaping of the session and later rating of behaviour seems to be the most appropriate method.

The family dynamics literature addresses self-disclosure in the context of studying communication patterns among family members. Self-disclosure issues are therefore addressed implicitly rather than explicitly. From a family dynamics perspective, self-disclosure can be examined structurally or functionally. Structural analysis of family self-disclosure focuses on both the interactions within “subsystems” and on the “boundaries” that differentiate the family subsystems. Functional analysis of family communications emphasises how content is communicated. The most preferred style of disclosure is the congruent mode, in which verbal content and nonverbal cues match.

Finally, research on the relationship between gender and SD presents inconsistent findings. Jourard and Lasakow (1985) found that men revealed less about themselves than women. Further studies of SD, however, did not always find a sex difference (see Cozby, 1973; Goodstein & Reinecker, 1974). Some studies have even found greater disclosure by men than by women under certain circumstances, such as in the initial development of an opposite-sex relationship (Davis, 1978; Derlega et al., 1985; Stokes et al., 1981).

Ideas raised in this study have been tested only with female subjects and thus may not generalize to other populations; this issue of generalizability particularly calls for research with male subjects. What is most crucial is that gender-related differences and preferences in disclosure can have consequences for how well individuals engage in a therapeutic relationship with a therapist of

either sex. The contribution that gender of both therapist and patient plays in decision governing SD were not explored in this study. This would appear to be a fruitful area of future study.

The results of this thesis also provided strong evidence that SOBS is a reliable instrument to assess self- and other-blaming attitudes experienced by the patients and relatives in the course of a psychotherapy assessment. While the validity of the existing measures is questionable (Turnquist, Harvey, & Anderson, 1988) and none of them assesses self/other-blaming attributes in the context of a family relationship, in this study, the SOBS demonstrated remarkable potential for the assessment of self/other-blaming attitudes in the course of a family interview. This study also provided some evidence for both the construct and predictive validity of the SOBS.

Prognostic value of the SOBS was supported by the findings in this study. Across patients, blaming mothers was associated with poor outcome in psychotherapy, whereas across relatives specifically mothers, a sense of feeling self-blame for the patients' eating problem did so.

The new classification of SB, primary (self-initiated) and secondary (other-initiated) self-blame, provides a clearer picture of the predictive power of SOBS.

Clearly, what is most needed at the present time is a study relating SOBS to symptoms. We know from both this and earlier studies (Humphrey, 1988) that bulimic patients experience greater blame in their families than anorexics. However, there is no a prior reason why it might be related to specific symptoms or symptom combinations, and this issue deserves some attention from researchers in the future. If SOBS is, as suggested by the present as well as previous findings, a measure of the responsiveness of certain individuals to particular symptoms in patients, it should

also be an index which is sensitive to changes in the symptoms of those patients. If it is the case, one might expect that the SOBS measure would be relatively unstable over time given symptomatic improvement in the patients.

Further research in this area needs to compare SOBS ratings of families of eating disorder patients at T1 and T2. A more detailed investigation of the factors involved in SOBS change would give obvious benefit to any intervention programs expressly designed to modify SOBS attitudes.

Further, the relationship between SB and OB would appear to be a fruitful area of future study. Does SB that occurs with OB, for example, lose its adaptive value, or is it similar to SB that occurs without OB? Is OB that occurs without SB more or less maladaptive than OB that occurs with SB? In addition, longitudinal studies designed to tap the coping implications of these two types of blame would be important contributions to our understanding of the relationship between coping and attributional strategies.

### **6.3. Concluding Statements**

In general, the conclusion can be drawn that the parents in this eating disorder sample revealed low levels on the EE scales, critical comments, hostility, emotional over-involvement, and positive remarks. This is particularly clear when these results are compared with those of the schizophrenia studies.

In general, the EE findings in this study did not support the two constructs of the psychosomatic model, enmeshment and overprotectiveness, for families containing an eating disorder patient. It would be argued that low levels of criticism and hostility do not in themselves indicate a “lack of

conflict resolution” because of the equal possibility of opposite, that is, higher level of conflict and criticism which have been established in a group of these families. However, the results showed some agreement with Minuchin’s et al. (1975, 1978) description of rigidity. On the whole, it was concluded that higher level of EOI or the qualities of enmeshment and overprotectiveness could be well explained as a high level of “commitment” (Dare et al., 1994) or “concern” (Hooley, 1985) towards the patient, at least in those families with a problem who are not necessarily dysfunctional (Frude, 1990).

We have learned more about the attitude-behaviour relationship in families with different levels of EE. Expressed emotion is a construct of considerable importance to clinicians and researchers alike. The more we understand about its complex nature, the better able we shall be to help the large number of psychiatric patients and their families.

On the basis of this study, one can infer that a critical attitude of the relative is associated with specific interactional behaviours of the relatives, patients, and the whole family. Therefore, a better understanding of both the nature of EE construct and the family EE patterns can be gained when it is evaluated within the family relationships. This study has demonstrated that at least two factors - a family attitude (EE), and a patient attribute (symptomatology) - interact in some manner with the treatment settings in determining outcome of treatment.

As predicted, the results revealed that EE is sensitive to changes in the patient. It also became apparent that EE can predict the treatment outcome in isolation. However, its predictive value was reduced in combination with other factors such as mothers’ SB.



Finally, as suggested by others too, EE should not be viewed as just another way of blaming families of psychiatric patients. In order to clarify this notion, potentially positive aspects of high EE and negative aspects of low EE require new investigation.

It was shown that both AN and BN patients who require outpatient treatment seem to equally display a personality pathology necessitating special attention in the treatment setting. Characterological differences between anorexic and bulimic patients, namely bulimic tendency to be histrionic and to a lesser degree anorexic tendency to be anxious, were important aspects of the present investigation. This finding provides some evidence in support of the notion that the different eating patterns may be associated with particular personality tendencies and that individual personality attributes may influence the expression of a particular eating pattern. This finding lends support to the pathoplastic hypothesis of personality (Akiskal, 1983) and to a lesser degree confirms the predisposing hypothesis which considers personality to provide a characterological predisposition to the development of eating disorders (Akiskal, 1983).

Both PD and mother's CC were found to be associated with poor response to treatment in eating disorders. Long-term treatments of some PDs have been emphasised (Dahl & Merskey, 1981; Parloff & Dies, 1977; Waldinger & Gunderson, 1987) in response to inadequacy of short-term treatment. It was suggested that an interactional perspective on the treatment of PDs could be a rather beneficial alternative. This approach also provides information about the diagnosis, course, and treatment of eating disorders, and influences clinical practice by determining whether patients with PDs require differential treatment. The study of PDs in eating disorders requires longitudinal assessment in order to clarify whether PDs have predictive value regarding the long-term course and outcome of eating disorders.

Personality disorder is likely to be important both for treatment planning and for prognosis in eating disorders. The influence of PD on the course of mental state diagnoses is also important and need greater emphasis in clinical practice, particularly when assessing prognosis.

Finally, an independent predictive value of PDs for progress in therapy was not supported, however, the possibility of the association between PDs and poor outcome in therapy was clarified. As mentioned before, small sample size did not allow to test this hypothesis properly.

The PRSS was shown to be a very useful instrument in assessing the patient's response style to therapist and therapy in the course of a psychotherapy assessment interview. Current findings suggested that the quality of the PRSS, SD and EEn, in the initial stages of treatment is predictive of a significant proportion of the final outcome variance. The results provided strong evidence for PRSS reliability.

The association of PRSS to therapeutic outcome has been shown for PRSS levels in different kinds of psychotherapy. From this, one can conclude that the scale is a patient-related measure rather than a therapist- and/or therapy- related factor. This study also provided confirmation for the importance of the patient-related factors for success in treatment. However, it has been shown that the PRSS levels might be the best predictor of the patient's progress in focal analytic therapy. Therefore, interaction of both patient-related (PRSS) and therapy-related (type of therapy) factors, could be considered as the best determinant of outcome.

The results also demonstrated the empirical value of the SOBS and provided strong evidence that the SOBS is a reliable instrument to assess self- and other-blaming attitudes experienced by the

patients and relatives in the course of a psychotherapy assessment interview. The results confirmed a high level of feeling of self-blame among eating disorder patients and their relatives. It was shown that in a sample of eating disorder patients, feelings of blame specially blaming someone else has been associated with personality disorders and poorer psychological well-being. The SOBS variable was examined and shown to influence the outcome at the termination of treatment period. Patients blaming mothers and mothers' self-blame were found to be associated with poor outcome in psychotherapy. This result confirmed the psychodynamic hypothesis of "turning against others" (Phillips, 1968) as a possible explanation for the relationship between patients blaming mothers and poor outcome in therapy.

The findings of the present study demonstrated two different communication styles between relatives and eating disorder patient. High EE families are characterized by attack-counterattack communication style, whereas low EE families are defined by attack-defensive communication style. The interactional pattern of EE (EOI, CC) and SOBS (SB, OB) variables explained by "power struggle" in the relationship of mother-daughter.

It was concluded that different clinical presentations of SB feelings which have different predictive value could be understood as two types of SB, primary (self-initiated) and secondary (other-initiated).

Supporting our hypothesis, the relationship between SOBS and EE scales for parents was clarified in two ways: (a) OB was positively associated with CC and HOS, and negatively related to warmth; (b) SB was positively associated with EOI (mothers).

Further attempts need to be made to understand the interaction between EE measures and SOBS measures. This would expand our understanding of the underlying mechanisms of these measures and their prognostic value for treatment outcome.

The results presented two interactional patterns between the SOBS and the PRSS variable:

1. Self-blame reinforces a sense of responsibility and consequently increases the patient's levels of PRSS.

2. Other-blame passes the responsibility on others and decreases the patient's levels of PRSS.

It was suggested that the former pattern explains the positive function of SB whereas the latter explains the negative function of OB.

Hopefully, this research will encourage both therapists and researchers to examine the many questions raised and perhaps stimulate other creative uses of both the PRSS and the SOBS to investigate family-personal factors important for treatment interventions. In my opinion, studies using such intensive research methodologies have the potential to add substantially to the literature, especially when their results complement those employing alternative empirical approaches.

## REFERENCES

- Abramson, L. Y., & Sackeim, H. A. (1977). A paradox in depression. Uncontrollability and self-blame. *Psychological Bulletin*, 84, 838-851.
- Affleck, G., Allen, D., McGrade, B. J., & McQueeney, M. (1982) Maternal causal attributions at hospital discharge of high risk infants. *American Journal of Mental Deficiency*, 86, 575-580. Affleck, G., Allen, D. A., Tennen, H., McGrade, B. J., & Ratzan, S. (1985) Causal and control cognitions in parent coping with a chronically ill child. *Journal of Social and Clinical Psychology*, 3, 369-379.
- Affleck, G., McGrade, B. J., Allen, D. A., & McQueeney, M. (1985). Mothers' beliefs about behavioral causes for their developmentally disabled infant's condition: What do they signify? *Journal of Pediatric Psychology*, 10, 293-303.
- Affleck, G., Pfeiffer, C., Tennen, H., & Fifield, J. (1987). Attributional processes in rheumatoid arthritis. *Arthritis and Rheumatism*, 30, 927-931.
- Affleck, G., Tennen, H., Croog, S., & Levine, S. (1987a). Causal attribution, perceived benefits, and morbidity after a heart attack: An 8-year study. *Journal of Consulting and Clinical Psychology*, 55, 29-35.
- Affleck, G., Tennen, H., Croog, S., & Levine, S. (1987b). Causal attribution, perceived control, and recovery from a heart attack. *Journal of Social and Clinical Psychology*, 5, 339-355.
- Akiskal, H. S. (1984). Characterologic manifestations of affective disorders: Toward a new conceptualization. *Integrative Psychiatry*, May-June, 83-8.
- Akiskal, H. S., Hirschfeld, R. M. A., & Yerevanian, B. I. (1983). The relationship of personality to affective disorders: A critical review. *Archives of General Psychiatry*, 40, 801-10.
- Altman, I., & Taylor, D. A. (1973) *Social penetration: The development of interpersonal*

*relationships*. New York: Holt, Rinehart & Winston, 1973.

American Psychiatric Association. (1987). *Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Revised (DSM-III-R). Washington, DC: APA

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. (DSM-IV). Washington, DC: APA

Ames-Frankel, J., Devlin, M. J., Walsh, B. T., Strasser, T. J., Sadik, C., Oldham, J. M., & Roose, S. P. (1992). Personality disorder diagnoses in patients with bulimia nervosa: Clinical correlates and changes in treatment. *Journal of Clinical Psychiatry*, 53, 90-96.

Archer, R. L., & Earle, W. B. (1983). The interpersonal orientations of disclosure. In: P. B. Paulus (Ed), *Basic Group Processes* (pp. 289-314). New York: Springer-Verlag.

Argyle, M., & Dean, J. (1965). Eye-contact, distance and affiliation. *Sociometry*, 28, 289-304.

Auld, F., & Wilkinson, W. C. (1973). Openness and Awareness of Communication During Psychotherapy. *Proceedings of the 81st Annual Convention of the American Psychological Association*, 8, 487-488.

Axtell, B., & Cole, C. W. (1971). Repression-sensitization response mode and verbal avoidance. *Journal of Personality and Social Psychology*, 18, 133-137.

Barrelet, L., Ferrero, F., Szigethy L., Giddey, C. & Pellizzer, G. (1990). Expressed emotion and first-admission schizophrenia: Nine-month follow-up in a French cultural environment. *British Journal of Psychiatry*, 156, 357-62.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive Therapy for Depression*. New York: Guilford Press.

Beck, A. T., Steer, R. A., & Garbin, M.C. (1988). Psychometric properties of the Beck Depression Inventory: twenty-five years of evaluation. *Clinical Psychology Review*, 8, 77-100.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for

- measuring depression. *Archives of General Psychiatry*, 4, 561-571.
- Bemis, K. M. (1978). Current approaches to the etiology and treatment of anorexia nervosa. *Psychological Bulletin*, 85, 593-617.
- Benedek, T. (1936). Dominant ideas and their relation to morbid cravings. *International Journal of Psychoanalysis*, 17, 40-56.
- Berg, H. & Derlega, V.J. (1987). Themes in the study of self-disclosure. In: V.J. Derlega and J. Berg (Eds), *Self-Disclosure: Theory, Research, and Therapy*. New York: Plenum Press.
- Berkowitz, R. (1987). Rating expressed emotion from initial family therapy sessions (a pilot study). *Journal of Family Therapy*, 9, 27-37.
- Beumont, P. J. V., Beardwood, C. J., & Russell, G. F. M. (1972). The occurrence of the syndrome of anorexia nervosa in male subjects. *Psychological Medicine*, 2, 216-231.
- Binswanger, L. (1959). The case of Ellen West. In: R. May, E. Angel, & H.F. Ellenberger (eds.) *Existence: A New Dimension in Psychiatry and Psychology*. New York: Basic Books.
- Blau, B. A. (1953). A Comparison of More Improved with Less Improved Clients Treated by Client-Centered Methods. In W. U. Snyder (Ed.), *Group Report of a Program of Research in Psychotherapy*. University Park: Psychotherapy Research Group, Pennsylvania State University.
- Blau, T. H. (1950). Report on a method of Predicting Success in Psychotherapy. *Journal of Clinical Psychology*, 6, 405-406.
- Bledin, K. D., MacCarthy B., Kuipere L., & Woods R. T. (1990). Daughters of people with dementia-Expressed emotion, strain and coping. *British Journal of Psychiatry*, 157,221-7.
- Blitzer, J. R., Rollins, N., & Blackwell, A. (1961). Children who starve themselves: Anorexia nervosa. *Psychosomatic Medicine*, 23, 269.
- Braaten, L. J. (1961). The Movement from Non-Self to Self in Client-Centered Psychotherapy.

*Journal of Counseling Psychology*, 8, 20-24.

Brown, G. W., Birley, J. L. T., & Wing, J. K. (1972). Influence of family life on the course of schizophrenic disorders: A replication. *British Journal of Psychiatry*, 121, 241-58.

Brown, G. W., Carstairs, G. M., & Topping, G. (1958). Post hospital adjustment of chronic mental patients. *Lancet*, ii, 685-9.

Brown, G. W., Monck, E. M., Carstairs, G. M., & Wing, J. K. (1962). Influence of family life on the course of schizophrenic illness. *British Journal of Preventive and Social Medicine*, 16, 55-68.

Brown, G. W. & Rutter, M. (1966). The measurement of family activities and relationships: a methodological study. *Human Relations*, 19, 241-263.

Bruch, H. (1961). Conceptual confusion in eating disorders. *Journal of Nervous and Mental Disease*, 133, 46-54.

Bruch, H. (1973). *Eating disorders: Obesity, anorexia nervosa and the person within*. New York: Basic Books.

Bruch, H. (1979). Anorexia Nervosa. In: R.J. Wurtman, & J.J. Wurtman (eds.), *Nutrition and the Brain*. New York: Raven Press.

Buhrich, N. (1981). Frequency of presentation of anorexia nervosa in Malaysia. *Australian and New Zealand Journal of Psychiatry*, 15, 153-155.

Bulman, R. J., & Wortman, C. B. (1977). Attributions of blame and coping in the "real world": Severe accident victims react to their lot. *Journal of Personality and Social Psychology*, 35, 351-363.

Burhenne, D., & Mirels, H. (1970). Self-disclosure in self-descriptive essay. *Journal of Consulting and Clinical Psychology*, 35, 409-413.

Calam, R., Waller, G., Slade, P., & Newton, T. (1990). Eating disorders and perceived



relationships with parents. *International Journal of Eating Disorders*, 9, 479-485.

Casper, R. C., Eckert, E., Halmi, K., Goldberg, S. C., & Davis, J. M. (1980). Bulimia: its incidence and clinical importance in patients with anorexia nervosa. *Archives of General Psychiatry*, 37, 1030-35.

Certner, B. C. (1973). "The Exchange of Self-Disclosures in Same-Sexed Groups of Strangers." *Journal of Consulting and Clinical Psychology*, 40, 292-297.

Chelune, G. J. (1975a). Self-disclosure: An elaboration of its basic dimensions. *Psychological Reports*, 36, 79-85.

Chelune, G. J. (1975b). "Sex Differences and the Relationship Between Repression-Sensitization and Self-Disclosure." *Psychological Reports*, 37, 920.

Chelune, G. J. (1975c). "Studies in the Behavioural and Self-Report Assessment of Self-Disclosure." Unpublished doctoral dissertation, University of Nevada at Reno.

Chelune, G.J. (1976a). A multidimensional look at sex and target differences in disclosure. *Psychological Reports*, 39, 259-62.

Chelune, G. J. (1976b). Reactions to male and female disclosure at two levels. *Journal of Personality and Social Psychology*, 34, 1000-1003.

Chelune, G. J. (1978). Nature and assessment of self-disclosing behavior. In: P. McReynolds (Ed.) *Advances in Psychological assessment* (Vol. 4). San Francisco: Jossey-Bass.

Chelune, G. J. (1979). *Self-disclosure*. San Francisco: Jossey-Bass.

Cozby, P. C. (1973). Self-disclosure: A literature review. *Psychological Bulletin*, 79, 73-91.

Craig, W. R. (1973). The Effects of Cognitive Similarity Between Client and Therapist upon the Quality and Outcome of the Psychotherapy Relationship. *Dissertation Abstracts International*, 34, 1272B.

Crisp, A. H. (1965). Some aspects of the evolution presentation and follow-up of anorexia

nervosa. *Proc R Soc Med*, 58, 814-20.

Crisp, A. H. (1970). Anorexia nervosa: feeding disorder, nervous malnutrition or weight phobia?

*World Review of Nutrition and Diet*, 12, 452-504.

Crisp, A. H. (1979). Fatness, metabolism and sexual behaviour. In: L. Carenza & L. Zichella

(Eds), *Emotion and Reproduction* (pp. 215-237). London: Academic Press.

Crisp, A. H., Hsu, L. K. G. & Stonehill, E. (1979). Personality, body weight and ultimate

outcome in anorexia nervosa. *Journal of Clinical Psychiatry*, 40, 332-4.

Crisp, A. H., Palmer, R. L., & Kalucy, R. S. (1976). How common is anorexia nervosa? A

prevalence study. *British Journal of Psychiatry*, 128, 549-554.

Crisp, A. H., & Toms, D. A. (1972). Primary anorexia nervosa or weight phobia in the male.

*British Medical Journal* i, 334-338.

Croog, S., & Levine, S. (1982). *Life after a heart attack: Social and Psychological factors eight*

*years later*. New York: Human Sciences Press.

Dally, P. (1969). *Anorexia Nervosa*. London: William Heineman Medical Books.

Dare, C. (1985). The family therapy of anorexia nervosa. *Journal of psychiatric research*, 19,

435-443.

Dare, C. (1992). Family scapegoating: an origin for hating. In: V. Varma (ed.), *Children Who*

*Hate*. London: David Fulton.

Dare, C. & Eisler, I. (1995). Family therapy. In: G. Szmukler, C. Dare, J. Treasure, *Handbook*

*of Eating Disorders: Theory, Treatment and Research*. Chichester: John Wiley & Sons.

Dare, C., Eisler, I., Russell, G. F. M., & Szmukler, G. (1990). The clinical and theoretical impact

of a controlled trial of family therapy in anorexia nervosa. *Journal of Marital and Family*

*Therapy*, 16, 39-57.

Dare, C. Le Grange, D., Eisler, I. & Rutherford, J. (1994). (in press) Redefining the

psychosomatic family: family process of 26 eating disorder families. *International Journal of Eating Disorders*, 16, 211-226.

Davis, J. D. (1978). When boy meets girl: Sex roles and the negotiation of intimacy in an acquaintance exercise. *Journal of Personality and Social Psychology*, 36, 684-692.

Davis, J. D., & Sloan M. L. (1974). The basis of interviewee matching and interviewer self-disclosure. *British Journal of Social and Clinical Psychology*, 13, 359-367.

Dent, H. R., & Salkovskis, P. M. (1986). Clinical measures of depression and obsessionality in non-clinical populations. *Behaviour Research and Therapy*, 24, 689-91.

Derlega, V. J., & Chaikin, A. L. (1977). Privacy and self-disclosure in social relationships. *Journal of Social Issues*, 33(3), 102-115.

Derlega, V. J., & Grzelak, J. (1979). Appropriateness of self-disclosure. In G. Chelune (Ed.), *Self-disclosure: Origins, patterns, and implications of openness in interpersonal relationships*. San Francisco: Jossey-Bass, 1979.

Derlega, V. J., Winstead, B. A., Wong, P. T. P., & Hunter, S. (1985). Gender effects in an initial encounter: A case where men exceed women in disclosure. *Journal of Social and Personal Relationships*, 2, 25-44.

Diem, K., & Lentner, C. (1970). *Geigy Scientific Tables*. Basel, Switzerland, JR Geigy, p. 711.

Dinicola, V. F. (1990). Anorexia Multiforme: self-starvation in historical and cultural context. II: Anorexia nervosa as a culture-reactive syndrome. *Transcultural Psychiatric Research Review*, 27, 245-286.

Dorr-Zegers, O. (1972). *Revista Chilena de Neuropsiquiatria*, 11, 27-41. English teanslated (1994): About a particular type of oral perversion in the female: Hyperphagia followed by vomiting. *International Journal of Eating Disorders*, 16, 117-132.

Doster, J. A. (1971). "The Disclosure Rating Scale." Unpublished rating manual, University of

Missouri.

- Doster, J. A., & Strickland, B. R. (1969). Perceived childrearing practices and self-disclosure patterns. *Journal of Consulting and Clinical Psychology, 33*, 382.
- Duddle, M. (1973). An increase of anorexia nervosa in a university population. *British Journal of Psychiatry, 123*, 711-712.
- Ehrlich, H. J., & Graeven, D. B. (1971). Reciprocal self-disclosure in a dyad. *Journal of Experimental Social Psychology, 7*, 389-400.
- Eisler, I. (1993). Family therapy for anorexia nervosa. In: S. Moorey and M. Hodes (eds) *Psychological Treatments in Human Disease and Illness*. London: Gaskill.
- Eisler, I. (1995). Family models of eating disorders. In: G. Szumukler, C. Dare, J. Treasure, *Handbook of Eating Disorders: Theory, Treatment and Research*. Chichester: John Wiley & Sons.
- Eisler, I., & Szumukler, G.I. (1985). Social class as a confounding variable in the eating attitudes test. *Journal of Psychiatric Research, 19*, 171-176.
- Eisler, I. & Szumukler, G.I., & Dare, C. (1985). Systematic observation and clinical insight - are they compatible? *Psychological Medicine, 15*, 173-188.
- Ellis, A. (1959). "Rationalism and its Therapeutic Applications." *Annual of Psychotherapy, 1*, 55-64.
- Fahy, T. (1991). Personality and treatment response in bulimia nervosa. presented at Les Troubles des conduites Alimentaires: Symposium International, Paris.
- Fahy, T. A., Eisler I., & Russell, G. F. M. (1993). Personality disorder and treatment response in bulimia nervosa. *British Journal of Psychiatry, 162*, 765-70.
- Fairburn, C. G. (1984). Bulimia: Its epidemiology and management. In: A.J. Stunkard & E. Stellar (Eds.), *Eating and Its Disorders*. New York: Raven Press, pp. 235-58.

- Fallon, B. A., Walsh, B. T., Sadik, C., Saoud, J. B., & Lukasik, V. (1991). Outcome and clinical course in inpatient bulimic women: A 2- to 9-year follow-up study. *Journal of Clinical Psychiatry*, 52, 272-278.
- Fenwick, S. (1880). *On atrophy of the stomach and on the nervous affections of the digestive organs*. London: Churchill Foster.
- Fincham, F. D., Beach, S.R., & Baucom, D. H. (1987). Attribution processes in distressed and nondistressed couples: 4. Self-partner attribution differences. *Journal of Personality and Social Psychology*, 52, 739-748.
- Fishmann-Havstad, L., & Marston, A. R. (1984). Weight loss maintenance as aspect of family emotion and process. *British Journal of Clinical Psychology*, 23, 265-71.
- Flanagan, D. A. J., & Wagner, H. L. (1991). Expressed emotion and panic-fear in the prediction of diet treatment compliance. *British Journal of Clinical Psychology*, 30, 231-40
- Fleiss, J. L. (1981). *Statistical Methods for Rates and Proportions*, 2nd ed. New York: Wiley.
- Frey, D., Rogner, O., Schuler, M., & Korte, C. (1985). Psychological determinants in the convalescence of accident patients. *Basic and Applied Social Psychology*, 6, 317-328.
- Fromm, E. (1959). *The Sane Society*. New York: Holt, Rinehart and Winston.
- Frude, N. (1990). *Understanding Family Problems*. Chichester: John Wiley & Sons.
- Garfinkel, P. E., & Garner, D. M. (1982). *Anorexia Nervosa: A Multidimensional Perspective*. New York: Brunner/Mazel.
- Garfinkel, P. E., Moldofsky, H., & Garner, D. M. (1980). The heterogeneity of anorexia nervosa. *Archives of General Psychiatry*, 37, 1036-40.
- Garner, D. M., & Garfinkel, P. E. (1980). Socio-cultural factors in the development of anorexia nervosa. *Psychological Medicine*, 10, 647-656.
- Garner, D. M., Garfinkel, P. E., & Bemis, K. (1982). A multidimensional psychotherapy for

anorexia nervosa. *International Journal of Eating Disorders*, 1, 3-46.

Garner, D. M., Olmsted, M. P., & Garfinkel, P. E. (1983). Does anorexia nervosa occur on a continuum? Subgroups of weight-preoccupied women and their relationship to anorexia nervosa. *International Journal of Eating Disorders*, 2, 11-20.

Garner, D. M., Rockert, W., Olmsted, M. P., Johnson, C., & Coscina, D.V. (1985). Psychoeducational principles in the treatment of bulimia and anorexia nervosa. In: D.M. Garner & P.E. Garfinkel (Eds.), *Handbook of Psychotherapy for Anorexia Nervosa and Bulimia*. New York: Guilford Press, pp. 513-72.

Gartner, A. F., Marcus, R. N., Halmi, K., & Loranger, A. W. (1989). DSM-III-R personality disorders in patients with eating disorders. *American Journal of Psychiatry*, 146, 1585-1591.

Gendlin, E. T., & others (1968). Focusing Ability in Psychotherapy, Personality and Creativity. In J. M. Shlien (Ed.), *Research in Psychotherapy*. Vol. 3. Washington, D.C.: American Psychological Association.

Gilhooley, M. L. M., & Whittick, J. E. (1989). Expressed emotion in caregiver of the dementing elderly. *British Journal of Medical Psychology*, 62, 265-72.

Gittleson, N. L. (1966). The effect of obsessions on depressive psychosis. *British Journal of Psychiatry*, 112, 253-9.

Goetz, P. L., Succop, R. A., Reinhart, J. B., & Miller, A. (1977). Anorexia nervosa in children: A follow-up study. *American Journal of Orthopsychiatry*, 47, 597-603.

Goodstein, L. D., & Reinecker, V. M. (1974). Factors affecting self-disclosure: A review of the literature. In: B. A. Maher (Ed), *Progress in Experimental Personality Research*, 7 (pp. 49-77). New York: Academic Press.

Greedharry, D. (1987). Expressed emotion in the families of the mentally handicapped: A pilot

study. *British Journal of Psychiatry*, 150, 400-402.

Gull, W.W. (1874). Anorexia nervosa (apepsia hysterica, anorexia hysterica). *Trans. Clin. Soc. Lond.*, 7, 222-228.

Gull, W. W. (1988). Anorexia Nervosa. *Lancet*, 1, 516.

Gwirstman, H. E., Roy-Byrne, P., Yager, J., & Gerner, R. H. (1983). Neuroendocrine abnormalities in bulimia. *The American Journal of Psychiatry*, 140, 559-63.

Haley, J. (1959). The family of the schizophrenic: A mod. *Journal of Nervous and Mental Disease*, 129, 357-374.

Halmi, K. A. (1974). Anorexia nervosa: Demographic and clinical features in 94 cases. *Psychosomatic Medicine*, 36, 18-26.

Halmi, K. A. (1992). Psychobiology and Treatment of Anorexia Nervosa and Bulimia Nervosa. Washington, DC: APP, Inc.

Havsted, L. F. (1979). Weight loss and weight loss maintenance as aspects of family emotional processes. Unpublished doctoral thesis, University of Southern California.

Healy, J. M., Stewart, A. J., & Copeland, A. P. (1993). *Personality and Social Psychology Bulletin*, 19, 279-289.

Heinemann, A., W., Bulka, M., & Smetak, S. (1988). Attributions and disability acceptance following traumatic injury: A replication and extension. *Rehabilitation Psychology*, 33, 195-206.

Henderson, M., & Freeman, C. P. L. (1987). A self rating scale for bulimia, The BITE. *British Journal of Psychiatry*, 150, 18-24.

Herzog, D. B., Keller, M. B., Lavori, P. W., Kenny, G. M., & Sacks, N. R. (1992). The prevalence of personality disorders in 210 women with eating disorders. *Journal of Clinical Psychiatry*, 53, 147-152.

- Herzog, D. B., Keller, M. B., Sacks, N. R., Yeh, C. J., & Lavori, P. W. (1992). Psychiatric comorbidity in treatment seeking anorexics and bulimics. *Journal of the American Academy of Child and Adolescent Psychiatry*, 31, 810-818.
- Hirschfeld, R. M. A., & Klerman, G. L. (1979). Personality attributes and appetite disorders. *American Journal of Psychiatry*, 135, 67-70.
- Hodgson, R. J., & Rachman, S. (1979). Obsessional-compulsive complaints. *Behaviour Research and Therapy*, 15, 389-95.
- Hogarty, G. E., Anderson, C. M., Reiss, D. J., Kornblith, S. J., Greenwald, D. P., Javna, C. D., & Madonia, M. J. (1986). Family Psychoeducational, Sociap Skills Training, and Maintenance Chemotherapy in the Aftercare Treatment of Schizophrenia - I. One-Year Effects of a Controlled Study on Relapse and Expressed Emotion. *Archieves of General Psychiatry*, 43, 633-642.
- Hooley, J. M. (1985). Expressed emotion: A review of the critical literature. *Clinical Psychology Review*, 5, 119-39.
- Hooley, J. M., & Hahlweg, K. (1986). The marriages and interaction patterns of depressed patients and their spouses: comparison of high and low EE dyads. In: M.J. Goldstein, I. Hand, & K. Hahlweg (eds.), *Treatment of Schizophrenia: Family Assessment and Intervention* (pp. 85-97). Berlin: Springer Verlag.
- Hooley, J. M., & Teasdale, J. (1989). Predictors of relapse in unipolar depressives, EE, marital distress and perceived criticism. *Journal of Abnormal Psychology*, 98, 229-35.
- Horowitz, L. M., Rosenberg, S. E., Baer, B. A., Ureno, G., & Villasenor, V. S. (1988). Inventory of interpersonal problems: psychometric properties and clinical applications. *Journal of Consulting and Clinical Psychology*, 56, 885-892.
- Horowitz, L. M., Rosenberg, S. E., Ureno, G., Kalehzan, B. M., & O'Halloran, P. (1989).



Psychodynamic formulation, consensual response method, and interpersonal problems.

*Journal of Consulting and Clinical Psychology*, 57, 599-606.

Humphrey, L. L. (1988). Relationships within subtypes of anorectic, bulimic, and normal families.

*J. Am. Acad. Child. Adoles. Psychiat.*, 27, 544-551.

Janoff-Bulman, R. (1979). Characterological versus behavioral self-blame: Inquiries into depression and rape. *Journal of Personality and Social Psychology*, 37, 1798-1809.

Janoff-Bulman, R. (1982). Esteem and control bases of blame: "Adaptive" strategies for victims versus observers. *Journal of Personality*, 50, 180-191.

Janoff-Bulman, R., Timko, C., & Carli, L. L. (1985). Cognitive biases in blaming the victim.

*Journal of Experimental Social Psychology*, 21, 161-177.

Joe, H. (1988). Extreme Probabilities for contingency tables under row and column independence

with application for Fisher's exact test. *Communications in Statistics A Theory and Methods*, 17, 3677-3685.

Johnson, C., Tobin, D., & Dennis, A. (1990). Difference in treatment outcome between borderline

and nonborderline bulimics at one-year follow-up. *International Journal of Eating Disorders*, 9, 617-27.

Jones, D. J., Fox, M. M., Babigian, H. M., & Hutton, H. E. (1980). Epidemiology of anorexia

nervosa in Monroe County, New York: 1960-76. *Psychosomatic Medicine*, 42, 551-558.

Jourard, S. M. (1959). Self-disclosure and other cathexis. *Journal of Abnormal and Social*

*Psychology*, 59, 428-431.

Jourard, S. M., & Richman, P. (1963). Disclosure output and input in college students. *Merrill-*

*Palmer Quarterly*, 9, 141-148.

Jourard, S. M. (1964). *The transparent self*. Princeton, N. J.: Van Nostrand.

Jourard, S. M. (1971). *The transparent self* (Rev. ed). New York: Van Nostrand Reinhold.

- Jourard, S. M., & Lasakow, P. (1958). Some factors in self-disclosure. *Journal of Abnormal and Social Psychology*, 56, 91-98.
- Jourard, S. M. & Resnick, J. L. (1970). The effect of high-revealing subjects on the self-disclosure of low-revealing subjects. *Journal of Humanistic Psychology*, 10, 84-93.
- Kaplan, M. F. (1967). Interview interaction of repressors and sensitizers. *Journal of consulting Psychology*, 31, 513-516.
- Karno, M., Jenkins, J. H., Silva, A. De la, Santana, F., Telles, S., Lopez, C., & Mintz, J. (1987). Expressed emotion and schizophrenic outcome among Mexican-American families. *Journal of Nervous and Mental Disease*, 175, 143-151.
- Katz J. L. (1985). Some reflections on the nature of the eating disorders. *International Journal of Eating Disorders*, 4, 617-626.
- Kavanagh, D. J. (1992). Recent developments in expressed emotion and schizophrenia. *British Journal of Psychiatry*, 160, 601-20.
- Kendell, R. E. & Discipio, W. J. (1968). Eysenck Personality Inventory scores of patients with depressive illness. *British Journal of Psychiatry*, 114, 767-70.
- Kendell, R. E., Hall, D. J., Hailey, A., & Babig, H. M. (1973). The epidemiology of anorexia nervosa. *Psychological Medicine*, 3, 200-203.
- Kennedy, S. H., McVey, G., & Katz R. (1990). Personality Disorders in anorexia nervosa and bulimia nervosa. *Journal of Psychiatric Research*, 24, 259-269.
- Kiecolt-Glaser, J. K., & Williams, D. A. (1987). Self-blame, compliance and distress among burn patients. *Journal of Personality and Social Psychology*, 53, 187-193.
- Kiesler, D. J. (1971). Patient Experiencing and Successful Outcome in Individual Psychotherapy of Schizophrenics and Psychoneurotics. *Journal of Consulting and Clinical Psychology*, 37, 370-385.

- Kiesler, D. J., Mathieu P., & Klein M. H. (1969). Process Movement in Therapy and Sampling Interviews. In C. R. Rogers, and others (Eds.), *The Therapeutic Relationship and Its Impact: A Study of Psychotherapy with Schizophrenics*. Madison: University of Wisconsin Press.
- Kinston, W. & Loader P. (1984). Eliciting whole-family interaction with a standardized clinical interview. *Journal of Family Therapy*, 6, 347-363.
- Kirtner, W. L., & Cartwright, D. S. (1958). Success and Failure in Client-Centered Therapy as a Function of Initial In-Therapy Behavior. *Journal of Consulting Psychology*, 22, 329-333.
- Klass, C. (1968). "The Effects of Experimenter Machiavellianism on Subject Self-Disclosure in the Initial Interview." *Dissertation Abstracts International*, 28, 3065-3066B.
- Kramer, S. (1974). A discussion of the paper by John A. Sours on "The anorexia nervosa syndrome." *International Journal of Psychoanalysis*, 55, 577-579.
- Lasegue, E. (1973). De l'anorexia hysterique. English translation in: M. R. Kaufman & M. Heiman (Eds), *Evolution of Psychosomatic Concepts*, New York: International University Press.
- Leff, J., & Vaughn, C. E. (1981). The role of maintenance therapy and relatives' expressed emotion in relapse of schizophrenia: A two year follow-up. *British Journal of Psychiatry*, 139, 102-104.
- Leff, J., & Vaughn, C. (1985). *Expressed Emotion in Families: Its Significance for Mental Illness*. Guilford: New York.
- Leff, J., Wig, N. N., Bedi, H., et al. (1990). Relatives' expressed emotion and the course of schizophrenia in Chandigarh: A two-year follow-up of a first contact sample. *British Journal of Psychiatry*, 156, 351-6.

- Leff, J., Wig, N. N., Ghosh, A., Bedi H., Menon, D. K., Kuipers, L., Korten, A., Ernberg, G., Day, R., Sartorius, N., & Jablensky, A. (1987). Expressed emotion and schizophrenia in North India. III. Influence of relatives' expressed emotion on the course of schizophrenia in Chandigarh. *British Journal of Psychiatry*, 151, 166-173.
- LeGrange, D. (1989). Anorexia nervosa and family therapy: A study of the changes in the individual and the family during the process of body weight restoration. Unpublished ph.D. Thesis, University of London.
- LeGrange, D., Eisler, I., Dare, C., & Hodes, M. (1992b). Family criticism and self starvation: a study of expressed emotion. *Journal of Family Therapy*, 14, 177-92.
- LeGrange, D., Eisler, I., Dare, C., & Russell, G. F. M. (1992c). Evaluation of family treatments in adolescent anorexia nervosa: a pilot study. *International Journal of Eating Disorders*, 12, 347-57.
- Lesser, L. I., Ashenden, B. J., Delruskey, M., & Eisenberg, L. (1960). Anorexia nervosa in children. *American Journal of Orthopsychiatry*, 30, 572-80.
- Levin, A. P., & Hyler, S. E. (1986). DSM-III personality Diagnosis in bulimia. *Comprehensive Psychiatry*, 47-53.
- Levin, A. P., Kahan, M., Lamm, J. B., & Spauster, E. (1993). Multiple personality in eating disorder patients. *International Journal of Eating Disorders*, 13, 235-9.
- Levinger, G., & Senn, D. (1967). Disclosure of feelings in marriage. *Merrill-palmer Quarterly*, 13, 237-249.
- Lorand, S. (1943). Anorexia nervosa: report of a case. *Psychosomatic Medicine*, 5, 282-292.
- Luborsky, L., Singer, B., Luborsky, L. (1975). Comparative of psychotherapies. *Arch Gen Psychiatry* 32:995.
- Mahon, N. E. (1982). The relationship of self-disclosure, interpersonal dependency, and life

hanges to loneliness in young adults. *Nursing Research*, 31, 343-347.

Major, B., Muller, P., & Hildebrandt, K. (1985). Attributions, expectations, and coping with abortion. *Journal of Personality and Social Psychology*, 48, 585-599.

Marce, L.-V. (1860). On a form of hypochondriacal delirium occurring consecutive to dyspepsia and characterized by refusal of food. *Journal of Psychological Med. Mental Pathol.* 13, 204-206.

Marks, M. N., Wieck, A., Seymour, A., Checkley, S. A., & Kumar, R. (1992). Women whose mental illnesses recur after childbirth and parentals' levels of expressed emotion during late pregnancy. *British Journal of Psychiatry*, 161, 211-216.

Marshall, V. G., Longwell, L., Goldstein, M. J., & Swanson, J. M. (1990). Family factors associated with aggressive symptomatology in boys with attention deficit hyperactivity disorder: A research note. *Journal of Child Psychology and Psychiatry*, 31, 629-36.

McClelland, L., Mynors-Wallis, L., Fahy, T., & Treasure, J. (1991). Saxual abuse, disordered personality, and eating disorders. *British Journal of Psychiatry*, 158, (suppl. 10) 63-8.

McCreadie, R. G. & Phillips, K. (1988). The Nithsdale schizophrenia survey. VII. Does relatives' high expressed emotion predict relapse? *British Journal of Psychiatry*, 152, 477-81.

McMillan, J. F., Gold, A., Crow T. J., et al. (1986). The Northwick Park study of first episodes of schizophrenia. IV. Expressed emotion and relapse. *British Journal of Psychiatry*, 148, 133-43.

McPeak, W. R. (1975). Family interaction as etiological factors in mental disorders: An analysis of the American Journal of Insanity, 1844-1848. *American Journal of Psychiatry*, 132, 1327-1329.

Mehrabian, A. (1972). *Non-Verbal Communication*. Chicago Aldine Atherton.

Mehta, C. R., & Patel, N. R. (1983). A network algorithm for performing Fisher's exact test in

r contingency tables. *Journal of the American Statistical Association*, 78, 427-434.

Meichenbaum, D. (1977). *Cognitive-Behavior Modification*. New York: Plenum Press.

Mendola, R. A., Tennen, H., Affleck, G., McCann, L., & Fitzgerald, T. (1990). Appraisal and adaptation among women with impaired fertility. *Cognitive Research and Therapy*, 14, 79-92.

Meyer, B., & Taylor, S. E. (1986). Adjustment to rape. *Journal of Personality and Social Psychology*, 50, 1222-1234.

Michalide, A., & Anderson, A. (1985). Subgroups of anorexia nervosa and bulimia: Validity and utility. *Journal of Psychiatric Research*, 19, 121-128.

Miklowitz, D. J., Goldstein, M. J., & Falloon, R. H. (1983). Premorbid and symptomatic characteristics of schizophrenics from families with high and low levels of Expressed Emotion. *Journal of Abnormal Psychology*, 92, 359-367.

Miklowitz, D. J., Goldstein, M. J., Nuechterlein, K. H., Synder, K. S., & Doane, J. A. (1986). Expressed emotion, affective style, lithium compliance, and relapse in recent onset mania. *Psychopharmacology Bulletin*, 22, 628-32.

Miklowitz, D. J., Goldstein, M. J., Nuechterlein, K. H., Synder, K. S., & Mintz, J. (1988). Family factors and the course of bipolar affective disorder. *Archives of General Psychiatry*, 45, 225-31.

Miller, L. C., Berg, J. H., & Archer, R. L. (1983). Openers: Individuals who elicit intimate self-disclosure. *Journal of Personality and Social Psychology*, 44, 1234-1244.

Miller, D. T., & Porter, C. A. (1983). Self-blame in victims of violence. *Journal of Social Issues*, 39(2), 139-152.

Minuchin, S. (1974). *Families and Family therapy*. Cambridge, Mass., Harvard University Press.

Minuchin, S., Rosman, B. L., & Baker, L. (1978). *Psychosomatic Families: Anorexia Nervosa*

*in Context*. Cambridge, Mass: Harvard University Press.

Minuchin, S. (1984). *Family Kaleidoscope*. Cambridge, MA: Harvard University Press.

Minuchin, S., Baker, L., Rosman, B. L., Liebman, R., Milnan, L., & Todd, T. (1975). A conceptual model of psychosomatic illness in children: Family organization and family therapy. *Archives of general Psychiatry*, 32, 1031-8.

Mitchell, K., & others (1973). *Antecedents to Psychotherapeutic Outcome*. NIMH Final Report, MH12306. Fayetteville: Arkansas Rehabilitation Research and Training Center, University of Arkansas.

Miyai, K., Yamamoto, T., Azokizawa, M., Ishibashi, K., & Kumahara, Y. (1975). Serum Thyroid hormones and thyrotropin in anorexia nervosa. *Journal of Clinical Endocrinology and Metabolism*, 40, 334-338.

Moline, R.A., Singh, S., Morris, A., & Meltzer, H.Y. (1985). Family and expressed emotion and relapse in schizophrenia in 24 urban American patients. *American Journal of Psychiatry*, 142, 1078-1081.

Montgomery, B. M. (1981). Verbal immediacy as a behavioral indicator of open communication content. *Communication Quarterly*, 30, 28-34.

Montgomery, B. M. (1984). Communication in intimate relationships: a research challenge. *Communication Quarterly*, 32, 318-325.

Morgan, H. G., & Hayward, A. E. (1988). Clinical assessment of anorexia nervosa. *British Journal of Psychiatry*, 152, 367-371..

Morgan, H. G., & Russel, G. F. M. (1975). Value of family background and clinical features as predictors of long term outcome in anorexia nervosa: A four-year follow-up study of 41 patients, *Psychological Medicine*, 5, 355-71.

Morton, R. (1694). *Phthysiologica: Or a Treatise of Consumptions*. London: S. Smith and B.

Walford.

Moulton, R. (1942). A psychosomatic study of anorexia nervosa including the use of vaginal smears. *Psychosomatic Medicine*, 4, 62-74.

Mowrer, O. H. (1961). *The crisis in psychiatry and religion*. New York: Van Nostrand.

Napier, A. Y. (1978). *The Family Crucible*. New York: Bantam Books.

Nielson, W. R., & MacDonald, M. R. (1988). Attributions of blame and coping following spinal cord injury: Is self-blame adaptive? *Journal of Social and Clinical Psychology*, 7, 163-175.

Norman, D. K., Blais, M. A., & Herzog, D. B. (1993). Personality characteristics of eating-disordered patients as identified by the Millon Clinical Multiaxial Inventory. *Journal of Personality Disorders*, 1-9.

Nuechterlein, K. H., & Dawson, M. E. (1984). A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin*, 10, 300-312.

Nuechterlein, K. H., Snyder, K. S., Dawson, M. E., Rappe, S., Gitlin, M., & Fogelson, D. (1986). Expressed emotion, fixed dose Fluphenazine decanoate maintenance, and relapse in recent-onset schizophrenia. *Psychopharmacology Bulletin*, 22, 633-639.

Orford, J., O'Reilly, P., & Goonatilleke, A. (1987). Expressed emotion and perceived family interaction in the key relatives of elderly patient with dementia. *Psychological Medicine*, 17, 963-70.

Palmer, R. L. (1979). Dietary chaos syndrome: A useful new term? *British Journal of Medical Psychology*, 52, 187-190.

Palmer, R. L. (1987). Bulimia: The nature of the syndrome, its epidemiology and its treatment. In: R. A. Boakes, D. A. Popplewell, & M. J. Burton (Eds), *Eating Habits: Food, physiology and learned behaviour* (pp. 1-23). Chichester: John Wiley.

Palmer, R. L., Oppenheimer, R., & Marshall, P. D. (1988). Eating disorder patients remember



their parnts: a study using the parental-bonding instrument. *International Journal of Eating Disorders*, 7, 101-106.

Parker, G., Johnston, P., & Hayward, L. (1988). Parental 'expressed emotion' as a predictor of schizophrenic relapse. *Archives of General Psychiatry*, 45, 806-13.

Parker, G., Tupling, H., & Brown, L.B. (1979). A parental bonding instrument. *British Journal of Medical Psychology*, 52, 1-10.

Patterson, M. L. (1990). Functions of non-verbal behaviour in social interaction. In H. Giles & W. P. Robinson (Eds), *Handbook of Language and Social Psychology* (pp. 101-120). Chichester, UK: John Wiley.

Pederson, D. M., & Breglio, V. J. (1968). Personality correlates of actual self-disclosure. *Psychological Reports*, 22, 492-501.

Peterson, C. (1979). Uncontrollability and self-blame in depression: Investigation of the paradox in a college population. *Journal of Abnormal Psychology*, 88, 620-624.

Peterson, C., Schwartz, S. M., & Seligman, M.E. P. (1981). Self-blame and depressive symptoms. *Journal of Personality and Social Psychology*, 41, 253-9.

Piran, N., Lerner, P., Garfinkel, P. E., Kennedy, S. H., & Brouillette, C. (1988). Personality disorders in anorexic patients. *International Journal of Eating Disorders*, 7, 589-599.

Pope, H.G., Frankenburg F.R., Hudson J.L., Jonas J., & Yurgelun-Todd, D. (1987). Is bulimia associated with borderline personality disorder? A controlled study. *Journal of Clinical Psychiatry*, 48, 181-4.

Pope, H. G., & Hudson, J. I. (1989). Are eating disorders associated with borderline personality disorder? A critical review. *International Journal og Eating Disorders*, 8, 1-9.

Prager, R. A. (1971). "The Relationship of Certain Client Characteristics to Therapist-Offered Conditions and Therapeutic outcome." *Dissertation Abstracts International*, 31, 5634-

5635B.

- Priebe, S., Wildgrube, C., & Muller-Oerlinghausen B. (1989). Lithium prophylaxis and expressed emotion. *British Journal of Psychiatry*, 154, 396-99.
- Powers, S. P., Covert, D. L., Brightwell, D. R., & Stevens, B. A. (1988). Other psychiatric disorders among bulimic patient *Comprehensive Psychiatry*, 29, 503-508.
- Reich, J. H., & Green, A. I. (1991). *The Journal of Nervous and Mental Disease*, 179, 74-82.
- Rogers, C. (1961) *On becoming a person*. Boston: Houghton-Mifflin.
- Rosen, I. (1957). The clinical significance of obsessions in schizophrenia. *J. Ment. Sci.*, 103, 773-85.
- Ross, J. L. (1977). Anorexia nervosa: An overview. *Bulletin of Meninger Clinic*, 41, 418-436.
- Rossiter, E. M., Agras, W. S., Telch, C. F., & Schneidre, J. A. (1993). Cluster B personality disorder characteristics predict outcome in the treatment of bulimia nervosa. *International Journal of Eating Disorders*, 13, 349-357.
- Russell, G. F. M. (1970). Anorexia nervosa: Its identity as an illness and its treatment. In *Modern Trends in Psychological Medicine* (ed. Price J.H.) Butterworths, London.
- Russell, G. F. M. (1979). Bulimia nervosa: An ominous variant of anorexia nervosa. *Psychological Medicine*, 9, 429.
- Russell, G. F. M. (1985). The changing nature of anorexia nervosa: an introduction to the conference. *Journal of Psychiatric research*, 19 (2/3), 101-109.
- Russell, G. F. M. (1995). Anorexia nervosa through time. In: G. Szumukler, C. Dare, J. Treasure, *Handbook of Eating Disorders: Theory, Treatment and Research*. Chichester: John Wiley & Sons.
- Russell, G. F. M., & Treasure, J. (1989). The modern history of anoraxia nervosa: an interpretayion of why the illness has changed. In: L.A. Schneider, S.J. Cooper, & K.A.

- Halmi (eds.) The Psychobiology of Human Eating Disorders: Principal and Clinical Perspectives. *Annals of the New York Academy of Sciences*, 575, 13-30.
- Rutter, M., & Brown, G. W. (1966). The reliability and validity of measures of family life and relationships in families containing a psychiatric patient. *Social Psychiatry*, 1, 38-53.
- Sandler, J. & Dare, C. (1970). The psychoanalytic concept of orality. *Journal of Psychosomatic Research*, 14, 211-222.
- Sarason, I. G., Ganzer, V. J., & Singer, M. (1972). "Effects of Modeled Self-Disclosure on the Verbal Behaviour of Persons Differing in Defensiveness." *Journal of Consulting and Clinical Psychology*, 39, 483-490.
- Schauble, P. G., & Pierce, R. M. (1974). Client In-Therapy Behavior: A Therapist Guide to Progress. *Psychotherapy: Theory, Research and Practice*, 11, 229-234.
- Schmidt, N. B., & Telch, M. J. (1990). Prevalence of personality disorders among bulimics, nonbulimic binge eaters, and normal controls. *Journal of Psychopathology and Behavioral Assessment*, 12, 169-185.
- Schwartz, R. C., Barrett, M. J., & Saba, G. (1985). Family therapy for bulimia, in Garner, D. M., Garner & P.E. Garfinkel (Eds.), *Handbook of Psychotherapy for Anorexia Nervosa and Bulimia*. New York: Guilford Press, pp. 513-72.
- Seeman J. (1949) A Study of the Process of Nondirective Therapy. *Journal of Consulting Psychology*, 13, 157-168.
- Selvini-Palazzoli, M. S. (1974). *Self-Starvation: From the Intrapsychic to the Transpersonal Approach to Anorexia Nervosa*. Human Context Books: Haywards Heath.
- Selvini-Palazzoli, M. S. (1978). *Self-Starvation: From Individual to Family Therapy in the Treatment of Anorexia Nervosa*. New York: Jason Aronson.
- Selvini-Palazzoli, M. S., Boscolo, L., Cecchin, G., & Prata, G. (1978). *Paradox and*

*Counterparadox*. New York: Jason Aronson.

- Shapiro, J. G., Krauss, H. H., & Truax, C. B. (1969). "Therapeutic Conditions and Disclosure Beyond the Therapeutic Encounter." *Journal of Counseling Psychology*, 16, 290-294.
- Shaver, K. G., & Drown, D. (1986). On causality, responsibility, and self-blame: A theoretical note. *Journal of Personality and Social Psychology*, 50, 697-702.
- Shore, R. A., & Porter, J. E. (1990). Normative and reliability data for 11 to 18 year olds on the Eating Disorders Inventory. *International Journal of Eating Disorders*, 9, 201-207.
- Shultz, T. R., Schleifer, M., & Altman, I. (1981). Judgements of causation, responsibility, and punishment in cases of harm-doing. *Canadian Journal of Behavioural Sciences*, 12, 238-253.
- Silverman, J. A. (1989). Louis-Victor Marce, 1824-1864: anorexia nervosa's forgotten man. *Psychological Medicine*, 19, 833-835.
- Skodol, A. E., Oldham, J. M., Hyler, S. E., Kellman, H. D., Doidge, N., & Davis, M. (1993). Comorbidity of DSM-III-R eating disorders and personality disorders. *International Journal of Eating Disorders*, 14, 402-16.
- Sloane, R. B. Staple, F. R. Cristol, A.H. Yorkston, N.J., &Whipple, K. (1975). Short-term analytically orientated psychotherapy versus behaviour therapy. *Am J Psychiatry* 132:4:373-377.
- Smith, M. L., Glass, G. V., & Miller, T. I. (1980). *The benefits of psychotherapy*. Baltimore: Johns Hopkins University Press.
- Sours, J. A. (1969). Anorexia nervosa: Nosology, diagnosis, developmental patterns and power-control dynamics. In: G. Caplan & S. Lebovici (Eds), *Adolescence: Psychological Perspectives*, (pp. 185-212). New York: Basic Books.
- Sours, J. A. (1974). The anorexia nervosa syndrome. *International Journal of Psychoanalysis*,

55, 567-76.

- Sours, J. A. (1980). *Starving to Death in a Sea of Objects*. New York: Jason Aronson.
- Staples, F. R., & others (1976) Process and Outcome in Psychotherapy and Behavior Therapy. *Journal of Consulting and Clinical Psychology, 44*, 340-350.
- Steele, B. L. (1948). The Amount of Exploration into Causes, Means, Goals and Agent: A Comparison of Successful and Unsuccessful Cases in Client-Centered Therapy. *Unpublished master's thesis*, Department of Psychology, University of Chicago.
- Steiger, H., Liguornik, K., Chapman, J., & Hussain, N. (1991). Personality and family disturbances in eating-disorder patients: Comparison of "restricters" and "bingers" to normal controls. *International Journal of Eating Disorders, 10*, 501-512.
- Steiger, H., Thibaudeau, J. Ghadirian, A. M., & Houle, L. (1992). Psychopathological features in bulimics as a function of Axis-II comorbidity: Isolation of mood-independent differences. *International Journal of Eating Disorders, 12*, 383-395.
- Steiger, H., Van der Feen, J., Goldstein, C., & Leichner, P. (1989). Defense styles and parental bonding in eating-disordered women. *International Journal of Eating Disorders, 8*, 131-140.
- Stierlin, H. (1981). *Separating Parents and Adolescents*. New York: Jason Aronson.
- Stierlin, H. (1983). Family dynamics in psychotic and severe psychosomatic disorders: A comparison. *Family System Medicine, 1*, 41-50.
- Stierlin, H., & Weber, G. (1989). *Unlocking the Family Door: A Systemic Approach to the Understanding and Treatment of Anorexia Nervosa*. New York: Brunner/Mazel.
- Stokes, J., Childs, L., & Fuehrer, A. (1981). Gender and sex roles as predictors of self-disclosure. *Journal of Counseling Psychology, 28*, 510-514.
- Stoler, N. (1963). Client Likeability: A Variable in the Study of Psychotherapy. *Journal of*

*Consulting Psychology*, 27, 175-178.

Strassberg, D. S., & others (1977). "Self-Disclosure: A Critical and Selective Review of Clinical Literature." *Comprehensive Psychiatry*, 18, 31-39.

Strenberger, L. G. & Bruns, G. L. (1990). Compulsive activity checklist and the Maudsley Obsessional-Compulsive Inventory: psychometric properties of the two measures of obsessive-compulsive disorder. *Behaviour Therapy*, 21, 117-27.

Strober, M. (1983). An empirically derived typology of anorexia nervosa. In P.L. Darby, P.E. Garfinkel, D.M. Garner & D.V. Coscina (Eds.), *Anorexia Nervosa: Recent Developments* (pp. 185-96). New York: Alan R. Liss.

Strober, M., Salkin, B., Burroughs, J., & Morrell, W. (1982). Validity of the bulimia-restrictor distinction in anorexia nervosa. *Journal of Nervous and Mental Disease*, 170, 345-51.

Strupp, H. H., & Hadley, S.W. (1979). Specific vs non-specific factors in psychotherapy. A controlled study of outcome. *Arch Gen Psychiatry* 36:1125-1136.

Stuart, R. B. (1980). *Helping Couples Change*. Guilford Press, New York.

Sturgeon, D., Turppin, G., Kuipers, L., Berkwitz, R., & Leff, J. (1984). Psychophysiological responses of schizophrenic patients to high and low expressed emotion relatives: a follow-up study. *British Journal of Psychiatry*, 145, 62-69.

Stunkard, A. J. (1959). Eating patterns and obesity. *Psychiatry Quarterly*, 33, 284-295.

Sullivan, S. H. (1954). *The Interpersonal Theory of Psychiatry*. New York: Norton.

Szmukler, G. I., Berkowitz, R., Eisler, I., Leff, J., & Dare, C. (1987). Expressed emotion in individual and family settings: A comparative study. *British Journal of Psychiatry*, 151, 174-8.

Szmukler, G., & Dare, C. (1991). The Maudsley study of family therapy in anorexia nervosa. In: D. B. Woodside and L. Shekter-Wolfson (eds), *Family Approaches to Eating Disorders*.

Washington, DC: American Psychiatric Press, Inc.

Szmukler, G. L., Eisler, I., Russell, G. F. M., & Dare, C. (1985). Anorexia nervosa, parental “expressed emotion” and dropping out of treatment. *British Journal of Psychiatry*, 147, 265-71.

Szmukler, G. I., & Russell, G. F. M. (1986). Outcome and prognosis of anorexia nervosa. In Brownell, K.D. & Forety J.P. (Eds.) *Handbook of Eating Disorders*, New York: Basic Books.

Szyrinski, V. (1973). Anorexia nervosa and psychotherapy. *American Journal of Psychotherapy*, 27, 492-505.

Swift, W. J., & Wonderlich, S. A. (1988). Personality factors and diagnosis in eating disorders: traits, disorders, and structures. In: *Diagnostic Issues in Anorexia Nervosa and Bulimia Nervosa*, eds. D. M. Garner & P. E. Garfinkel. New York: Brunner/Mazel.

Tarrier, N., & Barrowclough, C. (1987). A longitudinal psychophysiological assessment of a schizophrenic patient in relation to the Expressed Emotion of his relatives. *Behavioural Psychotherapy*, 15, 45-57.

Tarrier, N., Barrowclough, C., Porceddu, K., & Watts, S. (1988). The assessment of psychophysiological reactivity to the expressed emotion of the relatives of schizophrenic families. *British Journal of Psychiatry*, 152, 618-24.

Tarrier, N., Vaughn, C., Lader, M., & Leff, J.P. (1979). Bodily reactions to people and events in schizophrenia. *Archives of General Psychiatry*, 36, 311-315.

Taylor, D. A. (1968). The development of interpersonal relationships: Social penetration processes. *Journal of Social Psychology*, 75, 79-90.

Taylor, D. A., Altman, I., & Sorrentino, R. (1969). Interpersonal exchange as a function of rewards and costs and situational factors: Expectancy confirmation-disconfirmation.

*Journal of Experimental Social Psychology*, 5, 324-339.

Taylor, S. E., Lichtman, R. R., & Wood, J. V. (1984). Attributions, beliefs about control, and adjustment to breast cancer. *Journal of Personality and Social Psychology*, 46, 489-502.

Taylor, D. A., & Oberlander, L. (1969). Person-perception and self-disclosure: Motivational mechanisms in interpersonal processes. *Journal of Experimental Research in Personality*, 4, 14-28.

Tennen, H., & Affleck, G. (1990). Blaming others for treating events. *Psychological Bulletin*, 108, 209-232.

Tennen, H., Affleck, G., Allen, D. A., McGrade, B. J., & Ratzan, S. (1984). Causal attributions and coping with insulin-dependent diabetes. *Basic and Applied Social Psychology*, 5, 131-142.

Tennen, H., Affleck, G., & Gershman, K. (1986). Self-blame among parents of infants with perinatal complications: The role of self-protective motives. *Journal of Personality and Social Psychology*, 50, 690-696.

Theander, S. (1970). Anorexia nervosa: a psychiatric investigation of 94 female patients. *Acta Psychiatrica Scandinavica (Suppl.)*, 214, 1-94.

Theander, S. (1995). The essence of anorexia nervosa: comments on Gerald Russell's "anorexia nervosa through time". In: G. Szumukler, C. Dare, J. Treasure, *Handbook of Eating Disorders: Theory, Treatment and Research*. Chichester: John Wiley & Sons.

Thoma, H. (1967). *Anorexia Nervosa*. New York: International Universities Press.

Timko, C., & Janoff-Bulman, R. (1985). Attributions, vulnerability and psychological adjustment: The case of breast cancer. *Health Psychology*, 4, 521-546.

Tomlinson, T. M. (1967). The Therapeutic Process as Related to Outcome. In C. R. Rogers and others (Eds.), *The Therapeutic Relationship and Its Impact: A Study of Psychotherapy*



*with Schizophrenics*. Madison: University of Wisconsin Press.

- Tomlinson, T. M., & Hart, J. T. (1962). A Validation Study of the Process Scale. *Journal of Consulting Psychology, 26*, 74-78.
- Tomlinson, T. M., & Stoler, N. (1967). The Relationship Between Affective Evaluation and Rating of Therapy Process and Outcome with Schizophrenics. *Psychotherapy: Theory, Research and Practice, 4*, 14-18.
- Touyz, S. W., & Beumont, P. J. V. (1989). Anorexia and bulimia nervosa: In: P. J. V. Beumont & R. Hampshire (Eds), *Textbook of Psychiatry*. Melbourne: Blackwells Australia.
- Truax, C. B. (1961). A scale for the measurement of accurate empathy. *Psychiatric Institute Bulletin*, Wisconsin Psychiatric Institute, University of Wisconsin, 1, 12.
- Truax, C.B. & Carkhuff, R.R.(1964). "Concreteness: A Neglected Variable in Research in Psychotherapy." *Journal of Clinical Psychology, 20*, 264-267.
- Truax, C. B., & Carkhuff, R. R.(1965). Client and Therapist Transparency in the Psychotherapeutic Encounter. *Journal of Counseling Psychology, 12*, 3-9.
- Truax, C. B., & Wargo, D. G. (1969). Effects of Vicarious Therapy Pretraining and Alternate Sessions on Outcome in Group Psychotherapy with Outpatients. *Journal of Consulting and Clinical Psychology, 33*, 440-447.
- Truax, C. B., Wargo, D. G., & Volksdorf, N. R. (1970). Antecedents to Outcome in Group Counseling with Institutionalized Juvenile Delinquents: Effects of Therapeutic Conditions, Patient Self-Exploration, Alternate Sessions, and Vicarious Therapy Pretraining. *Journal of Abnormal Psychology, 76*, 235-242.
- Truax, C. B., & Wittmer, J. (1971). Patient Non-Personal References During Psychotherapy and Therapeutic Outcome. *Journal of Clinical Psychology, 27*, 300-302.
- Turnquist, D. C., Harvey, J. H., & Andersen, B. L. (1988). Attributions and adjustment to life-

threatening illness. *British Journal of Clinical Psychology*, 27, 55-65.

Tyrer, P. (1988). *Personality disorders: diagnosis, management and course*. London: Butterworth & Co.

Tyrer, P, and Alexander, J. (1979). Classification of personality disorder. *British Journal of Psychiatry*, 135, 163-167.

Tyrer, P, and Alexander M. S., Cichetti D., Cohen M. S., & Remington, M. (1979). Reliability of schedule for rating personality disorders. *British Journal of Psychiatry*, 135, 168-74.

Tyrer, P., Casey, P., & Gall, J. (1983). Relationship between neurosis and personality disorder. *British Journal of Psychiatry*, 142, 404-8.

Tyrer, P., & Seivewright, H. (1988). Studies of outcome. In P. Tyrer (Ed), *Personality Disorders* (pp. 119-137). London: Wright.

Valone, K., Norton, J. P., Goldstein, M. J., & Doane J. A. (1983). Parental expressed emotion and affective style in an adolescent sample at risk for schizophrenia spectrum disorders. *Journal of Abnormal Psychology*, 92, 399-407.

Vandereyken, W., & Pierloot, R. (1983). Drop-out during in-patient treatment of anorexia nervosa: a clinical study of 133 patients. *British Journal of Medical Psychology*, 56, 145-56.

Vandereyken, W. & Van Deth, R. (1990). A tribute to Lasegue's description of anorexia nervosa (1873), with a completion of its English translation. *British Journal of Psychiatry*, 157, 902-908.

Van Der Veen, F. (1967). Basic Elements in the Process of Psychotherapy. *Journal of Consulting Psychology*, 31, 295-301.

Van Furth, E. F. (1991). *Parental Expressed Emotion and Eating Disorders*. Unpublished Ph.D. thesis. Utrecht University, The Netherlands.

- Van Furth, E. F., Van Strien, D. C., Martina, L. M. L., Van Son, M. J. M., Hendrickx, J. J. P., & Van Engeland, H. (1996). Expressed emotion and the prediction of outcome in adolescent eating disorders. *International Journal of Eating Disorders*, 20, 19-31.
- Vaughn, C. E. (1989). Annotation: Expressed emotion in family relationship. *Journal of Child Psychology and Psychiatry*, 30, 13-22.
- Vaughn, C. E., & Leff, J. (1976a). The influence of family and social factors on the course of psychiatric illness: A comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry*, 129, 125-37.
- Vaughn, C. E., & Leff, J. (1976b). The measurement of expressed emotion in the families of psychiatric patients. *British Journal of Social and Clinical Psychology*, 15, 157-65.
- Vaughn, C. E., Snyder, K. S., Jones, S., Freeman, W. B., & Falloon, I. R. H. (1984). Family factors in schizophrenic relapse: A California replication of the British research on expressed emotion. *Archives of General Psychiatry*, 41, 1169-1177.
- Vitaliano, P. P., Becker, F., Russo, R., & Magana-Amato, A. (1989). Expressed emotion in spouse caregiver of patients with Alzheimer's disease. *Journal of Applied Social Sciences*, 13, 215-50.
- Vondracek, F. W. (1969). Behavioural measurement of self-disclosure. *Psychological Reports*, 25, 914.
- Vondracek, F. W. (1969). The study of self-disclosure in experimental interviews. *Journal of Psychology*, 72, 55-59.
- Walker, A., Rablen, R. A., & Rogers C. R. (1960). Development of a Scale to Measure Process Change in Psychotherapy. *Journal of Clinical Psychology*, 16, 79-85.
- Waller, J. V., Kaufman, M. R., & Deutsch, F. (1940). Anorexia nervosa: A psychosomatic entity. *Psychosomatic Medicine*, 2, 3.

- Wear, R. W., & Pratz, O. (1987). Test-retest reliability for the Eating Disorder Inventory. *International Journal of Eating Disorders*, 6, 767-769.
- Weddell, R. A. (1987). Social, functional, and neuropsychological determinants of the psychiatric symptoms of stroke patients receiving rehabilitation and living at home. *Scandinavian Journal of Rehabilitation in Medicine*, 19, 93-8.
- Weiss, R. S. (1975). *Marital separation*. New York: Basic Books.
- Weissman, M., & Rothwell, S. (1976). Assessment of social adjustment by self-report. *Archives of General Psychiatry*, 33, 1111-1115.
- Wermuth, B. M., Davis, K. L., Hollister, L. E., & Stunkard, A. J. (1977). Phenytoin treatment of the binge eating syndrome. *American Journal of Psychiatry*, 11, 1249-1253.
- Willi, J. & Grossmann, S. (1983). Epidemiology of anorexia nervosa in a defined region of Switzerland. *American Journal of Psychiatry*, 140, 564-567.
- Winawer, H. (1983). The Heidelberg concept: An introduction to the work of Helm Stierlin and his associates. *Family Systems Medicine*, 1, 36-40.
- Wirsching, M., & Stierlin, H. (1985). Psychosomatics. I. Psychosocial characteristics of psychosomatic patients and their families. *Family Systems Medicine*, 3, 6-16.
- Witenberg, S. H., Blanchard, E. B., Suls, J., Tennen, H., McCoy, G., & McGoldrick, M. D. (1983). Perceptions of control and causality as predictors of compliance and coping in hemodialysis. *Basic and Applied Social Psychology*, 4, 319-336.
- Wolfson, K. S. (1949). Clients' Exploration of Their Problems During Client-Centered Therapy. *Unpublished master's thesis*, Department of Psychology, University of Chicago, 1949.
- Wonderlich, S. A. & Swift, W. J. (1990). Borderline versus other personality disorders in the eating disorders. *International Journal of Eating Disorders*, 9, 617-28.
- Wonderlich, S. A., Swift, W. J., Slotnick, H. B., & Goodman, S. (1990). DSM-III-R personality

disorders in eating disorder subtypes. *International Journal of Eating Disorders*, 9, 607-616.

World Health Organization (1965). *International Classification of Disease*, eighth revision. Geneva.

Worthy, M., Gary, A. L., & Kahn, G. M. (1969). Self-disclosure as an exchange process. *Journal of Personality and Social Psychology*, 13, 59-63.

Whytt, R. (1764). Observations on the Nature, Causes, and Cure of those Disorders which have been commonly called Nervous, Hypochondriac or Hysterical to which are prefixed some remarks on the sympathy of the Nerves. Edinburgh: Becket, DeHondt and Balfour.

Yalom, I. D. (1970). *The Theory and Practice of Group Psychotherapy*. New York: Basic Books.

Yager, J. (1981). Anorexia nervosa and the family. In M. R. Lansky (Ed.), *Family therapy and major psychopathology* (pp. 249-280). New York: Grune and Stratton.

Yager, J. (1982). Family in the pathogenesis of anorexia nervosa. *Psychosomatic Medicine*, 44, 43-60.

Yager J. Landsverk J. Edlestein C.K. et al. (1989). Screening for axis II personality disorders in women with bulimic eating disorders. *Psychosomatics*, 30, 255-62.

Yates, W. R., Sielieni, B., Reich, J., & Brass, C. (1989). Comorbidity of bulimia nervosa and personality disorder. *Journal of Clinical Psychiatry*, 50, 57-59.

Yellowless, A. J. (1985). Anorexia and bulimia in anorexia nervosa. A study of psychosocial functioning and associated psychiatric symptomatology. *British Journal of Psychiatry*, 146, 648-652.

Zanarini, M. M., Frankenburg, F. R., Pope, H. G., Hudson, J. I., Yurgelun-Todd, D., & Cicchetti, C. J. (1990). Axis II comorbidity of normal-weight bulimia. *Comprehensive Psychiatry*, 30, 20-24.

## **APPENDICES**

### **APPENDIX A**

#### **EATING DISORDER CLINIC CONSENT FORM OUT-PATIENT PSYCHOLOGICAL TREATMENTS**

We would like to offer you a place in our out-patient treatment programme for anorexia nervosa. You are probably aware that there is more than one way of treating anorexia nervosa and that different treatments suit different patients. It is important that we continue to improve our knowledge of how best to help people suffering from anorexia nervosa and we cannot do this without active help from our patients. In order to learn whether one treatment is better than another we need your help to compare the available treatments. The treatment you receive as part of this study will depend on your particular place in the study.

During the course of your treatment you will be asked to see the investigator who will make an independent assessment of your progress. Similarly, after the treatment has ended, you will be asked to attend for follow-up interviews in order to see how you are getting on. A crucial aspect of these follow-up interviews will be to get some feedback from you about your personal experience of the treatment, of what you had found helpful and what unhelpful.

The assessment interviews are videotaped to make sure that we make full use of the information that you give us. During the treatment the therapist may from time to time also want to video tape some of the sessions. Strict confidentiality of all video tapes will always be observed and they will only be seen by professional staff within the Institute of Psychiatry and The Bethlem Royal and Maudsley Hospitals.

I accept the treatment that I am being offered, the nature of which has been explained to me. I also accept to take part in the research interviews.

.....  
(Name)

.....  
(Signature)

.....  
(Date)

**ACCEPTING THE TREATMENT TO WHICH YOU HAVE BEEN ASSIGNED IS  
NOT  
A CONDITION OF CONTINUED TREATMENT AT THE MAUDSLEY HOSPITAL.**

## APPENDIX B

### THE MORGAN-RUSSELL ASSESSMENT SCHEDULE

Scale	Subscales		
Food intake	A	A1	Restriction of food intake
		A2	Concern at body image
		A3	Body weight
Menstrual state	B	B	Menstrual pattern
Mental state	C	C	Disturbance of mental state
Psychosexual state	D	D1	Attitude towards sexual matters
		D2	Aims in sexual matters
		D3	Overt sexual behaviour
		D4	Attitude to menstruation if returned or if not returned
Socioeconomic state	E	E1	Relationship with family
		E2	Emancipation from family
		E3	Social contacts outside family
		E4	Social activities outside family
		E5	Employment record

For the purpose of correlating outcome with other clinical variables obtained retrospectively from analysis of the case notes, an average score S of all compound scales in each patient was calculated.

$$\text{Final average score } S = \frac{\text{scores } (A+B+C+D+E)}{5}$$

To express outcome crudely in this way might seem inappropriate, but ranking cases on the basis of these scores does appear to have some approximation to general impression of clinical outcome in the series as a whole.

#### Layout of Scales

*Scale G. Self-progress rating* (rated by patient and other informant)

Category	Worse	Static	Improved	Recovered
Score	0	1	2	3

This scale is not used in calculating average outcome score.

*Scale A. Food intake*

*Subscale A1. Dietary restriction*

Question: "Are you restricting your diet, or have you done so at any time in the last six months?"

Category	At all times	More than half the time	About half the time	Less than half the time	Nil
Score	0	3	6	9	12

Coding instructions: ignore minor carbohydrate restriction to the extent of being careful about the amount of sugar or bread, because such attitude is common even in normal individuals. Only true reduction of food intake below average levels is taken as significant for the purpose of rating on this scale.

*Sub scale A2. Worry about body weight or appearance*

Question: "Have you been worried about your weight or your appearance in any other way, at any time in the last six months?"

Category	At all times	More than half the time	About half the time	Less than half the time	Nil
Score	0	3	6	9	12

*Sub scale A3. Body weight*

Category	Always much deviation Sufficient to cause concern	Always deviated but only at times sufficient to cause concern	Usually near average (with- in $\pm 15\%$ average body weight but Occasionally deviant Sufficient to cause Concern	Near average at all times
Score	0	4	8	12

Coding instruction: difficulties arise here in interpretation of the degree of weight deviation which is regarded as sufficient to cause concern. The patient's opinion on this issue may of course be highly unreliable; occasionally over-anxious relatives also seemed to be highly biased on this issue. The rating was therefore made after reference to the reported weight fluctuations over the previous 6 months and the weight measured at interview. Near average weight is taken as within  $\pm 15\%$  of average body weight.

*Scale B. Menstrual pattern (in previous 6 months)*

Category	No menstrual loss at any time	Transient occasional menstrual loss, which is never cyclical	Irregular menstrual loss with Some cyclical Pattern	Regular and cyclical throughout
Score	0	4	8	12

*Scale C. Mental state (as observed at interview and reported abnormalities at any time in previous 6 months)*

Category	Grossly abnormal and psychotic with delusions+hallucinations	Marked disturbance but not psychotic	Mild disturbance	Normal
Score	0	4	8	12



Coding instruction: This scale is based on a mental-state assessment during interview, and information about the psychiatric status during the preceding 6 months. The distinction between 'marked' and 'mild' disturbance of one type was made on the basis of interference with general activities: thus, symptoms which prevented the patient working at any time in the 6-months period would be rated as 'marked'. If symptoms are judged present and significant (excluding marked ideas about food), yet they have not interfered with normal activities, these are rated as 'mild'.

*Scale D. Psychosexual state*

*Subscale D1. Attitude towards sexual matters*

Question: "What is your attitude towards sexual matters?"

Category	Active dislike	Variable: dislike or disinterest	Disinterest	Pleasurable
Score	0	4	8	12

*Subscale D2. Professed aims in sexual relationships*

Category	Wants to remain single	Would marry but fears to do so	Would marry appropriate person but would not want To have children	Definitely wants to marry and have children
Score	0	4	8	12

*Subscale D3. Overt sexual behaviour*

Category	Avoids heterosexual contacts	Occasional superficial affairs without pleasurable sexual relationship	Love affairs with pleasurable sexual relationship (may include married with children)
Score	0	6	12

*Subscale D4. Attitude to menstruation (if it has returned)*

Category	Active dislike	Variable: dislike or disinterest	Disinterest	Pleased that it has returned
Score	0	4	8	12

*Subscale D5. Attitude to menstruation (if it has not returned)*

Category	Pleased not returned	Variable: pleased or disinterested	Disinterest	Regrets not returned
Score	0	4	8	12

*Scale E. Socioeconomic state*

*Subscale E1. Relationship with nuclear family*

Question: "How would you assess your relationship with your parents (and siblings)?"

Category	Very unsatisfactory	Unsatisfactory	Indifferent	Satisfactory
Score	0	4	8	12

Coding instructions: In view of the fact that relationship may vary with different members of the family, the lowest individual rating is taken, whether it is with parent or sibling. When another informant is seen beside the patient, the final rating is taken as the average of these two scores.

*Subscale E2. Emancipation from family* (degree of adult autonomy without transferred dependency)

Category	Many difficulties sees no prospect of becoming independent to a satisfactory degree	As for 0 but at times feels difficult can be surmounted	Some difficulties but they are surmountable	No difficulties
Score	0	4	8	12

*Subscale E3. Personal contacts* (apart from family or partner)

Category	None	Few and Superficial	Many but superficial	Many close and superficial friends
Score	0	4	8	12

*Subscale E4. Social activities* (appropriate to status)

Category	Nil outside family	Solitary outside family	Variable: mainly solitary but some Group activities Outside family	Adequate group activities: mixed well outside family
Score	0	4	8	12

*Subscale E5. Employment record* (in previous 6 months)

Category	No paid employment	Up to 50% of the period In paid employment Or occasional Unpaid employment	More than 50% of the period in paid employment but less than 100%	Regular full time paid employment without absences
Score	0	4	8	12

## **APPENDIX C**

### **PROTOCOL OF THE STANDARDIZED CLINICAL FAMILY INTERVIEW (SCFI)**

#### **Introduction**

Introduce self.

Personally meet each family member. Check names and ages.

Thank family for attending.

Enquire about Missing school, Missing work, Any difficulty in coming. Apologise for any delays.

S: We are interested in the way families cope with illness, and about family life in general. Today we shall not be asking you any questions about coeliac disease, but would like to get to know how you are as a family.

S: In a family everybody is important and so we asked everyone to come up. We don't want just to hear Mummy and Daddy but what . . . . thinks, and . . . . and . . . . , etc.

Q: (To parents)  
Have you discussed this meeting with the children?  
(Whatever the reply, ask the children:)  
How did you feel about coming up?

S: The paper, crayons and box of toys is for (children's names) and you can play with them if you want, because that's a way you can show us what you are thinking. And of course you can say what you are thinking.

S: Everything today is shared experience. So anything anyone says is for everyone to hear.

S: You know that this interview is being watched and recorded on video-tape, and I understand that you are agreeable to this?  
It will only be seen by a few people directly involved in the research.  
For the same reason we would like to keep any drawings that are made.

#### **What sort of family**

Q: How would you describe yourself as a family?

Q: What words would you use to describe yourselves as a family?  
(Allow a silence.  
If the family replies, then follow this up.  
If not, then:)

#### **Cohesion**

S: Families vary a great deal in the way their members get together. Some are together almost all the time, e. g. the husband and wife work at home and the children are there too. Others have things organized differently, e. g. when the husband's job means he is away a lot and perhaps the wife has to work too, then this family is not together much of the time.

Q: How does your family fit on this wide range?  
(After this is clarified:)

- Q: Is this O. K. by everyone?  
(Attempt to get an idea of the home atmosphere)
- Q: Do you do anything together as a whole family?  
(After the spontaneous response:)  
Probe: Eating together, Watching TV together, Sleeping patterns and arrangements.

### **Who does what with whom**

- Q: Who does what with whom in your family?  
(Explore the spontaneous response.)  
(If an individual has been left out, then turn to him:)
- Q: Do you do things with anyone in particular, or do you prefer being on your own?  
(If any particular dyads have been left out:)
- Q: Do . . . and . . . do anything together?  
What about . . . ? etc. (Names for children; Mum/Dad)

### **Who is like whom**

- Q: Families are made up of separate people, but sometimes they are alike in many ways\_  
Who is like whom in your family?  
(Allow a silence)
- or (Attributions should be checked out: 'Johnny, Dad says that you have a bit of a temper like him. What do you think?')  
(If the family is one where everybody is the same, then look for differences:)
- Q: Although people in a family are often very much like each other, each person is different in some way  
How are people different in your family?

### **Life cycle**

- Q: A family moves through various phases: first there is the time before there are any children, then young babies come, then a stage of toddlers pre-school, then school children, then teenager life, and so on, till the children have all left\_  
How do you find it (in this phase)?
- Q: How does it compare to the earlier phase?  
Q: What are your thoughts about the coming phase?

### **Roles and responsibilities**

- Q: There are lots of responsibilities and jobs in running a home\_  
Who does what?  
(To the children if left out:)  
Are there any jobs for you?
- Q: Is this a fixed pattern?
- Q: Is it fair?

## Conflicts

- Q: Families often have conflicts and disagreements\_does yours?  
What are yours about?
- A. (If no response:)
- Q: There can be disagreements between you as husband and wife, between you both as parents and the children, or between X and Y (children's names)?  
(If no response, or denial of disagreement absolutely, then MOVE to Decisions below).
- B. (If spontaneous response is made:)
- Q: How do the disagreements happen?
- Q: Can you tell me what it's like?
- C. (If examples from earlier in the session have occurred:)  
Earlier it seemed that. . .
- D. Offer probes once the family have begun, following a hierarchical order:  
1. Inter-sibling. 2. Parent\_Child. 3. Marital.
- Q: (Between children:) Children often fight amongst themselves. Does X do anything that Y doesn't like? And the other way round?
- Q: Is there anything else the children fight about?
- Q: (Parent\_Child) Parents and children often argue about things\_  
What about with you? Time to do things?  
Going out?  
Bed-time?  
TV time?
- Q: (Husband-wife) Husbands and wives also have their differences\_
- Q: Do these occur in front of the children? .  
Do the children know about it when they're on?  
Probe: Managing the children  
Money

## Decisions

- Q: How do you go about making decisions that affect everybody?  
(If the example used is clearly role-oriented, e.g. car purchase, what to cook, then offer TV shows).
- Q: When there are conflicts or problems about decisions, how do things get worked out?  
(Follow up to be clear that resolution/compromise does occur and how)

## Discipline

- Q: Can you tell me about discipline in your family?
- Q: Who applies it and how? Who needs it? And when?
- Q: Does everyone think it's fair?

## **Relation to the environment**

Q: Families vary in the ways they get on with their relations\_  
What's it like with you?

Q: We've heard a lot about what you do in the home\_do you things in the neighbourhood?  
e.g. neighbours, clubs, local groups

## APPENDIX D

### THE BULIMIC INVESTIGATORY TEST, EDINBURGH (BITE)

1. Do you have a regular daily eating pattern?	YES	NO					
2. Are you a strict dieter?	YES	NO					
3. Do you feel a failure if you break your diet once?	YES	NO					
4. Do you count the calories of everything you eat, even when not on a diet?	YES	NO					
5. Do you ever fast for a whole day?	YES	NO					
6. . . . If yes, how often is this?	EVERY SECOND DAY	5					
	2-3 TIMES A WEEK	4					
	ONCE A WEEK	3					
	NOW AND THEN	2					
	HAVE ONCE	1					
7. Do you do any of the following to help you lose weight?							
	Never	Occasionally	Once a week	2-3 times Week	Daily	2-3 times a day	5+times a day
TAKE DIET PILLS	0	2	3	4	5	6	7
TAKE DIURETICS	0	2	3	4	5	6	7
TAKE LAXATIVES	0	2	3	4	5	6	7
MAKE YOURSELF VOMIT	0	2	3	4	5	6	7
8. Does your pattern of eating severely disrupt your life?	YES	NO					
9. Would you say that food dominated your life?	YES	NO					
10. Do you ever eat and eat until you are stopped by physical discomfort?	YES	NO					
11. Are there times when all you can think about is food?	YES	NO					
12. Do you eat sensibly in front of others and make up in private?	YES	NO					
13. Can you always stop eating when you want to?	YES	NO					
14. Do you ever experience overpowering urges to eat and eat and eat?	YES	NO					
15. When you are feeling anxious do you tend to eat a lot?	YES	NO					
16. Does the thought of becoming fat terrify you?	YES	NO					
17. Do you ever eat large amounts of food rapidly (not a meal)?	YES	NO					
18. Are you ashamed of your eating habits?	YES	NO					
19. Do you worry that you have no control over how much you eat?	YES	NO					
20. Do you turn to food for comfort?	YES	NO					
21. Are you able to leave food on the plate at the end of a meal?	YES	NO					
22. Do you deceive other people about how much you eat?	YES	NO					
23. Does how hungry you feel determine how much you eat?	YES	NO					
24. Do you ever binge on large amounts of food?	YES	NO					
25. . . . If yes, do such binges leave you feeling miserable?	YES	NO					
26. If you do binge, is this only when you are alone?	YES	NO					

- |   |                  |   |  |  |
|---|------------------|---|--|--|
| 27. If you do binge, how often is this? | HARDLY NEVER     | 1 |  |  |
|   | ONCE A MONTH     | 2 |  |  |
|   | ONCE A WEEK      | 3 |  |  |
|   | 2-3 TIMES A WEEK | 4 |  |  |
|   | DAILY            | 5 |  |  |
|   | 2-3 TIMES A DAY  | 6 |  |  |
- 
- |   |     |    |
|---|-----|----|
| 28. Would you go to great lengths to satisfy an urge to binge?  | YES | NO |
| 29. If you overeat do you feel very guilty?                     | YES | NO |
| 30. Do you ever eat in secret?                                  | YES | NO |
| 31. Are you eating habits what you would consider to be normal? | YES | NO |
| 32. Would you consider yourself to be a compulsive eater?       | YES | NO |
| 33. Does your weight fluctuate by more than 5 pounds in a week? | YES | NO |



## APPENDIX E

### THE INVENTORY OF INTERPERSONAL PROBLEMS (IIP)

Here is a list of problems that people report in relating to other people. Please read the list below, and for each item, consider whether that problem has been a problem for you with respect to any significant person in your life. Then select the number that describes how distressing that problem has been, and circle that number.

#### EXAMPLE

How much have you been distressed by this problem?

It is hard for me to:

	Not at all	A little bit	Moderately	Quite a bit	Extremely
00. Get along with my relatives.	0	1	2	(3)	4

#### Part I. The following are things you find hard to do with other people.

It is hard for me to:	Not at all	A little bit	Moderately	Quite a bit	Extremely
1. trust other people.	0	1	2	3	4
2. say "no" to other people.	0	1	2	3	4
3. join in on groups.	0	1	2	3	4
4. keep things private from other people.	0	1	2	3	4
5. let other people know what I want.	0	1	2	3	4
6. tell a person to stop bothering me.	0	1	2	3	4
7. introduce myself to new people.	0	1	2	3	4
8. confront people with problems that come up.	0	1	2	3	4
9. be assertive with another person.	0	1	2	3	4
10. make friends.	0	1	2	3	4
11. express my admiration for another person.	0	1	2	3	4
12. have someone dependent on me.	0	1	2	3	4
13. disagree with other people.	0	1	2	3	4
14. let other people know when I am angry.	0	1	2	3	4
15. make a long-term commitment to another person.	0	1	2	3	4
16. stick to my own point of view and not be swayed by other people.	0	1	2	3	4
17. be another person's boss.	0	1	2	3	4
18. do what another person wants me to do.	0	1	2	3	4
19. get along with people who have authority over me.	0	1	2	3	4
20. be aggressive toward other people when the situation calls for it.	0	1	2	3	4
21. compete against other people.	0	1	2	3	4

22. make reasonable demands of other people.	0	1	2	3	4
23. socialize with other people.	0	1	2	3	4
24. get out of a relationship that I don't want to be in.	0	1	2	3	4
25. take charge of my own affairs without help from other people.	0	1	2	3	4
26. show affection to people.	0	1	2	3	4
27. feel comfortable around other people.	0	1	2	3	4
28. get along with people.	0	1	2	3	4
29. understand another person's point of view.	0	1	2	3	4
30. tell personal things to other people.	0	1	2	3	4
31. believe that I am loveable to other people.	0	1	2	3	4
32. express my feeling to other people directly.	0	1	2	3	4
33. be firm when I need to be.	0	1	2	3	4
34. experience a feeling of love for another person.	0	1	2	3	4
35. be competitive when the situation calls for it.	0	1	2	3	4
36. set limits on other people.	0	1	2	3	4
37. be honest with other people.	0	1	2	3	4
38. be supportive of another person's goals in life.	0	1	2	3	4
39. feel close to other people.	0	1	2	3	4
40. really care about other people's problems.	0	1	2	3	4
41. argue with another person.	0	1	2	3	4
42. relax and enjoy myself when I go out with other people.	0	1	2	3	4
43. feel superior to another person.	0	1	2	3	4
44. become sexually aroused toward the person I really care about.	0	1	2	3	4
45. feel that I deserve another person's affection.	0	1	2	3	4
46. keep up my side of a friendship.	0	1	2	3	4
47. spend time alone.	0	1	2	3	4
48. give a gift to another person.	0	1	2	3	4
49. have loving and angry feeling towards the same person.	0	1	2	3	4
50. maintain a working relationship with someone I don't like.	0	1	2	3	4
51. set goals for myself without other people's advice.	0	1	2	3	4
52. accept another person's authority					

over me.	0	1	2	3	4
53. feel good about winning.	0	1	2	3	4
54. ignore criticism from other people.	0	1	2	3	4
55. feel like a separate person when I am in a relationship.	0	1	2	3	4
56. allow myself to be more successful than other people.	0	1	2	3	4
57. feel or act competent in my role as parent.	0	1	2	3	4
58. let myself feel angry at somebody I like.	0	1	2	3	4
59. respond sexually to another person.	0	1	2	3	4
60. accept praise from another person.	0	1	2	3	4
61. put somebody else's needs before my own.	0	1	2	3	4
62. give credit to another person for doing something well.	0	1	2	3	4
63. stay out of other people's business.	0	1	2	3	4
64. take instructions from people who have authority over me.	0	1	2	3	4
65. feel good about another person's happiness.	0	1	2	3	4
66. get over the feeling of loss after a relationship has ended.	0	1	2	3	4
67. ask other people to get together socially with me.	0	1	2	3	4
68. feel angry at other people.	0	1	2	3	4
69. give constructive criticism to another person.	0	1	2	3	4
70. experience sexual satisfaction.	0	1	2	3	4
71. open up and tell my feeling to another person.	0	1	2	3	4
72. forgive another person after I've been angry.	0	1	2	3	4
73. attend to my own welfare when somebody else is needy.	0	1	2	3	4
74. be assertive without worrying about hurting the other person's feelings.	0	1	2	3	4
75. be involved with another person without feeling trapped.	0	1	2	3	4
76. do work for my own sake instead of for someone else's approval.	0	1	2	3	4
77. be close to somebody without feeling that I'm betraying somebody else.	0	1	2	3	4
78. be self-confident when I am with other people.	0	1	2	3	4

**Part II. The following are things that you do too much.**

79. I fight with other people too much	0	1	2	3	4
80. I am too sensitive to criticism.	0	1	2	3	4
81. I feel too responsible for solving other people's problems.	0	1	2	3	4
82. I get irritated or annoyed too easily.	0	1	2	3	4
83. I am too easily persuaded by other people.	0	1	2	3	4
84. I want people to admire me too much.	0	1	2	3	4
85. I act like a child too much.	0	1	2	3	4
86. I am too dependent on other people.	0	1	2	3	4
87. I am too sensitive to rejection.	0	1	2	3	4
88. I open up to people too much.	0	1	2	3	4
89. I am too independent.	0	1	2	3	4
90. I am too aggressive toward other people.	0	1	2	3	4
91. I try to please other people too much.	0	1	2	3	4
92. I feel attacked by other people too much.	0	1	2	3	4
93. I feel too guilty for what I have done.	0	1	2	3	4
94. I clown around too much.	0	1	2	3	4
95. I want to be noticed too much.	0	1	2	3	4
96. I criticize other people too much.	0	1	2	3	4
97. I trust other people too much.	0	1	2	3	4
98. I try to control other people too much.	0	1	2	3	4
99. I avoid other people too much.	0	1	2	3	4
100. I am affected by another person's moods too much.	0	1	2	3	4
101. I put other people's needs before my own too much.	0	1	2	3	4
102. I try to change other people too much.	0	1	2	3	4
103. I am too gullible.	0	1	2	3	4
104. I am overly generous to other people.	0	1	2	3	4
105. I am too afraid of other people.	0	1	2	3	4
106. I worry too much about other people's reactions to me.	0	1	2	3	4
107. I am too suspicious of other people.	0	1	2	3	4
108. I am influenced too much by another person's thoughts and feelings.	0	1	2	3	4
109. I compliment other people too much.	0	1	2	3	4
110. I worry too much about disappointing other people.	0	1	2	3	4
111. I manipulate other people too much to get what I want.	0	1	2	3	4
112. I lose my temper too easily.	0	1	2	3	4
113. I tell personal things to other people too much.	0	1	2	3	4
114. I blame myself too much for causing					

other people's problems.	0	1	2	3	4
115. I am too easily bothered by other people making demands of me.	0	1	2	3	4
116. I argue with other people too much.	0	1	2	3	4
117. I am too envious and jealous of other people.	0	1	2	3	4
118. I keep other people at a distance too much.	0	1	2	3	4
119. I worry too much about my family's reaction to me.	0	1	2	3	4
120. I let other people take advantage of me too much.	0	1	2	3	4
121. I too easily lose a sense of myself when I am around a strong-minded person.	0	1	2	3	4
122. I feel too guilty for what I have failed to do.	0	1	2	3	4
123. I feel competitive even when the situation does not call for it.	0	1	2	3	4
124. I feel embarrassed in front of other people too much.	0	1	2	3	4
125. I feel too anxious when I am involved with another person.	0	1	2	3	4
126. I am affected by another person's misery too much.	0	1	2	3	4
127. I want to get revenge against people too much.	0	1	2	3	4

## APPENDIX F

### THE MAUDSLEY OBSESSIONAL-COMPULSIVE INVENTORY (MOCI)

**INSTRUCTION:** Please answer each question by putting a circle around the "TRUE" or the "FALSE" following the question. There are no right or wrong questions answers, and no trick questions. Work quickly and do not think too long about the exact meaning of the question.

1. I avoid using public telephones because of possible contamination.	TRUE	FALSE
2. I frequently get nasty thoughts and have difficulty in getting rid of them.	TRUE	FALSE
3. I am more concerned than most people about honesty.	TRUE	FALSE
4. I am often late because I can't seem to get through everything on time.	TRUE	FALSE
5. I don't worry unduly about contamination if I touch an animal.	TRUE	FALSE
6. I frequently have to check things (e.g. gas or water taps, doors, etc) several times.	TRUE	FALSE
7. I have a very strict conscience.	TRUE	FALSE
8. I find that almost every day I am upset by unpleasant thoughts that come into my mind against my will.	TRUE	FALSE
9. I do not worry unduly if I accidentally bump into someone.	TRUE	FALSE
10. I usually have serious doubts about the simple everyday things I do.	TRUE	FALSE
11. Neither of my parents was very strict during my childhood.	TRUE	FALSE
12. I tend to get behind in my work because I repeat things over and over again.	TRUE	FALSE
13. I use only an average amount of soap.	TRUE	FALSE
14. Some numbers are extremely unlucky.	TRUE	FALSE
15. I do not check letters over and over again before posting them.	TRUE	FALSE
16. I do not take a long time to dress in a morning.	TRUE	FALSE
17. I am not excessively concerned about cleanliness.	TRUE	FALSE
18. One of my major problems is that I pay too much attention to detail.	TRUE	FALSE
19. I can use well-kept toilets without any hesitation.	TRUE	FALSE
20. My major problem is repeated checking.	TRUE	FALSE
21. I am not unduly concerned about germs and diseases.	TRUE	FALSE
22. I do not tend to check things more than once.	TRUE	FALSE
23. I do not stick to a very strict routine when doing ordinary things.	TRUE	FALSE
24. My hand do not feel dirty after touching money.	TRUE	FALSE
25. I do not usually count when doing a routine task.	TRUE	FALSE
26. I take rather a long time to complete my washing in the morning.	TRUE	FALSE
27. I do not use a great deal of antiseptics.	TRUE	FALSE
28. I spend a lot of time every day checking things over and over again.	TRUE	FALSE

29. Hanging and folding my clothes at night does not take up a lot of time.
30. Even when I do something very carefully I often feel that it is not quite right.

TRUE	FALSE
TRUE	FALSE

