

https://dspace.org/

A single-beam positron emission computerized tomography study of trichotillomania in terms of cognitive behaviour therapy

Gordon, Charmaine

2008-07-07T12:57:39Z
http://hdl.handle.net/10539/5006

Downloaded from a DSpace repository. https://dspace.org/

A SINGLE-BEAM POSITRON EMISSION COMPUTERIZED TOMOGRAPHY STUDY OF TRICHOTILLOMANIA IN TERMS OF COGNITIVE BEHAVIOUR THERAPY

Charmaine Gordon

A thesis submitted to the Faculty of Health Sciences, University of the Witwatersrand, in fulfilment of the requirements for the degree of Doctor of Philosophy

Johannesburg, 2007

DECLARATION BY THE CANDIDATE

I, Charmaine Gordon declare that this thesis is my own work. It is being submitted for the degree of Doctor of Philosophy in the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at this or any other University.

_____

Charmaine Gordon

31st day of January, 2007

PUBLICATIONS AND CONFERENCE PRESENTATIONS

1 Regional cerebral perfusion pattern of ^{99m}Tc-HMAO SPECT in trichotillomania.
Berk, M., Gordon, C., Vangu, M.D.T., Esser, J.D., Boyd, I.H.
XXXIst CINP Congress. Glasgow, Scotland (1998)

2 Regional Cerebral Perfusion Pattern of tc-99m- HMPAO SPECT in trichotillomania.
Gordon, C., Vangu, MDT., Berk, M., Esser, J.D., Boyd, I.H.
The Journal of Neuropsychopharmagology 1999; 2: suppl. ; s215.

3 Trichotillomania : Self response from a South African population
Gordon, C. Berk, M.
Journal of Depression and Anxiety. 1999; 2:2

4 99mTc-HMPAO Brain SPET before and after Cognitive Behaviour Therapy in patients with trichotillomania
Gordon, C., Vangu, MDT, Boyd, IH. South African Society of Nuclear Medicine. 9th Biennial Congress. Bloemfontein, South Africa (2000)

5 99mTc-HMPAO Brain SPET before and after Cognitive Behaviour Therapy in patients with trichotillomania.
Gordon, C
Best Presentation Award. Department of Psychiatry – Research Day 2000

ABSTRACT

Trichotillomania was first defined over a hundred years ago as a self inflicted alopecia resulting from avulsion of hair. Previous Positron Emission Tomography (PET) studies have shown increased count density in the right superior parietal region of patients suffering from trichotillomania. It is unclear if this increase in count density might be a state or trait related marker of the disease. Research has indicated that Cognitive Behaviour Therapy can systematically modify cerebral metabolic activity which is significantly related to clinical outcome. In the case of Obsessive Compulsive Disorder (OCD), a decrease of metabolic activity has been demonstrated using Pet. The present study was undertaken to investigate whether similar metabolic changes as indicated by Single Beam Positron Emission Computerised Tomography. (SPECT), will be found in trichotillomania after Cognitive Behaviour Therapy.

Twelve patients diagnosed as suffering from trichotillomania, using DSM-IV criteria, underwent brain SPECT scanning using ^{99m}Tc-HMPAO. Scanning was performed before and after Cognitive Behaviour Therapy intervention. The psychotherapy was conducted in 12-16 sessions. The response to Cognitive Behaviour Therapy was assessed using the Psychiatric Institute Trichotillomania Scale and the Hamilton Anxiety Rating Scale. Ratings were completed at the beginning and end of the trial. The analysis of the scan data was done by comparing the left to the right superior parietal region. For each patient the region of interest was applied on the superior transaxial brain slices where the cingulate gyrus fully appeared. Each region of interest had the average number of counts normalised to the maximal cerebellar uptake.

Baseline studies showed a significant increased count of ^{99m}Tc-HMPAO in the right superior parietal areas compared to the same areas on the left ($p<0.0003$). This pattern normalised after Cognitive Behaviour Therapy, such that there was no significant difference between the right and left superior parietal areas in those patients who responded (n=9) to therapy. In the non-responder group (n=3), the pattern remained unchanged.

Using SPECT this study confirms previous reports of increased density in the right superior parietal lobe. In addition, it suggests that the increased count of ^{99m}Tc-HMPAO in the right parietal area is a state related disturbance in neurophysiology in this disorder, as it resolves with successful treatment. Of substantial importance is the fact that this study demonstrates a neurophysiological substrate and impact of psychotherapy. In conclusion this data suggests that the increased count density in the right superior parietal area is a state related marker of trichotillomania. ^{99m}Tc-HMPAO brain SPECT can therefore be used to monitor therapy of patients suffering from this disease.

ACKNOWLEDGEMENTS

The author extends her gratitude to the following individuals;

Prof M Berk, the initial project supervisor, for his unwavering trust, time, research and academic advise and encouragement.

Prof. C Szabo, final project supervisor, for his patience and objective guidance.

Prof. MDTHW Vangu for his valuable contribution to the neuro-imaging aspects of the study.

The staff of the Department of Nuclear Medicine for making available the radio active ingredient Ceretec, the use of the SPECT scanner and the acquisition of the imaging data.

Ms Botha of the Psychiatry out-patients department of the Johannesburg General Hospital for the efficient registering and booking of patients.

Prof. A Stuart, (RAU), for introducing the field of brain-behaviour and stimulating the interest in research

Mr K Bolon for his invaluable supervision and contribution to the Cognitive Behaviour Therapy aspect of the study.

The author extends a special thanks to Prof. HD Chandler without whose support, encouragement and patience this work would not have been possible.

DECLARATION **ii**

PUBLICATIONS AND PRESENTATIONS **iii**

ABSTRACT **iv**

ACKNOWLEDGEMENTS **v**

LIST OF FIGURES **vi**

LIST OF TABLES **vii**

TABLE OF CONTENTS **viii**

LIST OF FIGURES **ix**

LIST OF TABLES **x**

LIST OF FIGURES

2	Example of hair loss due to trichotillomania	9
3	Obsessive-compulsive related or spectrum of disorders	36
4.1	Generalized basal ganglia-thalamocortical circuits	46
4.2	Five proposed basal ganglia-thalamocortical circuits	47
8	^{99m}Tc-HMPAO	107
9.1	Representation of scores on the TCI-125	118
9.2	Regional 99mTc-HMPAO SPECT in trichotillomania demonstrating hyperperfusion in the right parietal region	123
9.3	Differences between regional uptake before (a) and after (b) Cognitive Behaviour Therapy in responders	126
9.4	No significant difference in levels of hyperperfusion between hemispheres in non-responders, before (a) and after (b) Cognitive Behaviour Therapy	127

LIST OF TABLES

2.1	Differences between early and later onset of trichotillomania	11
2.2	Methods used by trichotillomanic sufferers to resist hair pulling	13
2.3	Summary of Axis II disorders co-morbid in trichotillomania	19
2.4	Summary of Axis I disorders co-morbid in trichotillomania	22
6.1	Habit reversal training	71
6.2	Cognitive Behaviour Therapy model for trichotillomania	73
8	Cognitive Behavioural Therapy treatment programme	110
9.1	Subject demographic data	113
9.2	Subject illness history	114
9.3	Hair pulling sites in present study	116
9.4	Results obtained from the TCI-125	117
9.5	Results of the comparison of the differences between the right and the left superior parietal area at baseline SPECT acquisition	122
9.6	Second analysis. Group that responded. Comparison of the differences between the two hemispheres, using only one pair of region of interest before and after treatment	125
9.7	Second analysis. Group that did not respond. Comparison of the differences between the two hemispheres, using only one pair of region of interest, before and after treatment	127
9.8	Second analysis. Comparison of relative counts in one hemisphere before treatment with those in the same hemisphere after treatment – Group that responded	129
9.9	Comparison of total scores before and after Cognitive Behaviour Therapy on the Psychiatric Institute Trichotillomania Scale (PITS)	131
9.10	Comparison of anxiety rating before and after Cognitive Behaviour Therapy	133

1. INTRODUCTION 1

2. GENERAL OVERVIEW OF TRICHOTILLOMANIA 4

2.1 Definition 4
2.2 An overview of the history of trichotillomania 5
2.3 The clinical picture of trichotillomania 9
2.4 The heterogeneity of trichotillomania 13
2.5 Etiology and epidemiology of trichotillomania 15
2.6 Psychiatric co-morbidity and personality traits in trichotillomania 17
2.6.1 Personality traits in trichotillomania 17
2.6.2 Temperament 19
2.6.3 Psychiatric co-morbidity in trichotillomania 20
2.7 Parallels of trichotillomania in animal models 23

3. THE CLASSIFICATION OF TRICHOTILLOMANIA 29

3.1 Impulse control disorders 29
3.2 Trichotillomania and obsessive-compulsive disorders 31
3.2.1 Similarities and differences between trichotillomania and obsessive-compulsive disorder 32
3.3 Trichotillomania as part of the OCD spectrum of disorders (OCD's) 35
3.4 Overlap of OCD and OCD spectrum of disorders 37
3.5 Trichotillomania and Tourette syndrome 38

4. THE NEUROBIOLOGICAL BASIS OF TRICHOTILLOMANIA 40
4.1 Neurochemistry 40
4.2 Neuroanatomy and neurophysiology 43
4.2.1 Positron emission tomography in trichotillomania (PET) 43
4.2.2 Neurocircuitry and the basal ganglia in terms of the OCD spectrum of disorders 45
4.2.3 Functional magnetic resonance imaging in trichotillomania and related OCD spectrum of disorders 50
4.3 SPECT (Single Beam Positron Emission Computerized Tomography and trichotillomania 55

5. THE NEUROPSYCHOLOGY OF TRICHOTILLOMANIA 56

5.1 Trichotillomania 56
5.2 Differences between trichotillomania and OCD 60
6. THE TREATMENT OF TRICHOTILLOMANIA 62
6.1 Pharmacological treatment 63
6.2 Hypnosis 65
6.3 Psychoanalytic and psychodynamic viewpoint 66
6.4 Behavioural therapy 69
6.4.1 Behavioural therapy and obsessive-compulsive spectrum disorders 72
6.5 Cognitive Behaviour Therapy 72
6.5.1 Cognitive Behaviour Therapy and the treatment of the OCD spectrum of disorders 74
6.6 General treatment outcome 76

7. THEORY OF COGNITIVE BEHAVIOUR THERAPY 78

7.1 Historical background 78
7.2 Cognitive theory of personality 81
7.3 Methodological assumptions 83
7.4 Cognitions as a target of change 87
7.5 Cognitive Behaviour Intervention methods 91
7.5.1 Cognitive restructuring 92
7.5.2 Problem solving 94
7.5.3 Self-instruction training 95
7.6 Research hypothesis 98

8. SUBJECTS, PROCEDURE AND METHODS 100

8.1 Study design 100
8.2 Subjects 100
8.2.1 Criteria for selection 100
8.2.2 Subject sample 101
8.3 Procedures 101
8.3.1 Ethical clearance 102
8.3.2 Informed concent 103
8.3.3 Rating scales 103
8.3.3.1 Rating the severity of trichotillomania 103
8.3.3.2 Rating the level of anxiety 104
8.3.3.3 The Temperament and Character Inventory-125 (TCI-125) 104
8.3.4 Acquisition of the first or baseline

^{99m}Tc-HMPAO SPECT scan 107
8.3.5 Cognitive Behaviour Therapy programme (CBT) 109
8.3.6 Acquisition of the second ^{99m}Tc-HMPAO SPECT scan 110
8.3.7 Statistical analysis 111

9. RESULTS 112

9.1 Subjects 112
9.2 The Temperament and Character Inventory-125 (TCI-125) 117
9.3 Results of the comparison of the differences between the right and left superior parietal area at baseline SPECT acquisition 121
9.4 Second analysis – ^{99m}Tc-HMPAO SPECT after Cognitive Behaviour Therapy intervention 124
9.4.1 Comparison between the right and left hemisphere , before and after treatment 124
9.4.2 Comparison of the same hemisphere before and after treatment 128

9.5 Comparison of total score before and after treatment on the Psychiatric Institute Trichotillomania Scale (PITS) 129
9.6 Comparison of total scores before and after treatment on the Hamilton Anxiety Rating Scale 132

10. DISCUSSION 134

11. CONCLUSIONS AND RECOMMENDATIONS 140

11.1 Conclusion and hypothesis testing 140
11.2 Weaknesses of the study and future research 142

REFERENCES 145

APPENDIX 190

A1. Ethics clearance certificate 190
A2. Information and consent form 191
A3. Psychiatric Institute Trichotillomania Scale (PITS) 193
A4. The Hamilton Anxiety Rating Scale 194
A5. The Temperament and Character Inventory - 125 (TCI-125) 195

CHAPTER ONE

INTRODUCTION

Trichotillomania (TTM) otherwise known as compulsive hair pulling is a psychiatric disorder, which although first defined more than a hundred years ago has only relatively recently become a subject of scientific focus. Whereas the clinical manifestations have been well described in the literature there is a paucity of data regarding the underlying pathophysiological features of trichotillomania. Although some treatment models have been published more scientific validation is needed.

Due to the clinical and pharmacological similarities between trichotillomania and obsessive-compulsive disorder (OCD) it has been postulated that the pathophysiological pattern of trichotillomania as well as the Cognitive Behaviour Therapy intervention thereof could also be similar to that of OCD. Single-beam Positron Emission Computerized Tomography (SPECT) has not yet been utilized to investigate trichotillomania. It has furthermore not yet been documented whether Cognitive Behaviour Therapy could effect changes at the neurophysiological level in trichotillomania after successful treatment.

In this study an attempt is made to demonstrate that by using SPECT an altered neurophysiological substrate exists in trichotillomania at baseline and furthermore that clinically successful Cognitive Behaviour Therapy intervention can effect detectable SPECT changes at a neurophysiological level. The study methodology will be described in the chapters to follow, as well as a general description of chapter content.

Chapter 2 gives a general overview of trichotillomania in terms of its definition, diagnostic criteria, clinical picture, history, heterogeneity, etiology, epidemiology and parallels in animal models.

Chapter 3 describes the classification of trichotillomania, lists the similarities between trichotillomania and OCD and presents the arguments for classifying trichotillomania as part of the Obsessive Compulsive Spectrum of disorders as set out in the scientific literature.

In Chapter 4 the neurochemistry, neuroanatomy and neurophysiology of trichotillomania are described as well as the role that the basal ganglia and neurocircuitry seems to plays in the Obsessive Compulsive Spectrum of disorders. Neuroimaging such as SPECT, PET and *f*MRI are reviewed. The neurobiology of trichotillomania is described in context of the Obsessive Compulsive Spectrum of disorders.

Chapter 5 reviews the neuropsychology of trichotillomania.

In chapter 6 psychiatric co-morbidity and personality traits in trichotillomania are listed.

In chapter 7 the treatment of trichotillomania is discussed.

Theory of Cognitive Behaviour Therapy follows in chapter 8.

In chapter 9 the methods employed in this study are discussed.

In chapter 10 the results of the study are presented.

A discussion of the findings of the present study follows in chapter 11.

Chapter 12 presents the conclusions and recommendations for future studies.

CHAPTER TWO

GENERAL OVERVIEW OF TRICHOTILLOMANIA

2.1 Definition

The term trichotillomania or compulsive hair-pulling was first used over a hundred years ago, by the French dermatologist Hallopeau (1889). He defined trichotillomania, Greek for hair (thrix), to pull out (tillein) and insanity or frenzy (mania), thus Ahair-pulling madness≅ (Rothbaum and Ninan, 1994) as a self-inflicted alopecia resulting from avulsion of hair (Dean, Nelson and Moss, 1992). Other terms such as trichopilomania (Taylor, 1963), hair plucking (Sticher, Abramovitz and Newcomer, 1980), trichologia (Stedman=s Medical Dictionary, 1982), trichotillohabitus (Jillson, 1983), chronic hair pulling (Christenson, Mackenzie and Mitchell, 1991), trichotillosis (Tahan and Fogt, 1992) and compulsive hair pulling (Schlosser, Black and Blum, 1994) have been suggested in the literature.

According to the DSM-IV (American Psychiatric Association, 1994), the essential feature of trichotillomania is Athe recurrent failure to resist impulses to pull out one=s own hair, resulting in a noticeable hair loss≅. A further diagnostic requirement is that the hair pulling is preceded by an increasing sense of tension that gives way to a sense of relief or gratification once the hair has been pulled out.

2.2 An overview of the history of trichotillomania

Compulsive hair pulling has probably been around as long as there have been living beings with hair to pull. The earliest medical reference to hair pulling was by Hippocrates who wrote in *Epidemics I;*

>Then we must consider his speech, his mannerisms, his silences, his thoughts, his habits of sleep or wakefulness and his dreams, their nature and time. Next we must note whether he plucks his hair, scratches or weeps=. (Lloyd, 1983, p. 100).

In the *Epidemics III*, Hippocrates described what seems to be the first case report of trichotillomania, in the context of severe depression and grief. He wrote;

>At Thasos the wife of Delearces, who lay on the level ground, took a high fever with shivering as the result of grief. From the start she used to wrap herself up, always remaining silent while she groped about, scratching and plucking out hair, and alternatively wept and laughed= (Lloyd, 1983, p.137).

Another early reference to hair pulling is found in the Bible in the book of Ezra (9.3) in the Old Testament, where Ezra describes:

>and when I heard this thing, I rent my garment and my mantle, and plucked off the hair of my head and of my beard=. (Hodder and Stroughton **eds.**, 1984 p. 49)

Hair pulling was first given the status of a classifiable symptom by Hallopeau (1889), to describe a young man who was plucking out his hair in tufts (Damodaran, Jayalekshmi and Khanna, 1995). For the first part of the previous century only a few references were made to trichotillomania in the literature. Greenberg and Sarner

(1965) state that although dermatologists continued to point out the probable psychogenic etiology of the symptom, the psychiatric literature on the subject remained scant. In a literature review they found that only ten references dealt with trichotillomania and that only 28 cases were recorded at length. However, for each decade from the 1940's through the 1980's the number of articles has virtually doubled (Graber and Arndt, 1993). Until recently trichotillomania has remained on the periphery of psychiatrist's awareness. Many psychiatrists have never seen the extent of hair loss that may be associated with this condition (Winchel, 1992). Perhaps as a reflection of insight into the inappropriateness of the behaviour, trichotillomania sufferers hide their affliction and do not seek treatment and this secrecy may explain why the disorder had been poorly studied and understood(Swedo and Leonard, 1992).

As far as can be determined, the first systematic study of the symptoms and syndrome of trichotillomania, was undertaken by Greenberg and Sarner (1965) who evaluated 19 cases of this illness between 1960 and 1963. They described their results in terms of psychoanalytic theory and viewed the symptoms as related to problems in psychosexual development (Greenberg, 1969). In contrast, behavioural theories viewed hair pulling as an anxiety-reducing habit comparable to thumb sucking, nose picking or fingernail biting (Azrin and Nunn, 1973). The behavioural literature suggested that behaviour therapy for trichotillomania routinely results in some level of success (Friman, Finney and Christophersen, 1984).

While medications have been used to treat psychiatric disorders for the past 40 years, it is only within the last decade that significant strides have been made in the pharmacotherapy of trichotillomania (Keuthen, O=Sullivan, Sprich and Buckminster,

1998). The first controlled drug treatment trial in which clomipramine and despiramine were compared was undertaken by Swedo, Leonard, Rapoport, Lenane, Goldberger and Cheslow (1989), as will be discussed further. Since then the effect of other Selective Serotonin Re-uptake Inhibitors (SSRI) antidepressants (Koran, Ringold and Hewlett, 1992 ; Stanley, Breckenridge and Swann, 1997), neuroleptics (Stein and Hollander, 1992) mood stabilizers (Christenson, Popkin, Mackenzie and Realmuto, 1991) and opiate antagonists (Mahr, 1993) have been undertaken. These will be described in more detail later.

Very little is known about the neurophysiology of trichotillomania. The first and only study using positron emission tomography (PET) to investigate trichotillomania was undertaken by Swedo, Rapoport, Leonard, Shapiro and Grady, (1991). In this study 10 woman suffering from trichotillomania were compared to 20 control subjects. A striking, cerebral glucose hyper metabolism and increased normalized regional metabolic rates in the right superior parietal region and the right and left cerebella were found (see detailed description further on). No study has been undertaken to investigate trichotillomania using SPECT.

Following the previous research, two more studies were published in the literature, both using magnetic resonance imaging (MRI) as an investigative tool. One study (Grachev, 1997), using MRI-based morphometric topographic parcellation in a sample of 10 patients compared with 10 normal subjects, found that the trichotillomania patients exhibited a significantly reduced left inferior frontal gyrus volume as well as a reduction in the cuneal cortex. The other study (O=Sullivan, Rauch, Breiter, Grachev, Baer and Kennedy, 1997) using the same sample group and

acquisition as the previously mentioned study, reported a decreased left putamen volume in the trichotillomania group.

Few neuropsychological investigations have been undertaken to determine neuropsychological functioning in trichotillomania. As far as can be determined only three neuropsychological studies of trichotillomania have been published. The first study compared 21 trichotillomania patients with a control group as well as a group of OCD sufferers and a mixed anxiety and depression group (Rettew, Cheslow, Rapoport Leonard, Lenane, Black and Swedo, 1991). This study was limited to two neuropsychological tests and found visual-spatial dysfunction in the trichotillomania group. A second study comprising 20 trichotillomania patients compared with a control group, utilized five neuropsychological tests and indicated visual-spatial memory difficulty (Keuthen, Savage O=Sullivan, Brown and Shera, 1997). The third study (Stanley, Hannay and Breckenridge, 1997) compared 20 trichotillomania patients to a control group using an extensive neuropsychological battery. These researchers reported dysfunction in speed and motor tests. They explain their findings in terms of anxiety. The neuropsychology of trichotillomania will be described in full in a further chapter.

2.3 The clinical picture of trichotillomania

Hair may be pulled from any part of the body, but the most common sites for pulling are the scalp (Figure 2), eyelashes, eyebrows and pubic area. The extent of hair loss may vary from one coin sized area of alopecia to several. Hairs are usually completely pulled out of the follicle, but broken shafts may be present. Re-grown hair generally appears normal but due to repeated damage to the follicle, new hairs may be coarser and curlier than the original hair (Muller and Winkelmann, 1972). Most of the time hair pulling occurs in solitude, with the exception that sometimes hair can be pulled in the presence of core family members. Hair pulling may be performed during periods of relaxation and distraction as well as stress related situations (Winchel, 1992). The episodes of hair pulling may last from a few seconds to a few hours (Swedo, 1993).

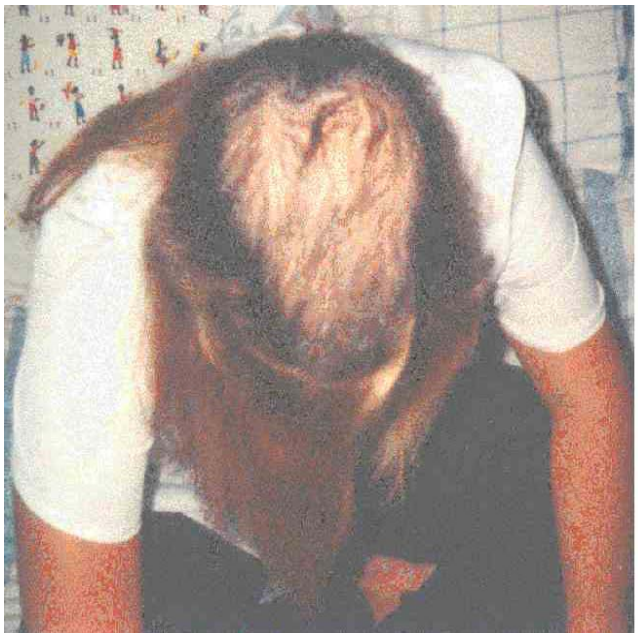


Figure 2 Example of hair loss on the scalp. The patient is compelled to pull out hair, one strand at a time. (Photo reproduced by permission – subject number 11)

More than half of patients prefer to pull hairs that possess certain textural qualities (Mansueto, 1990). Such hairs are usually described as >thick=, >coarse=, >stubby= or >kinky=. Since continual hair pulling can lead to textural hair changes, initial pulling

of normal hair may promote future pulling via the production of increasing numbers of >just right= hairs (Christenson and Crow, 1996).

As trichotillomania sufferers mostly keep their disorder a secret out of embarrassment or fear of being thought mentally ill, they often seek treatment for secondary symptoms such as depression, anxiety and dermatological diseases (Greenberg and Sarner, 1965). The patient often denies the self-induced nature of the disorder and parents of trichotillomania children may also demonstrate denial or rationalization (Muller,1987). The clinical course of trichotillomania is usually characterized by exacerbation and remission and, in most instances, the disorder is chronic (Krishnan, Davidson and Guajardo, 1985). Muller (1987) reported that trichotillomania can usually be diagnosed or at least strongly suspected from the clinical presentation. However, it remains a diagnosis of exclusion (Adam and Kashani, 1990).

Trichotillomania presents in two forms: early childhood onset or later onset. (Table 2.1) When trichotillomania begins between the ages of 2 and 6 years, it is usually a mild condition with a duration of several weeks to several months, often responding to simple interventions, (Winchel, 1992). Dawber, (1985), found male predominance in early childhood. Later onset between the ages of 11 and 20, may be severe, chronic and last for decades. In the later onset group it seems that females outnumber males 4 to 1 (Ratner, 1989).

Table 2.1 Differences between early and later onset trichotillomania (Winchel, 1992)

	EARLY ONSET	**LATE ONSET**
Age of onset	Infant and toddler to about age 6	Approx. 6 years and up, may

		peak around age 13 years
Gender ratio	Frequency among girls and boys similar	Females seem to greatly outnumber males
Duration	Often brief (weeks to several months)	May last up to decades
Prognosis	Often responds to simple interventions	Unclear, but for some, refractory to all treatments
Association with other pathology	Increased developmental and family pathology has been suggested, but no convincing evidence available. May be associated with other habits Eg. Thumb-sucking)	Apparent high lifetime incidence of mood and anxiety disorders

Oral behaviours such as such as running the hair along the lips, mouth or teeth are common amongst trichotillomania sufferers. Between 5% and 18% of patients with trichotillomania ingest parts of hair or whole hair (trichophagia), (Christenson et al., 1991). Complete chewing occurs in 33% and hair ingestion in 10% of trichotillomania patients, and some form of oral manipulation of pulled hair occurs in 48% of patients. The most serious consequence of trichotillomania is the development of trichobezoars (hair balls) or trichophytobezoars (clots of hair and vegetable matter), when patients eat the pulled out hair. This results in intestinal obstruction, gastro-intestinal bleeding, bowel perforation, acute pancreatitis, and obstructive jaundice (Dean, et al., 1992), and can lead to mortality if untreated (Christenson. Ritzvedt and Mackenzie, 1993). Another variant, although rare, is the "Rapunzel Syndrome', in which twisted masses of long hair may stretch from the stomach as far down as the colon (Winchel, 1992). The primary dermatological complication involved with trichotillomania is that of alopecia areata with fungal infections (Greenberg and Sarner, 1965 ; Muller, 1987).

It is reported that trichotillomania in childhood starts as a habit disorder, seen in association with thumb sucking, nail biting, nose picking (Oranje, Peereboom-Wynia and DeRaeymaker, 1986) and compulsive masturbation (Blount and Finch, 1987). Severe depression, anxiety, borderline personality, academic problems and over-concern with weight have been observed in association with trichotillomania (Adam and Kashani, 1990; Christenson, 1995). However, most recent research (as will be described in much more detail later), associates trichotillomania with the Obsessive-compulsive spectrum of disorders, which include tics (Lutzker and Lamazor, 1985), Gilles de la Tourette syndrome (Stein and Hollander, 1992), body dysmorphic disorder (Hollander, Liebowitz and Winchel, 1989) and OCD (Swedo, et al.,1989 ; Jenike, 1989).

Trichotillomania sufferers attempt several methods to resist hair pulling. In their study of 60 patients with trichotillomania, Christenson et al., (1991a) summarised the most common methods of resistance. (Table 2.2).

Table 2.2 **Methods used by trichotillomania sufferers to resist hair pulling (Christenson et al., 1991a)**

	Method	%

Barrier on head	20	33
Change in activities	13	22
Keeping hands occupied	12	20
Wearing mittens / gloves	12	20
Sitting on hands	9	15
Taping fingers together	5	8
Altering fingernails	5	8
Hiding tweezers	4	7
Chewing gum	3	5
Petroleum jelly on hands or hair	3	5

2.4 The heterogeneity of trichotillomania

Although the diagnostic criteria for trichotillomania suggest homogeneity amongst sufferers, phenomenological and descriptive studies have suggested that hair pulling is heterogeneous (Christenson and Crow, 1996). According to Christenson, Mackenzie and Mitchell (1991), 17% of 60 chronic hair pullers did not meet the mounting tension and or tension reduction/gratification criteria for trichotillomania despite clinically meaningful alopecia and distress. In a study of 10 children and adolescents presenting with the symptoms of trichotillomania, Reeve, Bernstein and Christenson (1992) noted that, on similar grounds, none of these patients would have met diagnostic criteria.

Two types of hair pulling behaviour have been described in the literature (Christenson et al., 1993; Winchel, 1992). One group of hair pullers tend to focus their attention on the

actual pulling that is taking place at that moment. They are usually distracted from other thoughts and activities by pulling, and usually describe the mounting tension and tension reduction qualities as described frequently in the literature. Approximately 25% of patients describe this as their predominant style of pulling (Christenson et al., 1996).

However, the majority of trichotillomania sufferers describe most of their behaviour as an automatic reaction that is characterized by hair pulling that occurs parallel to common high risk, sedentary or contemplative hair pulling situations such as reading, studying, watching television, driving, speaking on the phone or lying in bed at night (Winchel, 1992). Hair pulling is thus performed as if out of awareness or in only partial awareness (Azrin and Nunn, 1977 ; Mansueto, 1990). This lack of insight into involvement with the hair is referred to as A*belle indeference*≅ (Schnurr and Davidson, 1989). Some patients refer to hair pulling as a >trans-like= state. However, whether the hair pulling at times can represent a dissociative state, has not been adequately researched (Stein, Christenson and Hollander, 1999). This group of hair pullers also shares phenomenological similarities with the experience of obsessions and compulsions, and its presence in study populations may explain observed relationships between trichotillomania and OCD (Christenson, et al., 1993).

The site of pulling further contributes to heterogeneity in trichotillomania. Whereas hair pulling mostly involves the hair on the scalp, hair can also be pulled from the eyebrows, eyelashes, pubic hair or any other part of the body, either on its own or in combination (Muller and Winkelman, 1972).

The hair pulling behaviour differ between males and females. The onset of trichotillomania in males is usually at a younger age and follows a different pattern as will be described further (Ratner, 1989).

2.5 Etiology and epidemiology of trichotillomania

The true prevalence of trichotillomania is difficult to determine as most sufferers are secretive and make efforts to conceal the effects of the disorder (Rothbaum and Ninan, 1994). Few researchers have made estimates of the prevalence of trichotillomania. Ini-tially researchers surmised that trichotillomania is a relatively rare disorder. Earlier reports (Anderson and Dean, 1956) found three (0,6%) out of 500 patients at a child guidance clinic suffered from trichotillomania, and Schacter (1961) found only five (0,05%) of 10 000 children with psychiatric disorders, could be diagnosed with trichotillomania. Manino and Delgado (1969) found that only seven (0, 5%) out of 1368 patients at their health centre suffered from trichotillomania.

However, Azrin and Nunn (1977) estimated that as many as 8 million Americans (2-3%) might be affected. This would make trichotillomania as common as OCD. Kaplan (1990) estimated that between 2 and 8 million (1% to 4%) of Americans engage in this behaviour. Christenson, Pyle and Mitchell (1991) indicated that trichotillomania might be more common than was previously estimated. In a survey of 2579 female college students they found that, provided that the DSM-III-R criteria were not strictly adhered to by omitting the tension-reduction aspects, the rates would increase to 3,4% in females and to 1,5% in males. More recently Rothbaum, Shaw,

Morris and Ninan, (1993) indicated that, from their study amongst college students, 10-13% engage in hair pulling.

The etiology (cause) of trichotillomania is unknown (Swedo, 1993; Minichiello, O=Sullivan, Osgood-Hynes and Baer, 1994). A variety of etiologic mechanisms have been proposed and supported by data from small samples but have not been definitively established. For example, preliminary data suggest that some trichotillomania may be familial, whereas other studies demonstrate neuropsychological abnormalities or psychosocial dysfunction (Swedo, 1993). The importance of genetic or familial influences is unclear (Christenson, Mackenzie and Reeve, 1992). Investigators have found higher than expected rates of OCD in the first degree relatives of individuals with trichotillomania (Lenane, Swedo, Rapoport, Leonard, Screery and Guratt, 1992; King, Scahill, Vitulano, Schwab-Stone, Tercyak and Riddle et al., 1995).

At present no single theory appears to explain trichotillomania. The possible existence of various types of compulsive hair pulling behaviour with differing etiologies could account for the phenomenological differences in expression of the disorder, the heterogeneity of treatment response and the neuropsychological and neuroimaging data described (Minichiello et al., 1994).

Swedo (1993) as well as Swedo and Leonard (1992) propose an ethologic model for trichotillomania. They hypothesize that trichotillomania (as well as some other OCD related, or spectrum of disorders, such as nail biting), may represent pathological variants of normal grooming behaviours. Phenomenologically the stereotyped nature of the pulling, the way it is triggered by feeling the stubble of new hair growth and the

similarity of presentations, imply a fixed action pattern or pre-programmed behaviour that has been inappropriately released. These fixed action patterns are typically inhibited in humans. However, Swedo and Leonard (1992) hypothesize that in the pathological grooming compulsions, either the drive state is altered so the behaviour is no longer inhibited, or the key releasing stimulus causes inappropriate release of the ritualized behaviour. In trichotillomania and OCD, the drive state or the key releasing stimulus could be over stimulated by some common mechanism, which is normalised by the serotonin reuptake blocking group of drugs.

It has also been hypothesized that the basal ganglia as well as the fronto-cortical pathways are involved in both OCD and trichotillomania, as will be described in detail later on. It is postulated that damage to the basal ganglia caused by Streptococcus might leads to increased incidences of Sydenham=s chorea, OCD and hair pulling (Swedo, et al.,1987 ; Swedo, 1989b ; Stein et al., 1997).

2.6 Psychiatric co-morbidity and personality traits in trichotillomania

2.6.1 Personality traits in trichotillomania

Similar to most other areas in trichotillomania, not much research has been conducted to investigate personality characteristics of patients suffering from trichotillomania. An early study (Galski, 1983) suggested that trichotillomania sufferers seem to have borderline personality organization, poor frustration tolerance, poor impulse control, poor object constancy and ego boundary difficulties. The study was limited by a small sample size.

A further three studies utilizing formal DSM-III-R criteria conducted by Swedo and Leonard (1992), Schlosser (1994) and Christensen, Chernoff-Clementz and Clementz (1992a), yielded fairly similar results to each other. A summary of these results appear in Table 2.3.

Mansueto (1990) investigated psychopathological co-morbidity in these patients by summarising what the patients reported when interviewed. He found that 84% of patients reported difficulties with low self esteem, 82% experienced a diminished sense of attractiveness and 80% experienced some sort of shame or embarrassment. Secondary effects to the trichotillomania that were reported included guardedness (61%), loss of spontaneity (39%), loss of sleep (32%), irritability (23%) and feeling >weird= (11%). Furthermore he found that 66% of participants in his study reported depressive feelings, while as high as 68% of patients reported feelings of anxiety.

Table 2.3 **Summary of Axis II disorders co-morbidly in trichotillomania (Christenson et al., 1992a)**

SIPD = The Structured Interview for Personality Disorders SIPD-R = The Structured Interview for DSM-III-R Personality Disorders	Swedo and Leonard (1992)	Schlosser (1994)	Christensen, et al. (1992a)
	SIPD	SIPD-R	SIPD-R
	n = 43 %	n = 22 %	n = 48 %
Histrionic	26		14.6
Borderline	18	14	2.1
Passive-aggressive	16		6.3
Dependent	5		8.3
Schizoid	2	14	2.1
Narcissistic	2		0
Avoidant	2	14	10.4
OCD		27	8.3
Paranoid			4.2

2.6.2 Temperament

Papalia and Olds (1988) describes temperament as a person's characteristic style of approaching people and situations. These styles seem to be inborn and to remain stable in adulthood. From a long-term follow-up study, Kagan (1982) describes nine temperamental elements namely; activity level, regularity in biological functioning (sleeping, eating, eliminating), readiness to accept new people and situations,

adaptability to change, intensity of responses, sensitivity to noise, light and other sensory stimuli, mood (cheerfulness or unhappiness), distractibility and persistence.

Clonninger (1986) proposed a biosocial theory of personality which describes four elements of temperament which are termed Novelty Seeking, Harm Avoidance, Reward Dependence and Persistence. This will be discussed in more detail in chapter 8. A study undertaken by Lochner, Seedat, Du Toit, Nel, Niehaus and Stein (2005) indicated that higher scores on Novelty Seeking and Harm Avoidance were present in a group of 49 females suffering from trichotillomania.

2.6.3 Psychiatric co-morbidity in trichotillomania

An early study (Greenberg and Sarner, 1965), comprising 19 trichotillomanic patients indicated that two of the patients were diagnosed as suffering from a >schizophrenia reaction=, 7 with a >borderline= state, 3 with mixed psychoneurosis and 7obsessive-compulsive psychoneurosis. Furthermore, 13 patients (68%) were moderately to severely depressed, 9 or 47% were pre-occupied with somatic concerns and 12 or 63% were overly concerned with their weight. Fourteen patients (74%) developed moderate to severe difficulties in school subsequent to onset of hair pulling, including over-meticulousness and diminished performance. Given that the research was conducted prior to operationalised criteria the results cannot be compared to more recent findings.

In a more recent study with 43 children , adolescents and adults suffering from trichotillomania, Swedo et al., (1992) found that as far as comorbidity was concerned,

39% of their patients demonstrated unipolar depression, 32% showed symptoms of generalised anxiety disorder, 16% OCD, 15 % substance abuse, 5% bipolar disorder, 5% panic disorder, 5% anorexia or bulimia and 2% phobic disorders.

Christenson (1995) conducted intensive interviews with 186 trichotillomanic sufferers. Axis I diagnoses were made using the Minnesota Trichotillomania Assessment Inventory, which incorporates a partial checklist for DSM-III-R diagnostic criteria. Their findings are summarised in Table 2.4. Considering the percentage of the disorders mentioned, it seems that both the affective disorders (59.1 %) and the anxiety disorders (82.9%) are very prominent in trichotillomania.

As far as can be determined no formal study to investigate the role of anxiety in trichotillomania has been conducted. However, as can be seen from the previously mentioned studies (Swedo et al, 1992; Schlosser et al, 1994 ; Christensen et al, 1992a), high levels of anxiety are consistently reported. Anxiety was also considered a co-morbid factor in the parallel animal models discussed previously. In the neuropsychological investigation previously described, Stanley, et al. (1997b) provided support for the notion that trichotillomania may be best understood as an anxiety or affective-based disorder.

Table 2.4 Axis I disorders co-morbid in trichotillomania (Christenson, 1995).

Diagnosis	*N*	*Proportion of sample (%)*
Mood disorders		
Major depression	**96**	**51.6**
Dysthymia	**9**	**4.8**
Bipolar Disorder	**0**	**0**
Bipolar disorder (NOS)	**5**	**2.7**
Psychotic disorders		
Schizophrenia	**0**	**0**
Scizoaffective Disorder	**2**	**1.1**
Anxiety disorders		
Panic disorders without agoraphobia	**10**	**5.4**
Panic disorder with agoraphobia	**10**	**5.4**
Agoraphobia	**3**	**1.6**
OCD	**25**	**13.4**
Generalized anxiety disorder	**50**	**27.0**
Social phobia	**21**	**11.3**
Simple phobia	**35**	**18.8**
Eating disorders		
Bulimia nervosa	**15**	**8.1**
Anorexia nervosa	**3**	**1.6**
Eating disorders (NOS)	**21**	**11.3**
Substance abuse / dependence		
Alcohol	**36**	**19.4**
Other substances	**30**	**16.1**
Tic disorders		
Tourette=s disorder	**1**	**0.005**
Chronic motor tic	**6**	**3.2**

As with OCD, where ritualistic behaviour has an anxiety reduction function the hair pulling behaviour in trichotillomania seems to have a similar effect on reducing anxiety (Christenson et al, 1991; Mansueto, 1991). It has been reported that the tension that precipitates hair pulling may relate to specific thoughts about hair, but it frequently reflects more general feelings of arousal without clear situational or

cognitive precipitants (Stanley and Cohen, 1999). Other affective states such as boredom, anger, depression, frustration, indecision and fatigue have also been noted in trichotillomania (Christenson, 1993; Mansueto, 1991). Furthermore some patients report that hair pulling actually creates arousal and is energizing, whereas others describe a more dissociative state produced by the behaviour (Stanley et al., 1999).

2.7 Parallels of trichotillomania in animal models

A specialized branch of comparative psychology is concerned with investigating the similarities among behavioural phenomena in human beings and selected animal species (Bordnick, Thyer and Ritchie, 1994). A neuroethological model of trichotillomania, portraying the disorder as a disruption of the normal grooming response present in all primates, has been proposed by several authors (Keuthen, et al., 1998; Minichiello et al., 1994; Bordnick et al., 1994; Swedo and Leonard, 1992). Animal models of stereotypical compulsive behaviours such as those noted in acral lick in dogs, crib biting in horses, psychogenic alopecia in cats and feather picking in birds may have some relevance in a conceptual understanding of trichotillomania as falling within the spectrum of OCD (Bordnick et al., 1994; Minichiello et al. 1994; Stein, Shoulber, Helton and Hollander, 1992).

Psychogenic alopecia is a self-grooming abnormality that is initiated or intensified by non-organic causes, and results in large areas of the cat=s body becoming denuded of hair. It appears to be more prevalent in strictly indoor cats in comparison to those cats which are given access to the outdoors. It is not known whether cats in the wild suffer

from this condition, but it has been observed that wild cats in captivity display symptoms (Kenny, 1994). According to Stein et al, (1999), clinical experience indicates that many cats suffering from psychogenic alopecia seem to have anxious temperaments or have been exposed to situations that induced conflict or frustration. Medical or psychological causes may be involved in the initiation of psychogenic alopecia, but the licking behaviour may persist following resolution of the medical condition (Young and Manning, 1984). Cats showing evidence of psychogenic alopecia usually have a history of exposure to a potentially psychologically disturbing incident or change in their environment (Voith and Borchelt, 1996). Chronic frustration or conflict leads to stress and environmental pressures, including social interaction and husbandry issues, loss of a family member, change in the owner=s daily routine, a move to new surroundings or physical change in the same environment. Boarding, hospitalization, social stress and dominance disputes in a household with multiple cats and territorial aggression may cause anxiety and trigger hair pulling (Merchant, 1994; Muller, Kirk and Scott, 1989). Some success has been achieved by treating these cats with anxiolytics (Kunkle, 1995 ; Merchant, 1994), opioid and dopamine antagonists (Kenny, 1994) and serotonin reuptake inhibitors (Dodman, 1994). While all demonstrate initial symptomatic improvement, most do not have a permanent or lasting effect (Stein et al., 1999).

Canine acral lick dermatitis is a psychogenic disorder of dogs characterized by repetitive licking and chewing at the distal extremities of either fore or hind limbs, resulting in ulceration and, in some cases, secondary infection (Stein., Dodman and Borchelt, 1994). As with avian and feline abnormal grooming behaviours, true

psychogenic acral lick dermatitis is not reported to occur in free-ranging wild canine populations and appears to be a condition that is associated with the various stressors encountered in captivity (Stein et al., 1999). Breeds more frequently affected are those that work closely and form strong attachments to humans, so that it is not surprising that some dogs with acral lick dermatitis also exhibit separation anxiety or other anxiety-related conditions. In a clinical study at Tufts University Behaviour Clinic, it has been shown that approximately 70% of the dogs diagnosed with acral lick dermatitis have co-morbid fear and anxiety based conditions, such as noise phobia, separation anxiety, or anxiety-related territorial aggression. It appears that these dogs express their anxieties as displacement grooming that proceeds unchecked either because of the self-reinforcing nature of the behaviour or because the consummatory response does not produce the normal satiation (Moon-Fanelli, Dodman, O=Sullivan, 1998). As in the case of psychogenic alopecia in cats, the treatment of acral lick dermatitis in dogs demonstrates initial improvement but relapse after medication (such as described above) was withdrawn (Stein et al., 1999).

Kennedy and Draper (1991) suggested that feather picking disorder represents an exaggeration of normal preening/grooming behaviour. According to Harrison (1986) Feather picking generally applies to all mutilation of the feathers by the beak and includes chewing or plucking. Clinical observations that many birds with feather picking disorder engage in biting the end off the feathers or play with and chew the plucked feathers have been reported (Bordnick et al. (1994). Psychogenic feather-picking in birds in the wild has not been reported but seems to be common in captive birds, specifically those that are social (Grindlinger and Ramsay, 1991). For birds,

captivity may promote chronic conflict, and what may begin as a displacement activity can develop into a grooming abnormality with serious physical consequences. As with psychogenic alopecia and canine acral lick dermatitis, several medical conditions such as allergies, endo-parasites, ecto-parasites, infectious dermatitis or folliculitis, systemic disease, malnutrition and poor environmental conditions may give rise to the symptoms (Iglauer and Rasmin, 1993 ; Rosenthal, 1996). However, it is often observed that despite resolution of the underlying medical condition, feather picking may continue (Johnson, 1987). A variety of environmental conditions has been hypothesized to produce conflict in captive birds. These include environmental changes, lack of mental or physical stimulation, overcrowding or social isolation, separation anxiety, territorial and dominance disputes in multi-bird aviaries and the lack of natural outlets for sexual and breeding activities (Davis, 1991; Kennedy and Draper, 1991). Furthermore, pet birds from social species require considerable social interaction with their owners, and feather picking may function as an attention seeking behaviour (Stein, Woerper and Reed-Blake, 1999). Rosskopf et al., (1986) pose that continual maintenance of the behaviour may result from habit or auto-reinforcement of the behaviour as an anxiety alleviating activity. Bordnick et al., (1994) noted the potential importance of operant factors in reinforcing such behaviour, which although not associated with the onset of the condition, may result in an increase in the frequency and duration of feather picking. Johnson (1987) suggested that psychological disturbances expressed as feather picking and mutilation are symptoms of serotonin and norepinephrine imbalances. Furthermore an association between anxious temperaments and feather picking has been noted in a study of psittacine birds (Stein, et al., 1999). Birds demonstrated anxious

temperaments by screaming when separated from their owners, destructive behaviours, fear of novelty, recoiling postures and hissing vocalization (Johnson, 1987).

Similarities between trichotillomania and feather picking disorder have been described. Kennedy and Draper (1991) suggested that negative affective states such as depression in birds can lead to feather picking. Christenson, et al., (1993), in a study comprising 75 trichotillomanic patients found two major groupings of cues which evoked hair pulling, those associated with negative affective states (e.g. depression, anxiety and frustration) and those associated with sedentary activities. Many of the psychosocial stressors connected with trichotillomania in humans have been linked to feather picking disorder in birds and include stress, being alone, boredom, separation, anxiety, loss and frustration (Bordnick, et. al., 1994). Both trichotillomania (Swedo et al., 1989a) and feather picking disorder (Grindlinger and Ramsay, 1991) seem to respond to the serotonin re-uptake inhibitor Clomipramine.

Psychological intervention to treat feather picking disease involves either environmental manipulation or behavioural methods derived from operant theory. Providing the bird with a more stimulating environment, either through greater social contact with humans or other birds (thus reducing the time that the bird spends alone) and providing sufficient toys have all been recommended. A treatment based upon the operant principles of positive punishment involves spraying the bird=s feathers with a foul tasting substance to discourage plucking (Bordnick, 1994). A number of behavioural interventions involving a cross spectrum of approaches such as self-monitoring, habit reversal, negative practice, covert desensitization, engaging in

competing responses, recognising and avoiding cues that evoke plucking, and mild punishment, have been applied to treat trichotillomania (Azrin and Nunn, 1973). Physical or mechanical restraints, such as wearing gloves, cutting nails short or putting oil or petroleum jelly on the hair at night have been described as being useful against compulsive hair pulling in humans, while fitting a light weight plastic collar around the birds neck to prevent picking in birds, has been described (Bordnick, 1994).

CHAPTER THREE

THE CLASSIFICATION OF TRICHOTILLOMANIA

Trichotillomania might not be a unitary disorder. The complex phenomenological aspects of trichotillomania and questions where it should fall diagnostically are areas of continuing debate. These diagnostic dilemmas could be due to the heterogeneity of trichotillomania, the result of shared neurocircuitry, or pathophysiology between different but overlapping conditions that makes the behavioural separation of these conditions impractical at present (Ninan, Mansueto, Rothbaum, O'Sullivan and Nemeroff, 1999 ; Keuthan, et al., 1999).

3.1 Impulse control disorders

As described previously, the DSM-IV-TR (American Psychiatric Association, 2002) currently classifies trichotillomania as an impulse control disorder-not elsewhere classified, and defines it by the following criteria ;

DSM IV DIAGNOSTIC CRITERIA FOR TRICHOTILLOMANIA

- X Recurrent failure to resist impulses to pull out one's own hair, resulting in noticeable hair loss.
- X Increasing sense of tension immediately before pulling out the hair.
- X Gratification or a sense of relief when pulling out the hair.
- X No association with a pre-existing inflammation of the skin, and not a response to a delusion or a hallucination.

The impulse control disorders (ICDs) are broadly defined as mental disorders characterized by irresistible impulses to perform harmful or senseless behaviours (McElroy, Hudson, Pope, et al., 1992). The DSM-IV -TR (APA, 2002) defines the essential feature of an impulse control disorder as the failure to resist an impulse, drive, or temptation to perform some act that is harmful to the person or to others. It further specifies that for most ICDs, the individual feels an increasing sense of tension or arousal before committing the act and then experiences pleasure, gratification, or relief at the time of committing the act. Currently the ICDs are classified in the DSM-IV-TR (APA, 2002) as Impulse Control Disorders Not Elsewhere Classified and include intermittent explosive disorder, kleptomania, pathological gambling, pyromania and trichotillomania. An additional division, the ICDs Not Otherwise Specified (NOS) is included. ICDs presumably meeting criteria for ICD NOS are compulsive buying or shopping (also called buying mania or oniomania), repetitive self-mutilation, nonparaphilis sexual addictions (also called sexual compulsions), onychophagia (nail biting) and compulsive skin picking.

The International Classification of Diseases, 10th Revision (ICD-10), (WHO, 2004), classifies trichotillomania under the heading, *Disorders of adult personality and behaviour*, and groups it with habit and impulse disorders. The ICD-10 defines trichotillomania as : *"A disorder characterized by noticeable hair loss due to a recurrent failure to resist impulses to pull out hairs. The hair-pulling is usually preceded by mounting tension and is followed by a sense of relief or gratification. This diagnosis should not be made if there is a pre-existing inflammation of the skin, or if the hair-pulling is in response to a delusion or a hallucination"* (WHO, 2002, p.214).

3.2 Trichotillomania and OCD

The hair pulling of trichotillomania and the excessive hand-washing of OCD share some characteristics such as comparable neuropsychological deficits, related family histories, similar patterns of response to drug and behaviour therapy and a variety of clinical features with other grooming or habit disorders such as compulsive skin picking and pathological nail-biting (onychophagia), (Swedo, 1993). In each case the grooming is performed in an appropriate manner, but excessively. The patients from the various groups acknowledge the irrationality of such behaviour and try to resist performing the ritual. Although striking clinical differences such as the absence of obsessional thoughts and the monosymptomatic nature of hair pulling in trichotillomania exist, it is postulated that these disorders are related (Swedo, 1993). It is suggested that the threshold for repetitive grooming behaviours has been lowered by genetic susceptibility and further decreased in response to environmental changes

such as stress or pathogen-triggered autoimmune reactions, so that the behaviours are released frequently and in inappropriate situations in these patients (Swedo,1989).

3.2.1 Similarities and differences between trichotillomania and OCD

Most of the research investigating and describing trichotillomania have been comparative studies with OCD (Rettew, et al., 1991; Lenane, et al., 1992; Stanley, Swann, Bowers, Davis and Taylor, 1992; Stein and Hollander, 1992a; Stein, Hutt, Spitz and Hollander, 1993; Stein, Hollander, Simeon, Cohen, Islam and Aronowitz, 1994).

Stanley et al., (1992) state that, as far as behaviour is concerned, both trichotillomania and OCD are characterised by repetitive performance of a maladaptive motor behaviour over which patients perceive diminished control. However, hair pulling most often occurs when patients are alone, while obsessive-compulsive rituals are often performed secretively but sometimes in the presence of others. Hair pulling occurs most often when the patient is engaged in sedentary activities, while compulsions occur in a wide range of situations, often in association with obsessive ideation. Obsessions are a common feature in OCD while trichotillomania is usually not characterized by obsessive thoughts related to hair pulling. Trichotillomania does

not share the OCD characteristic of ordering, arranging or symmetry and is also not associated with motor compulsions such as tapping or touching. Both hair pulling and compulsions are experienced as decreasing anxiety. However, a variety of affective states such as anger, boredom and sadness are described as precipitants of hair pulling, while this is not the case in OCD. Hair pulling often produces feelings of pleasure, whereas compulsions do not. Sensory stimuli can precipitate and reinforce hair pulling but do not play a central role in OCD. It is further suggested that trichotillomania patients report receiving significantly more pleasure during performance of target behaviours than OCD patients (Stanley et al., 1992).

In terms of epidemiology, a higher incidence of trichotillomania exists in OCD families exists than in the general population. From work undertaken by Lenane, et al., (1992), it appears that there is a trend towards a higher rate of OCD in trichotillomania sufferers= families than trichotillomania in OCD offspring. Trichotillomania further differs from OCD in that it seems to appear predominantly in boys when the onset is early and mostly in females when the onset is later (Dawber, 1985; Ratner, 1989; Winchel, 1992). Generally the onset of trichotillomania is at an earlier age than that of OCD. Although both trichotillomania and OCD are associated with high rates of coexisting anxiety and affective disorders, patients suffering from trichotillomania score lower on measures of anxiety and depression than is the case in OCD (Stanley et al., 1992). Although, trichotillomania sufferers are less likely to exhibit associated pathology such as avoidance, pathological responsibility and obsessional slowness, they tend to be less indecisive than OCD patients, (Stanley, et al., 1992).

As far as neuroimaging is concerned, PET studies fail to provide evidence for a direct connection between these two groups (Swedo, et al., 1991). Although trichotillomania studies are still in the preliminary stages, suggestions for dysfunction in the parietal lobes (Swedo, et al., 1991) and the putamen (Grachev, 1997) are indicated for trichotillomania, whereas dysfunction in the caudate nucleus and orbito-frontal area are suggested in the case of OCD (Robinson, Wu and Nunne, 1995).

Rettew, et al., (1991), compared trichotillomania and OCD to normal controls in a neuropsychological study. The trichotillomania group made significantly more errors than did the control subjects on two of the subtests that measure visual-spatial memory, while the OCD patients only differed from the control group on one of the subtests.

Neurochemically, both OCD and trichotillomania symptoms are affected when challenged by serotonin agents such as the partial agonist *m*-chlorophenylpiperazine (m-CPP), (Hollander, DeCaria and Nitescu, 1992, Zohar, Mueller and Insel, 1987). However, according to Stein et al., (1999), preliminary evidence suggests that disorders characterised as obsessive and impulsive may demonstrate opposing behaviours and endocrine responses. In response to serotonin agonists such as m-CPP patients with compulsive disorders have shown increased negative affect, increased obsessional thoughts and compulsive urges (Buttinger, Hollander and Walsh, 1990). On the other hand patients with impulsive disorders (Hollander, Stein and DeCaria,

1994) as well as pathological gamblers (DeCaria, Stein and Cohen, 1993) have not demonstrated `a dysphoric response but showed a euphoric response to m-CPP.

As far as treatment is concerned, both trichotillomania and OCD seem to respond to serotonergic re-uptake blockers, even though the patterns of response may differ (Swedo, et al., 1989). Serotonergic treatment may lose effectiveness in chronic treatment of trichotillomania but not in OCD. Clomipramine has been demonstrated to be more effective for both OCD and trichotillomania than desipramine, which is a noradrenergic agent. However, fluoxetine is effective for OCD but not for trichotillomania. Lithium treatment seems to be beneficial in the case of trichotillomania, but not for OCD (Christenson, et al., 1991c). Behavioural techniques used in the treatment of OCD, which include emphasis on exposure and response prevention, are also reported to be helpful for trichotillomania (Vitulano, King, Scahill, and Cohen, 1992). However whereas the technique of habit reversal (which will be described in detail further on) is used in trichotillomania (Azrin and Nunn, 1972) the technique of Exposure Response Prevention (ERP) is used in OCD (Salvovski, 1989; Schwartz, Stroessel, Baxter, Martin, Phelps, 1996; Schwartz, 1998). ERP is a four-step programme specifically used in the treatment of OCD whereby the patients firstly becomes aware of the obsessive behaviour, secondly attribute this behaviour in their minds to the OCD and not to something that they chose to do, thirdly to learn to resist the compulsion and fourthly deal with the increasing anxiety due to resisting the compulsion.

3.3 Trichotillomania as part of the obsessive-compulsive spectrum of disorders

Recent observations indicate that trichotillomania may be best grouped with OCD in a spectrum of disorders having in common, pathological compulsions of excessive grooming (Swedo and Leonard, 1992).

While obsessive thinking and compulsive behaviour primarily characterize OCD, they may also be found in other disorders. As a result, obsessive-compulsive-related, or spectrum, disorders have been suggested (Hollander, 1993a; Hollander 1993b). The obsessive-compulsive spectrum disorders have emerged as a unique category of related disorders with important diagnostic, etiological and treatment implications (Hollander and Wong, 1995; Hollander, Kwon, Stein et al., 1996; Hollander 1998). The obsessive-compulsive spectrum of disorders can be divided in three main subgroups (Figure 3.1). The first subgroup has to do with >preoccupation with bodily appearance= and includes body dysmorphic disorder (BDD), hypochondriasis, anorexia nervosa and depersonalisation. The second subgroup, termed >impulse control disorders= includes trichotillomania, pathological gambling, kleptomania and compulsive shopping. The third subgroup, which frequently affects the basal ganglia, termed >neurological disorders= include autism, Sydenham=s chorea, torticollis and Tourette's syndrome, (Hollander and Wong, 1995).

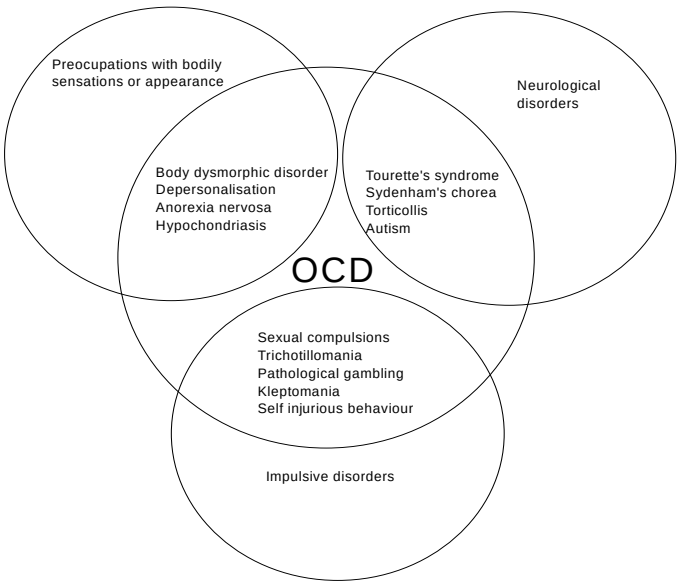


Figure 3 Obsessive-compulsive related or spectrum of disorders (Hollander, 1998)

Although psychiatry attempts to classify the obsessive-compulsive spectrum of disorders primarily into categories, dimensional aspects of various spectra may also be identified (Hollander et al., 1996). One dimension may be conceptually defined as estimation of risk which may include a risk aversive or >compulsive= endpoint where an estimation of the probability of future harm is apparent and a risk seeking or an >impulsive= endpoint, involving action without full consideration of the negative consequences of behaviour. Although compulsive and impulsive disorders can be viewed as lying at opposite ends of the dimension, they are similar in that they involve repetitive behaviour and a defect in the mechanism to inhibit or delay acting on these behaviours (Hollander, 1993a; Hollander 1993b; Hollander et al., 1996). However, the driving mechanism behind the behaviours differs. Compulsive individuals may be hyper vigilant and attempt to avoid harm and reduce anxiety or discomfort associated with the rituals. In contrast impulsive individuals are risk seekers who try to maximise pleasure, arousal or gratification and who may exhibit

antisocial behaviours (Hollander, 1998). It appears that men and woman behave differently with respect to impulsivity (Mc Elroy et al., 1995). Typical female behaviour includes stealing, hair pulling, self injurious behaviour, compulsive shopping and binge eating. Male impulsive behaviours include intermittent explosive disorder, gambling, setting fires and sexual impulsion (Hollander, 1998).

3.4 Overlap of OCD and obsessive-compulsive spectrum of disorders

The features of the obsessive-compulsive spectrum of disorders defined as sharing specific features with OCD (Hollander et al., 1996) include;

1 clinical symptoms - repetitive thoughts and/or behaviour

2 associated features - age of onset, clinical course, family history and co-morbidity

3 etiology - biological and neurological factors, such as serotonin and frontal lobe activity

4 response to treatment - selective efficacy of anti-obsessional treatments, including serotonin re-uptake inhibitors and behavioural therapy

Aetiologically there may be two specific mechanisms at work in the obsessive-compulsive spectrum of disorders. Firstly, both serotonin and frontal lobe activity are increased in those individuals displaying compulsive behaviour, whereas impulsive behaviours may be associated with decreased pre-synaptic serotonin and frontal activity. Secondly, there appears to be a preferential response to anti-obsessional

drugs, particularly SSRI=s and other agents which act by inhibiting the re-uptake of serotonin, for example clomipramine (Hollander and Wong, 1995).

3.5 Trichotillomania and Tourette syndrome

There is a paucity of data regarding the neurobiology of trichotillomania and it=s possible pathophysiological relationship with other obsessive-compulsive spectrum of disorders. However, comorbidity data as well as clinical, phenomenological and structural neuroimaging studies of trichotillomania, OCD and Tourette syndrome suggest that trichotillomania may be more closely related to Tourette syndrome than to OCD (Grachev, 1997). Certain neurological similarities have furthermore been noted between these conditions. Decreased volume of the putamen has been reported in females suffering from trichotillomania (O=Sullivan et al., 1997) and in a similar finding Peterson, Riddle, Cohen et al., (1993), using MRI demonstrated decreased left putamen volumes in subjects with Tourette syndrome.

According to Ninan et al., (1999) somatosensory sensations, parathesias and prodromal urges frequently precede hair pulling episodes and are associated with the urge to pull. Thus patients often focus on certain body sites for hair pulling and sometimes describe tingling or other sensations in these areas as driving the motor-pulling response. This is also characteristic of Tourette syndrome in which somatosensory urges seem to drive motor tics.

Sensorimotor phenomena frequently accompany tics in Tourette syndrome. These experiences include premonitory feelings or urges that are relieved with the performance of the act and a need to perform tics or compulsions until they are felt to be Ajust right≅ (Leckman, Walker, Goodman, Pauls and Cohen (1994). This Ajust right≅ feeling is also characteristic of trichotillomania (Christenson and Crow, 1996).

Furthermore Azrin and Nunn (1973; 1980) have demonstrated that habit reversal techniques are successful in the treatment of Tourette syndrome (1973) as well as in the treatment of trichotillomania (1980). In both instances treatment focused on using behaviour techniques such as awareness, learning of alternative responses, application of alternative responses, identification of specific stress factors and specific tension reduction techniques (Azrin and Nunn, 1980; Evers and Wettering, 1994) as will be discussed further on.

CHAPTER FOUR

THE NEUROBIOLOGICAL BASIS OF TRICHOTILLOMANIA

4.1 Neurochemistry

It has been postulated that in all animals across the phylogenetic level, a behavioural activation system as well as a behavioural inhibition system exists (Stein and Hollander, 1993). It has further been suggested that serotonergic neurons play a role in behavioural inhibition. Decrease in serotonergic transmission leads to an inability to adopt passive or waiting attitudes or to accept situations that need strong inhibitory actions (Soubrie, 1986). Pharmacotherapy studies have also demonstrated that medication acting on the serotonergic system has been effective in the treatment of impulsivity (Stein, Hollander and Liebowitz, 1993).

According to Hollander et al., (1993) the serotonergic functions in OCD may be correlated with neuroanatomical localized functional changes. The patients with compulsivity are not only characterized by serotonin hyperfunction but also by frontal lobe hyperfunction. Patients with impulsivity may be characterized by serotonin hypofunction and frontal hypofunction. Alternatively, Stein and Hollander (1993) suggest that both impulsivity and compulsivity may be conceptualized in terms of serotonin dysregulation or frontal dysinhibition, as both disorders respond to the same medication (serotonin reuptake blockers, SRI's) although in slightly different patterns. Compulsive disorders show a delayed but sustained response while impulsive disorders have a more rapid response but greater relapse over time. Furthermore

according to Stein et al., (1999), the monoamine neurotransmitters such as serotonin and dopamine, which are spread throughout the brain and which are affected by psychiatric medication, are known to play a role in the mediation of certain kinds of repetitive behaviours.

Serotonin has been hypothesized to be involved in trichotillomania. There is an association between the activation of serotonergic neurons and the execution of repetitive motor behaviours (Jacobs and Fornal, 1995), and more specifically, there is evidence of a link between serotonergic activity and grooming behaviours that might be analogous to hair pulling (Randall 1988, Traber 1988) as discussed previously. Pharmacotherapeutic dissection studies of unwanted repetitive behaviours have relied on the different neurotransmitter effects of structurally similar agents (Stein et al., 1999). For example, although clomipramine and desipramine are both tricyclic antidepressants, clomipramine is a serotonin reuptake inhibitor, but desipramine is primarily a norepinephrine reuptake inhibitor. In a cross-over study comparing the effects of clomipramine versus desipramine, Swedo, Rapoport et al., (1989) indicated that not only OCD, but also hair pulling and other symptoms such as nail biting, compulsive autistic behaviour and stereotypic movement disorder responded preferentially to clomipramine. Such findings seem to confirm the hypothesis that the serotonergic system plays an important role in mediating trichotillomania and other obsessive-compulsive spectrum disorders. On the other hand, there may also be significant differences between the neurochemistry of OCD and trichotillomania (Stein, Simeone and Cohen, 1995) such that extrapolation of the findings on OCD to trichotillomania may be questionable (Stein et al., 1999). For example, while

cerebrospinal fluid measures have suggested that some OCD patients have increased CSF 5-hydroxyindolacetic acid (5-HIAA) levels, (which is a metabolite of 5HT) and that these fall significantly during successful treatment with clomipramine (Thoren, Asberg and Bertilsson, 1980), a similar study by Ninan, Rothbaum and Stipetic (1992) did not find a difference in CSF (5-HIAA) levels between trichotillomania patients and controls. However, in trichotillomania patients, baseline CSF (5- HIAA) levels correlated significantly with the degree of response to serotonin reuptake inhibitors, a finding which suggests that both OCD and trichotillomania responses to selective serotonin reuptake inhibitors (SSRIs) are accompanied by a decrease in the levels of 5-HIAA (Stein et al., 1999). Furthermore meta-analysis has indicated that the serotonin reuptake inhibitors are effective in the treatment of OCD (Stein et al., 1995) and there is also evidence that OCD requires higher doses of these agents as well as a longer course of administration than do depression and other anxiety disorders (Stein et al., 1999). However, the efficacy of SSRI=s in trichotillomania remains questionable. Pollard et al., (1991) and Stein and Hollander (1992), have further indicated that the initial response to SSRI=s in trichotillomania may be lost over time. According to Stein et al., (1999), an interesting aspect of the meta-analytic studies of OCD is the finding that the less specific serotonin agents (e.g., clomipramine) may be more effective than highly specific drugs (e.g., Sertraline). This suggests that other neurotransmitter systems than the serotonin system may also play an important part in OCD.

It has been suggested that the dopamine neurotransmitter system is also involved in trichotillomania (Stein and Hollander 1992b). Extensive research has indicated

dopaminergic mediation of stereotypic movements, including grooming in animals (Cooper and Dourish, 1990), and dopaminergic agents have been found useful in the treatment of veterinary grooming disorders (Kenny, 1994). Similarly the opioid system may be involved in trichotillomania. A study undertaken by Mahr (1993) demonstrated significant improvement in controlling the hair pulling behaviour by using the opiate antagonist, naltroxene,

4.2 Neuroanatomy and neurophysiology

Although trichotillomania has been recognised for more than a century, not much is known of the underlying etiology or pathophysiology of the illness. As will be described further on, one study (Swedo et al, 1991) used PET to investigate trichotillomania. A few studies using SPECT to investigate trichotillomania (Vythilingum, Warwick, Kradenburg, Hugo, Van Heerden and Stein, 2002; Stein, Van Heerden, Hugo, Kradenburg, Warwick, Zungu-Dirwayi and Seedat, 2002) have been published. Further research using *f*MRI have been undertaken and will be described in more detail in the following text (Stein, Coetzer, Lee, Davids and Bouwer, 1997; Grachev, 1997; O'Sullivan et al, 1997; Rauch, Wright, Savage, Martis, McMullin, Wedig et al, 2007; Keuthen, Makris, Schlerf, Martis, Savage, McMullin, et al, 2007).

4.2.1 Positron emission tomography in trichotillomania (PET)

Swedo et al., (1991), firstly distinguished the neurophysiological properties of trichotillomania. In their study, ten women with trichotillomania were compared to 20 controls, using positron emission tomography (PET). The results from this study indicated that the mean global metabolic rate of the patients with trichotillomania was striking and significantly higher than that of the controls. Furthermore, no global metabolic rate of an individual patient with trichotillomania fell below the mean of the normal group. Individual analyses were performed for 46 brain regions. In all regions, the mean rate for glucose metabolic activity in the patients with trichotillomania was significantly higher than for the normal control group. The regional data were normalized by forming a ratio of regional glucose metabolism to global glucose metabolism. In most cases the ratio was not different between the patients with trichotillomania and the normal controls, despite higher regional absolute values in the patients with trichotillomania. However, three of the 46 ratios examined (right superior parietal, right cerebellum and left cerebellum) demonstrated differences between groups. In this study, Swedo et al., (1991) furthermore reported that the observed metabolic differences between the patients with trichotillomania and controls were not similar to the differences reported in a previous study between patients with childhood-onset OCD and controls. Direct comparisons between the scans of patients with trichotillomania and those with OCD did not show similar patterns of regional metabolic abnormalities. The patients with trichotillomania had a significantly elevated mean global metabolism, while the patients with OCD had a slightly, but not significantly, increased mean global metabolic rate as compared with controls. Furthermore, the patients with OCD, when compared with their controls, had significantly elevated metabolic ratios in the anterior cingulate gyri, caudate nuclei,

prefrontal and orbital frontal regions, while the patients with trichotillomania did not demonstrate this.

The researchers gave two explanations for possible cerebellar involvement. One implying that it was not the trichotillomania that is responsible for the cerebellar glucose metabolic activity but rather chronic anxiety experienced by these patients, (as was also found in a study by Reiman, Raichle, Robins, Butler, Herscovitz, Fox and Perlmutter, 1986). Secondly, (on the basis of research findings of Roland, Eriksson, Widen, Stone-Elander, 1989), they postulated that the cerebellar involvement could be explained by disturbances of tactile recognition or motor abnormalities, since the simple motor ritual of hair pulling is frequently accompanied by a tactile component, such as stroking the lips with the pulled hair, or pulling until 'just the right hair' is found.

4.2.2 Neurocircuitry and the basal ganglia in terms of the obsessive-compulsive spectrum of disorders

Various studies have indicated that the basal ganglia and the cortical striates that subserve it are involved in the obsessive-compulsive spectrum of disorders.

Early research (Evert and Thach, 1969; Allen and Tsukahara, 1974) suggested that the basal ganglia serves primarily to integrate diverse inputs from the entire cerebral cortex and to funnel these influences, via the ventrolateral thalamus, to the motor cortex. In particular, the basal ganglia were thought to provide a route whereby

influences from the cortical association areas might be transmitted to the motor cortex and thereby participate in the initiation and control of movement.

In a revised view, DeLong and Georgopoulous (1981) stressed the apparent maintained segregation of influences from the sensorimotor and association cortices through the basal ganglia-thalamocortical pathway. Alexander, DeLong and Strick (1986), sustained the concept of segregated basal ganglia-thalamocortical pathways and reinforced the general principle that basal ganglia influences are transmitted only to restricted portions of the frontal lobes - even though the striatum receives projections from nearly the entire neocortex, (Figure 4.1).

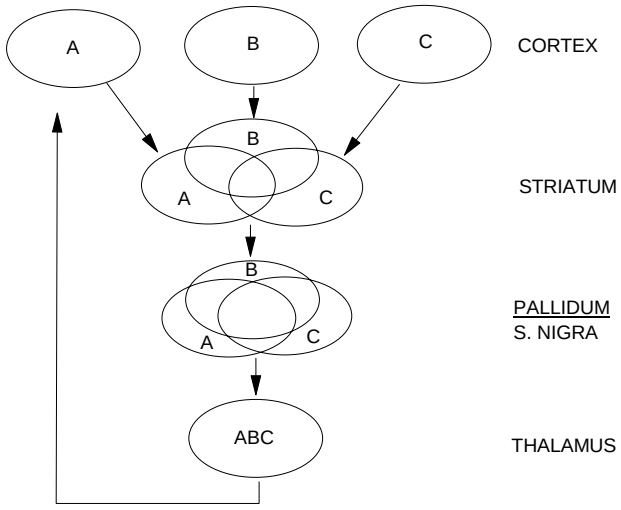


Figure 4.1 Generalized basal ganglia-thalamocortical circuit. Diagram of the proposed basal ganglia-thalamocortical circuits. Each circuit receives output from several functionally related cortical areas (A,B,C) that send partially overlapping projections to a restricted portion of the striatum. These striatal regions send further converging projections to the globus pallidus and substantia nigra, which in turn project to a specific region of the thalamus. Each thalamic region projects back to one of the cortical areas that feeds into the circuit, thereby completing the 'closed loop' portion of the circuit (Alexander et al., 1986, p 360).

Alexander et al., 1986 furthermore proposed five parallel circuits, (Figure 4.2). Each circuit receives multiple, partially overlapping cortico-striatal inputs, which are progressively integrated in their subsequent passage through the pallidum, and the substantia nigra to a restricted portion of the thalamus and from there back to a single cortical area. It appears that each basal ganglia-thalamocortical circuit receives its multiple cortico-striatal inputs only from cortical areas that are functionally related (and usually interconnected).

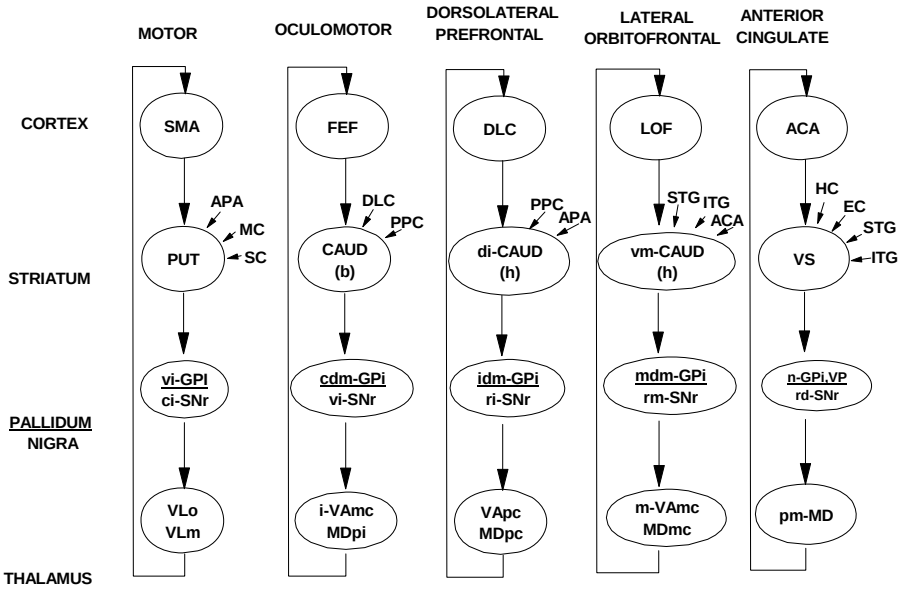


Figure 4.2 Five proposed basal ganglia-thalamocortical circuits. Parallel organization of the five basal ganglia-thalamocortical circuits. Each circuit engages specific regions of the cerebral cortex, striatum, pallidum, substantia nigra and thalamus.

Abbreviations : ACA: anterior cingulate area; APA: arcuate premotor area; CAUD: caudate, (b) body, (h) head; DLC: dorsolateral prefrontal cortex; EC: entorhinal cortex; FEF: frontal eye fields; GPi: internal segment of the globus pallidus; HC: hippocampul cortex; ITG: inferior temporal gyrus; LOF: lateral orbitofrontal cortex; MC: motor cortex; MDpl: medialis dorsalis pars paralamellaris; MDmc: medialis dorsalis parsmagnocellularis; MDpc: medialis dorsalis pars parvocellularis; PPC: posterior parietal cortex; PUT: putamen; SC: somatosensory cortex; SMA: supplementary motor area; SNR: substantia nigra pars reticulate; STG: superior temporal gyrus; VAmc: ventralis anterior pars magnocellularis; Vapc: ventralis anterior pars parvocellularis; VLm: ventralis lateralis pars medialis; VLo: ventralis lateralis pars oralis; VP: ventral pallidum; VS: ventral striatum; cl: caudolateral; cdm-: caudal dorsomedial; dl: dorsolateral; l: lateral; ldm-: lateral dorsomedial; m-:

> medial; mdm-: medial dorsomedial; pm-: posteromedial; rd-: rostrodorsal; rl:-rostrolateral; rm-: rostromedial; vm-: ventromedial; vl-: ventrolateral (Alexander et al., 1986, p 364)

The organization of these pathways with their close proximity to each other within the basal ganglia provides a model for understanding how pathology in this region might generate a range of symptoms including disorders of movement, affect and cognition. Outside the basal ganglia these pathways are not so closely aligned and lesions would be expected to produce more limited and discrete impairments of cognitive, limbic, or motor function. Disruptions occurring within the basal ganglia would be more likely to produce symptoms of all of these functions (Pantelis, Barnes and Nelson, 1992).

The hypothesis that Rapoport and Wise (1988) put forward concerning the pathways and processes underlying the basal ganglia-OCD relationship is based on a model of an innate release mechanism in the basal ganglia, namely a detection mechanism for recognizing specific aspects of stimuli (key or sign stimuli) and a release mechanism for the species specific behavioural response (known as a fixed action pattern). Usually detection of the key stimulus causes release (execution) of the appropriate behaviour. However two kinds of behaviour can occur in the absence of a key stimulus: vacuum behaviour (Ingle and Cruise, 1985) and displacement behaviour (Lorenz, 1981). Vacuum behaviour refers to a situation when a behaviour pattern is carried out while the normal releaser is absent, for instance, a bird may carry out the insect-catching movement even though there is no insect present (Brigandt, 2005). Displacement behaviour refers to the performance by an animal of an act inappropriate for the stimulus or stimuli that evoked it. This behaviour usually occurs when an animal is torn between two conflicting drives, such as fear and aggression. Displacement activities often consist of comfort movements, such as grooming,

scratching, drinking or eating (Encyclopaedia Britannica, 2006). According to the model that Rapoport and Wise (1988) have put forward, a hyperactive striatal circuit (or an input to it) might periodically produce an output of a vacuum behaviour or a displacement activity causing a movement pattern such as excessive grooming or an obsessive thought.

Following the work of Rapoport et al., (1988), Saxena, Brody, Schwartz and Baxter, (1998) proposed a model of imbalance of direct > indirect pathway tone in the orbitofrontal subcortical circuit. In the classical model proposed by Alexander, Crutcher and De Long (1990), frontal subcortical loop impulses along the direct pathway disinhibit the thalamus and activate the system in a self-perpetuating positive feedback loop, while activity along the indirect pathway provides negative feedback, inhibiting the thalamus. Thus the direct and indirect pathways appear to balance each other and allow for both facilitation and suppression of complex motor programmes via their opposite effects on the thalamo-cortical activations.

However, according to Saxena et al., (1998) OCD symptomatology may be the result of a >captured= signal in the direct orbitofrontal subcortical pathway or positive feedback loop. The captured signal may be due to an imbalance between direct and indirect pathway tone, with the direct tone having greater influence than the indirect pathway, with greater inhibition of the globus pallidus interna, leading to a greater thalamo-cortical activation. The excess tone in the direct relative to the indirect basal ganglia pathway may allow obsessive concerns and their attendant compulsive

behaviours to focus attention to themselves and result in an inability to shift to other behaviour.

Similarly March, Alexander, Packard, Zhu, Wingard, Quackenbush and Peterson (2004) demonstrated that striatal learning systems are uniquely dysfunctional in both children and adults suffering from Tourette syndrome. Neuroimaging studies have suggested that the pathophysiology of Tourette syndrome involves disturbances of the basal ganglia (Peterson, et al., 1993) and related corticostriatal-thalamocortical circuitry (Leckman, 2002). According to March et al., (2004), the parathesias associated with the phenomenon as well as defective striatal learning in Tourette syndrome may be symptoms of cortical-striatal dysregulation within the pathways from the sensorimotor cortex via the putamen.

Finally as far as neurotransmitters involved in the basal ganglia circuitry in normal brains are concerned, the major input to the basal ganglia, is a glutamatergic, excitory projection from the cerebral cortex to the striatum. Other inputs to the striatum include the dopaminergic input from the midbrain and the serotonergic input from the raphe in the brainstem (Rapoport and Wise 1988). Within the basal ganglia, inhibitory gamma-aminobutyric acid (GABA) neurons project from the striatum to the globus pallidus, and the major output of the basal ganglia is a GABA-ergic inhibitory projection from the globus pallidus to the thalamus, which in turn sends an excitory input to the cortex. In other words, a major pathway to, through, and out of the basal ganglia consists of a four-neuron loop (1) from cortex to striatum (excitory), (2) from striatum to globus pallidus (inhibitory), (3) from globus pallidus to thalamus (inhibitory), and (4) from the thalamus to the cortex (excitory) (Rapoport et al., 1988).

Furthermore a higher concentration of serotonin (5-HT) and serotonin receptors (5-HT2) in the basal ganglia of rat brains were found in studies undertaken by Pazos, Cortes and Palacios, (1985), Pazos and Palacios (1985), Steinbush (1981) and Stuart, Slater, Unwin and Crossman (1986).

4.2.3 Functional Magnetic Resonance Imaging in trichotillomania and related obsessive-compulsive spectrum disorders

Few MRI studies of trichotillomania, mostly postdating the start of the present study have been undertaken. Stein et al, (1997) used MRI to compare caudate volumes and ventricular-brain ratios between women with OCD (n=13), trichotillomania (n=17) and healthy controls (n=12). No significant differences were found between women with OCD, trichotillomania and normal controls.

Two MRI studies of trichotillomania have been published (Grachev, 1997 and O=Sullivan et al., 1997). From the descriptive data it appears that both of these studies used the same sample (n=10) and control (n=10) groups, and acquired the experimental data from the same instrument at the same time. However, Grachev (1997) focussed on describing reduced basal ganglia volumes in trichotillomania, while O=Sullivan et al. (1997) focussed on MRI-based morphometric topographic parcellation of human neocortex.

O=Sullivan et al. (1997) indicated that the trichotillomania group exhibited significantly decreased left putamen (13.2%) volumes contributing to significantly

smaller (9.7%) left lenticulate volumes as compared to normal controls. According to these authors, the origin of decreased putamen volumes in trichotillomania is unknown. However, referring to the work of both McDonald, Husain, Doraiswami, Figel, Boyko and Krishnan (1991) and O=Sullivan et al. (1997), the putamen volume is known to decrease with age, but this does not explain the observed group difference in comparison with age matched controls. O=Sullivan et al., (1997), postulate that the putamen findings may represent abnormalities intrinsic to the putamen. However it is also possible that these putamen differences represent a neurodevelopmental or structural adaptation to hypo- or hyper-function in other brain systems.

Grachev (1997) reported that the mean total cerebral cortex volume as measured on coronal images was not significantly different between trichotillomania patients and a normal control group. There were no significant volumetric differences of the precentral gyrus, postcentral gyrus, supplementary motor cortex, frontal opercular cortex or the opercular cortex between the two groups. However, a *post hoc* analysis demonstrated significant volumetric differences between the two groups in the left inferior frontal gyrus in the triangularis division as well as in the cuneal cortex. The trichotillomania group exhibited a significantly reduced triangularis volume of 27%, and enlarged right cuneal cortex volume of 40% compared to normal controls. Following these results Grachev (1997) proposed a hypothesis that enlargement of the right cuneal cortex in trichotillomania may reflect the complex interactions between the visual and sensorimotor cortices when the relay mechanism between them is not working in the normal pattern and significantly more neuronal stimuli are able to get to the targets generating the repetitive or compulsive behaviour seen in

trichotillomania. This process in the condition of reduced inhibition of non-rewarded responses in a go-no-go paradigm, is making the work of the relay mechanism much easier and possibly supports the finding regarding volumetric changes in the triangularis or inferior frontal areas.

The inferior frontal gyrus together with the tissues within and around the principle sulcus and on the dorsal prefrontal convexity (Broadmann=s area 9 and 10), provides the closed loop portion of the cortico-striatal input on the dorsolateral prefrontal circuit, and is consistent with the neuroanatomical models of OCD and related disorders. Furthermore, Grachev (1997) drawing on the work of Haber, Kuishio, Mizobuchi and Lynd-Balta (1995), states that recent electron microscope studies confirmed the strong links between the inferior pre-frontal cortex and nucleus accumbens, between the cuneal cortex and the inferior prefrontal cortex through the system of indirect parietal and temporal cortical pathways that is consistent with his study=s findings in trichotillomania.

According to Grachev (1997) single-cell recording and lesion studies suggest that neurons of the dorsolateral prefrontal cortex are selectively related to the retention of spatial or certain types of visual information. He continues that these cells may participate in the functional representation of external space in the motivational aspects of movement initiation, in the modulation of response inhibition in tasks that require a delay of motor responses and also those that require the inhibition of non-rewarded responses in a go-no-go paradigm, which could explain the formation of repetitive or compulsive systems in trichotillomania.

These findings of Grachev (1997) give empirical support for the inclusion of trichotillomania in the obsessive-compulsive spectrum of disorders on the basis of the structural brain retrocallosal abnormalities in patients with OCD. Based on these findings, the retrocallosal white matter volume is reduced by 14% in the right hemisphere and 10% in the left. Moreover, the dorsolateral prefrontal cortex, which includes the triangularis, and tissue within and around the principal sulcus and on the dorsal prefrontal convexity, provides the closed loop portion of the cortico-striatal input to the dorsolateral prefrontal circuit and is consistent with neuroanatomical models of OCD, Tourette syndrome and related disorders. Neuroimaging studies suggest that the pathophysiology of Tourette syndrome involves disturbances of the basal ganglia and related corticostriatal-thalamocortical circuitry (Leckman, 2002). Finding of smaller caudate volumes in children and adults with Tourette syndrome suggest that the motor and cognitive functions the striatum subserves may be dysfunctional in persons who have this condition (Peterson, Thomas, Kane, Scahill, Zhang and Bronen, 2003).

The search for refining the understanding of the neural circuits and its behavioural connection continues. Most recently Mataix-Cols, Wooderson, Lawrence, Mrammer, Speckens, and Phillips (2004) demonstrated that different OCD symptom dimensions are mediated by relatively distinct components of frontostriatal-thalamic circuits implicated in cognitive and emotional processing. According to these authors OCD may be best conceptualised as a spectrum of multiple overlapping syndromes rather that a unitary nosologic entity.

Rauch, et al, (2007) compared brain activation during implicit sequence learning in 10 females suffering from trichotillomania with healthy controls. They found no significant differences in implicit learning or in activation within the striatum, hippocampus or other brain regions. Their findings did not provide evidence for cortico-striatal dysfunction in trichotillomania.

Using *f*MRI Keuthen et al, (2007) demonstrated reduced cerebellar volumes in 14 females with trichotillomania as compared to an age, education and gender matched normal control group. Robust correlation between left primary sensorimotor cluster volumes and trichotillomania severity was noted. Given the significant motor involvement in trichotillomania symptomatology these results are not surprising. Individuals suffering from trichotillomania frequently exhibit idiosyncratic motor routines focused on the identification of hairs with specific textures or colours.

4.3 SPECT (Single beam Positron Emission Computerized Tomography) and Trichotillomania

Few SPECT studies have been undertaken to investigate trichotillomania. SPECT is a practical tool that is more readily available than PET, which is found more often at academic centres. SPECT offers advantages of decreased cost, the need for less intensively trained technicians, and the ability to carry out clinical testing in facilities lacking a cyclotron (Schukit, 1992). These factors raise the hope of more routine use of this imaging technique in clinical settings. A detailed description of SPECT is provided in chapter 9.

Using SPECT Stein et al, (2002) compared functional brain imagery and pharmacotherapy in trichotillomania before and after treatment with the selective serotonin reuptake inhibitor Citalopram. Pharmacotherapy led to significantly reduced activity in inferior-posterior and other frontal regions. The authors concluded that their data are to some extent consistent with work suggesting that trichotillomania, like OCD, is marked by corticostriatal circuits.

A further SPECT study of a set of twins suggested that more severe trichotillomania symptoms are associated with more extensive and pronounced perfusion abnormalities (Vythilingun et al, 2002).. Their findings were consistent with the neuroimagery studies as described by Swedo et al, (1991), Grachev (1997) and O'Sullivan et al, (1997).

CHAPTER FIVE

THE NEUROPSYCHOLOGY OF TRICHOTILLOMANIA

5.1 Trichotillomania

Neuropsychological assessment allows a more detailed determination of possible impairments in brain function (Stein, et al., 1999). Whereas neuropsychological impairments in OCD are increasingly being understood in terms of abnormalities in

structural and functional brain imaging (Stein et al.,1999), similar comparisons in TTM have not yet been determined and is possibly an area for future research. This chapter is included in the present study to provide an overview of the neuropsychological research that has been published to date.

Unfortunately, relatively few neuropsychological studies in trichotillomania patients have been reported. As far as can be determined, only three studies have been undertaken to examine the neuropsychology of trichotillomania. Rettew et al., (1991) administered two neuropsychological tests, the Money Road Map test and the Stylus Maze test, to 21patients suffering from trichotillomania, and the results were compared with those of age- and sex-matched groups with OCD (n=12), other anxiety disorders (n=17) and normal controls (n=16). The Stylus Maze, a test of spatial memory, has been reported to be particularly sensitive to patients with frontal, right temporal, and right parieto-temporo-occipital lesions (Milner, 1965). This test consisted of a 10 X 10 grid of circular metal contacts. The patients were asked to trace an invisible pathway from the lower right corner to the upper left corner of the grid with a metal tipped probe, moving from one contact to another and touching the contacts with the probe. A low tone signalled the correct path while a high tone indicated an error. When an error occurred, subjects were to go back to their last correct move and try another direction. The Road Map test (Money, Alexander and Walker, 1965) requires an ability to mentally rotate one=s position in space while maintaining directional sense. This test has been shown to discriminate between patients with right parietal and left frontal lesions and those with lesions in other brain areas (Butters et al., 1972). For this task, the patient was seated in front of a piece of

paper on which a pathway was drawn through a simulated road map and was told to imagine walking along this pathway. While keeping head and body straight, the patient was instructed to say whether to turn left or right at each corner. The first half of the pathway proceeds in a direction away from the patient=s body position, before turning and moving towards the subject.

Whereas no differences were found between any of the groups on the Money Road Map test, significant group differences on the Stylus Maze suggest that trichotillomania patients may have problems with spatial processing. An association between performance on the Stylus Maze test with trichotillomania severity and outcome also supports the hypothesis that neuropsychological abnormalities might be a manifestation of the underlying disorder. Limitations of this preliminary study include the small sample size and the limited number of neuropsychological tests employed.

A further study compared neuropsychological functioning in 20 subjects suffering from trichotillomania with 20 controls. Keuthen et al., (1996) investigated executive functioning by making use of the Odd Man Out test (Flowers and Roberson, 1985) and the Visual Verbal test (Feldman and Drasgow, 1951). Visual spatial functioning was assessed by the copy part of the Complex Figure of Rey (Osterrieth and Rey, 1944). The Mental Rotation test (Vandenburg and Kuse, 1978) was used to determine spatial rotational ability. Tests for attention and immediate memory included the Digit Span subtest from the Wechsler Adult Intelligent Scale -Revised (Wechsler, 1981) and the spatial and verbal span conditions of the Delayed Recognition Span test

(Moss, Alberts, Butters and Payne, 1986). Non-verbal memory was measured by the immediate and delayed recall conditions of the Complex Figure of Rey Test. No group difference was found to exist on any of the verbal memory or attention tests. However, significant differences at the .05 alpha level were found on the Odd Man Out Test and the immediate recall part of the Complex Figure of Rey Test. Executive dysfunction was found in hair pullers on the Odd Man Out Test for the stimuli of shapes but not letters. The researchers speculated that the finding might reflect greater difficulty in maintaining a mental set when dealing with shapes, which are less likely to be subject to verbal mediation strategies.

An extensive neuropsychological study of the cognitive functioning in trichotillomania was undertaken by Stanley et al., (1997). These researchers compared 21 trichotillomanic patients to 17 normal controls. They administered a broad neuropsychological battery which included assessment of cognitive ability using the WAIS-R. Auditory perception and language was measured by the Auditory Discrimination test (Wepman, 1973), the Token Test (Benton and Hamsher, 1976), the Controlled Oral Word Association Test (Benton and Hamsher, 1976) and the Boston Naming Test (Kaplan, Goodglass and Weintraub, 1983). Visual-perceptual, visual-spatial and visual-constructive abilities were assessed by the Visual Form Discrimination Test (Benton, Hamsher, Varney and Spreen, 1983), the Facial Recognition Test (Benton et al, 1983), the Judgement of Line Orientation Test (Benton et al., 1983) and the Complex Figure of Rey (Osterrieth and Rey, 1944). Somatosensory function was assessed by the Right-Left Discrimination Test and the Finger Localization Test (Benton et al., 1983) and the Tactual Performance Test

(Halstead, 1947). For motor ability the Finger Tapping Test (Reitan and Davidson, 1974) and the Grooved Pegboard (Matthews and Klove, 1964) were used. The Complex Figure of Rey, the Benton Visual Retention Test (Benton, 1974) and the Californian Verbal Learning Test (Delis, Kramer, Kaplan and Ober, 1983) were administered to assess memory. For concept formation the researchers used the Booklet Category Test (DeFillipis and McCampbell, 1979) and the Wisconsin Card Sorting Test (Heaton, 1981). For attention, search, tracking and information processing speed, the Visual Search and Attention Test (Trenerry, Crosson, DeBoe and Leber, 1990), the Trail Making Test (Reitan, 1959), the Paced Auditory Serial Addition Test (Gronwall and Sampson, 1974) and the Stroop Test (Trennery et al., 1990), were administered. The researchers furthermore compared result from the neuropsychological assessment with the Beck Depression Inventory (Beck, 1978) and the Spielberger State-Trait Anxiety Inventory (Spielberger, Gorsuch and Lushene, 1970).

According to Stanley et al., (1997b), consistent differences between the trichotillomanic group and the control group appeared on performance measures of divided attention. They also found a correlation between affective state (anxiety / depression) and measures of divided attention. They furthermore describe a significant relationship between affective state and performance on speeded motor tasks. According to these authors, these findings may have implications for the conceptualisation of trichotillomania, providing support for the notion that trichotillomania may be best understood as an anxiety- or affective based disorder.

This study failed to find neuropsychological deficits in visual spatial ability, motor function, or executive dysfunction.

Visual spatial dysfunctioning in hair pullers has also been described by Stein et al., (1994) in a study investigating neurological soft signs in female trichotillomania patients, OCD patients and healthy controls. Visual spatial functioning was assessed with the face-hand test, evaluation of right left confusion on self and the examiner, and the drawing of a cube.

5.2 Differences between trichotillomania and OCD

Investigations comparing the neuropsychology of trichotillomania and OCD are limited and provide inconsistent findings regarding potential overlap between the disorders.

A study including 11 patients suffering from trichotillomania, 17 patients with OCD compared to 16 age and education matched healthy controls, failed to demonstrate any differences when a battery of tests said to be sensitive to basal ganglia damage, such as seen in Huntington=s Disease, was undertaken by Martin, Pigot, Lalonde, Dalton, Dubert and Murphy, (1993). The tests that were used included the WAIS-R Vocabulary and Block Design subtests, the Money Road Map Test, the Verbal Fluency Test, Simple and Choice Visual Reaction Time, a Visual Search Task, the California Verbal Learning Test and the Room Test. The authors interpreted these findings as reflecting neuropathological differences between Huntington=s disease,

OCD and trichotillomania, despite the suggestion of common basal ganglia involvement.

In the study by Rettew et al., (1991), described above, no significant differences were seen between the trichotillomania and OCD groups, a finding that provides some support for a potential neurobiological overlap between the disorders. However, differences between trichotillomania, OCD and the two control groups did not occur consistently, and limitations resulting from small sample sizes and a limited number of neuropsychological tests need to be considered when interpreting the data.

Similarly, as described previously, Keuthen et al., (1996) found group differences in non-verbal memory and executive functioning, two of the three cognitive domains in which OCD patients have been shown to be impaired relative to control groups. However, the authors state that interpretation of these test results should be handled cautiously, given the specific tests used to assess cognitive functions and the multiple number of t- tests performed. In contrast to this, Stanley et al., (1997b), in their findings, failed to support the suggestion that trichotillomania is a variant of OCD, given failure to document neuropsychological deficits in visuospatial ability, motor function or executive function.

Additional research is required in order to delineate the neuropsychology of trichotillomania. As was mentioned earlier in this chapter, neuropsychological impairments in OCD are increasingly being understood in terms of abnormalities in structural and functional brain imaging (Stein et al., 1999). The same kinds of relationships still need to be ascertained whether they exist for trichotillomania

CHAPTER SIX

THE TREATMENT OF TRICHOTILLOMANIA

The existing treatment literature raises serious concerns regarding effective treatment for trichotillomania. Treatment studies are plagued with conflicting results, a lack of large scale controlled treatment trials and limited long-term follow up of patients. For the most part, there is a predominance of case studies and case series, as opposed to more methodological rigorously controlled group designs (Keuthen. et. al, 1998). At the beginning of the present study, psychometric properties of the assessment instruments have not been adequately studied, with only one scale (The Psychiatric Institute Trichotillomania Scale – PITS) having known reliability and validity (O=Sullivan, Keuthen, Hayday, Ricacardi, Buttolp and Jenike, 1995). Subsequently Keuthen, O'Sullivan, Ricciardi, Shera, Savage, Borgmaa et al, (1995) have established the Massachusetts General Hospital Hairpulling Scale (MGHHS). This is a seven-item self-report measure that assesses urges to pull hair, time spent pulling, perceived control, and distress associated with pulling. All items are scored using a 0 to 4 Likert scale, and address hairpulling behaviour during the preceding week. Initial studies on the psychometric properties of the MGHHS have shown high internal consistency and moderate convergent validity with other self-report measures of hairpulling. The MGHHS also shows divergent validity with measures of depression

and anxiety, and appears sensitive to changes to level of symptom severity. It also demonstrates excellent test-retest reliability (Hajcak, Franklin, Simons and Keuthen, 2006). Thus while existing empirical data support the efficacy of behavioural and pharmacological approaches, they are insufficient for the development of treatment paradigms.

6.1 Pharmacological treatment

Numerous reports of beneficial pharmacological treatments for trichotillomania exist. However, most are single case studies and reviews of short-term drug trials. There is a lack of randomized, controlled drug studies and some controlled outcome data indicates conflicting results (O=Sullivan, Keuthen, Rodriquiz, Pigott, L'Heuex, Grady et. al., 1998).

Although a variety of medications have been used in the treatment of trichotillomania, the most effective intervention seems to be treatment with antidepressants that specifically inhibit serotonin re-uptake, such as clomipramine (Swedo et al., 1989; Pollard, Ibe, Krojanker, Kitchen, Bronson and Flynn, 1991) and imipramine (Weller, Weller and Carr, 1989).

A study by Swedo et al. (1989) suggested that in trichotillomania the serotonin re-uptake blocker clomipramine was more effective than the norepinephrine re-uptake blocker desipramine in reducing hair pulling, a finding similar to that obtained earlier in OCD.

Subsequent controlled drug trials focused on the anti-obsessional serotonergic antidepressants given the prevailing conceptual view of trichotillomania as a variant of OCD. A controlled comparison of clomipramine, fluoxetine and placebo, indicated the superiority of both active drugs over placebo with no differences between drugs (Pigott, L'Heuex, Grady, Bernstein, Dubert, Rubenstein et al., 1992). Augmentation of serotonergic agents with dopamine blockers such as pimozide also plays a beneficial role in the treatment of trichotillomania (Stein and Hollander, 1992b). Opposing views regarding the efficacy of fluoxetine treatment exists, and no conclusive studies have yet been reported (Swedo, 1993). Two placebo-controlled double-blind trials of fluoxetine have failed to document a superior response as compared to placebo (Christenson, Mackenzie, Mitchell and Callies, 1991b; Streichenwein and Thornby, 1995). However, a retrospective review of trichotillomania patients treated with clomipramine, fluoxetine or fluvoxamine indicated that all treatment responders relapsed after initial improvement (Iancu, Weitzman, Kindler, Sasson and Zohar, 1996). It is further reported that mood stabilizers such as lithium carbonate, reduces hair pulling activities (Christenson, et al., 1991c).

The opiate antagonist, naltroxene, has been the only non-antidepressant medication to undergo a controlled study (Mahr, 1993). A significant improvement in hair pulling symptomatology was demonstrated in a 6 weeks, placebo-controlled, double-blind, and parallel treatment design.

Finally, topical ointments including antibiotics, steroids and analgesics have been studied in the light of the infection and inflammation that some hair pullers experience, as well as the sensory itch that is described by some patients. Topical flucinolone (0.01%), in conjunction with clomipramine, has yielded positive results (Black and Blum, 1992 ; Gupta and Freimer, 1993). Capsaicin, has also been used successfully in conjunction with behaviour therapy (Ristvedt and Christensen, 1996).

6.2 Hypnosis

Few formal studies of the use of hypnosis for trichotillomania have been published (Stein, et al., 1999; Winchel, 1992; Rowen, 1981). A review of the literature on the use of hypnosis in the treatment of trichotillomania, which included a computer search, uncovered only 14 published reports involving 30 cases (Stein et al., 1999). Most of these reports describe single case histories. Two cases were discussed by Fabri and Dye (1974). They described the use of suggestions of relaxation, tranquillity and diminution of anxiety and they reinforced the patients' sense of control over their behaviour. Gardner (1978) describes one case where a girl of 8 years old responded to suggestions of control. Rowen, (1981), used age regression on a 21 year old male patient. At a regressed state a suggestion was made that he would feel a slight discomfort when the hair was pulled, and he 'would feel a slight ping=. For each successive year this >ping= was to increase in intensity so that by the age of 21 years each pull would be distinctly uncomfortable, but not necessary painful. At a six months follow up session the patient reported continued complete freedom from the hair pulling.

In most published reports of the use of hypnosis in the treatment of trichotillomania, theoretical orientations or clinical preference seems to determine methods of trans induction, symptom targets, and specific hypnotic suggestions (Stein et al., 1999). For example psychodynamic hypnotherapy may include age regression to uncover emotional conflicts presumably to the onset of trichotillomania (Hynes, 1982), whereas hypno-behavioural treatment typically uses relaxation to relieve tension along with suggestions for behavioural change (Galski, 1981). The general conclusion from these reports is that a variety of hypnotic suggestions can be effective, but hypnosis is most likely to succeed with patients with a high hypnotic susceptibility (Minichiello et al., 1994).

6.3 Psychoanalytic and psychodynamic viewpoint

Clinical literature does not provide substantial evidence for the efficacy of psychoanalytic or psychodynamic approaches in reducing the symptoms of trichotillomania. A lack of success and high remission rates have been reported (Phillipopoulos, 1961; Monroe et al., 1963; Mannino et al., 1969; Keuthen, Stein and Christensen, 2001). In some cases the process of involving introspection may accelerate the self-destructive symptoms (Cordle and Long, 1980). Keuthen et al., (2001) reported that some hair pullers have noted that psychodynamic therapy has resulted in harmful outcomes when they were interpreted to have sexual conflicts or labelled as masochistic because they extracted their hair. Furthermore, according to

Keuthen at al., (2001) for many or even most people suffering from trichotillomania it is quite plausible that hair pulling has no symbolic meaning at all.

Traditionally the psychoanalytic literature ascribes various symbolic meanings to trichotillomania, emphasizing its pathological nature, and suggested that its aetiology was a function of disrupted psychosexual development (Greenberg, 1969). As early as 1936, Berg (1936) described hair pulling in terms of a sexual conflict. According to Barahal (1940), who described the symbolic importance of hair in myth and customs (i.e. symbol of strength, beauty, mourning and castration) saw hair pulling as a sexual maladjustment. Zaiden (1951) as cited in Galski (1983) differentiated between the pulling out of scalp hair from that of pulling eyebrows, eyelashes or pubic hair. He considered the former as a more serious infliction, as an attempt to escape from an unbearable sexual situation (e.g. marriage), and the latter as a mild neurotic symptom used to release unsatisfied sexual tension (a masturbatory substitute). Sperling (1954) described hair is a symbol for unconscious bi-sexual conflicts. His analysis revealed that certain features specific to the hair, which is part of the body in both sexes, and grows back spontaneously when removed, made the hair especially suitable for the dramatization of this conflict. Sperling (1954) is of the opinion that unless the bi-sexual conflict is resolved, these patients continue to act out their conflicting feelings through varying emphasis on the male-female symbolic meaning of the hair. It was found that the cutting off or losing of the hair is regarded as an expression of castration, the giving up of the feminine part.

Greenberg et al. (1965), agrees with Monroe and Abse (1963) that the psychopathology of trichotillomania is multi determined. Multi fixation points at all levels of psychosexual development are indicated. Pulling, saving or eating the hair, symbolise an oral regression; the incorporation of, and identification with, the mother, as well as the reassurance against her loss. At the anal stage of the child=s development, hair plucking expresses rage and frustration directed towards the object and the internalized superego. According to Monroe et al. (1963) hair pulling resembles other masochistic practices in that the patients attempts to administer a controlled amount of self-punishment to avoid the uncontrollable wrath of a paranoid-sadistic parent - or representation of the parent within the psychic structure. Greenberg et al.(1965) describe that at the genital stage, hair pulling in the female demonstrates to the mother that the daughter will deny femininity and give up the oedipal struggle. These authors state; *A... the symptom also identifies the daughter with the father as to say : >If I become a man by cutting my hair, then mother will take me for her lover=*≅. In the male the symptom expresses the child=s denial of incestuous impulses through auto-castration but may still indicate his identification with a balding father as mother=s bed partner. For both sexes, hairlessness may emphasize the innocent childishness of the patient who has renounced all claims to genital sexuality. According to Greenberg et al. (1965), another important factor in the genesis in trichotillomania is the adolescent process itself, with it=s changes in body image, upsurge of sexual energy, and increasing striving for personal autonomy and self-realization. These authors conceptualise trichotillomania in adolescents as an ongoing compromise between the patient=s drives for self assertion and mastery, and the parental needs to maintain an unhealthy status quo.

The psychodynamics of trichotillomania have been related primarily to emotional deprivation or frustration of the child=s need by the mother (Mannino et al., 1969). The former authors conclude that the mother cannot develop a healthy symbiosis with her offspring because of her own unresolved emotional conflicts and intense unsatisfied dependency needs. They use the term >healthy symbiosis= to describe the mutual dependency between mother and child that occurs in normal development, and >unhealthy symbiosis= where the mother seems to need the child as much if not more than the child needs the mother. The mother loses perspective about the child=s growth and development and depersonalises the child by rewarding clinging, dependent behaviour and punishing directly or indirectly any natural attempt of the child to become less reliant on her.

Galski (1983) believes that children who become trichotillomanic around prepubescence have earlier experienced protracted doting, overprotection and intrusion into their lives with parents with their own symbiotic-parasitic needs. As a result the children suffer psychological damage which seriously disrupts the normal development of the ego, producing non-specific ego weakness and causing failure to achieve object-constancy. Galski (1983) surmises that without the establishment of object constancy an individual requires visible evidence that the object/person capable of gratifying basic security needs is present or available. In trichotillomania the hair seems to symbolize the need gratifying object or person who is lost when the hair is pulled out, and more importantly, regained when it is eaten. It is the latter component of trichotillomania, i.e., reincorporation of the need gratifying object/person, which

reassures the patient that infantile needs can be gratified and security can be re-established. Unfortunately, the trichotillomanic patient is driven to repeatedly and compulsively remove hair so that it can be regained temporarily since object constancy is never really established.

6.4 Behavioural Therapy

In contrast to the psychoanalytic literature, the behavioural literature has been less provocative about the aetiology, less dramatic about the pathology of the symptoms, and less pessimistic about the prognosis (Friman et al., 1984).

Several behavioural therapy techniques have been described. Most of these studies are single case studies, and mostly a combination of behavioural techniques has been used. Techniques such as thought stopping (Taylor, 1963), self-monitoring, including saving the pulled hairs and bringing them to the therapist weekly (Bayer, 1972), response costing (Bayer, 1972), relaxation techniques (Bornstein and Rychard, 1978), operant self-control (Cordle et al, 1980), awareness training (Miltenberger and Fuqua, 1985) and rational-emotive therapy followed by self instructional training (Bernard, Kratochwill and Keefauver, 1983) have been employed in the treatment of trichotillomania.

Another behavioural technique that has been shown to have positive results, is that of covert desensitization. This is a self management strategy wherein patients, with a therapist's guidance, punish their own maladaptive behaviours by imagining highly

stressful events when either the behaviour or the urges to pull occur (Cautela, 1967). Levine (1976) was the first to describe the treatment of trichotillomania applying covert desensitization in an adult, who for 5 years had unsuccessfully been in psychoanalysis for the problem behaviour. One week after the introduction of covert desensitization, the patient=s hair pulling dropped to zero which was maintained at follow-up.

The method of habit reversal, developed by Azrin et al., (1973) seems to have the highest significant success rate (Swedo, 1993). This technique, as most of the previously mentioned techniques is not one specific technique, but rather a combination of techniques. The technique involves teaching the subject to employ competing reactions after increasing awareness and identifying response precursors as well as using relaxation training, habit interruption and prevention training, as described in Table 6.1.

Table 6.1 Habit Reversal Training

Identifying response precursors *Since hair pulling does not occur in isolation from other actions , the patients is assisted in identifying and learning which actions are precursors to hair pulling (e.g. straightening the hair, touching the face or head, standing in front of a mirror, watching TV, reading or studying, driving or talking on the telephone.*

Competing reaction training*. Each patient is taught a specific response pattern that would be incompatible with the hair pulling, and would, therefore, prevent the habit from being continually intertwined in normal activities. Is addition, the incompatible movement was designed to have the characteristics of (i) being opposite to the hair pulling movement, (ii) capable of being maintained for several minutes, (iii) producing heightened awareness by an isometric testing of the muscles involved in the movement and (iv) being socially inconspicuous and easily compatible with normal ongoing activities but, still incompatible with the hair pulling. Patients were instructed to engage in the competing responses for about three minutes following either the temptation to pull or actual pulling.*

Relaxation training*. Relaxation is a self control procedure for patients for whom anxiety or stress is a major trigger of their pulling. Patients are taught progressive deep muscle relaxation Jacobson (1929) and autogenic training (Luthe, 1969) as well as breathing control (Luthe, 1969; Davis, Robbins-Eschelman,and Mackay, 1995).*

In their study, Azrin, Nunn and Franz (1980) compared 19 trichotillomanic patients treated with the habit reversal technique to 15 patients treated by utilizing negative practice. For the negative practice persons the maximum reduction of hair pulling was 69% during the third week after training and the habit returned gradually so that at the three month follow-up it was reduced by only 50%. The habit reversal method decreased the hair pulling in their study by 99% on the first day after treatment and this general degree of reduction prevailed. After 4 months, hair pulling was reduced by 91% and at the 22 month follow-up by 87%.

While behavioural treatment approaches offer considerable promise in the treatment of trichotillomania, their limited empirical study significantly restricts knowledge of which techniques, in what format, are of benefit for which hair pullers (Keuthen, et. al., 1998).

6.4.1 Behaviour Therapy and the obsessive-compulsive spectrum of disorders

Numerous research demonstrating comparable effectiveness of behaviour therapy and pharmacological intervention in the treatment of general psychiatric illnesses such as unipolar major depression (Klerman, 1990), panic and agoraphobic disorders (Mavissakalian and Michelson, 1986), OCD (Baxter, Schwartz, Bergman, Szuba, Guze. Mazziotta et al., 1992) and Tourette syndrome (Evers et al., 1994) have been published.

Exposure Response Prevention (ERP) is a behavioural treatment specific to OCD whereby patients learn to perform adaptive behaviours instead of pathological ones in response to the intrusive thoughts and urges which comprise the core symptoms of OCD (Schwartz, 1998). To be successful in therapy, patients must be able to tolerate the acutely uncomfortable feelings that arise as a result of not performing the neutralising behaviour. Research demonstrating the effectiveness of this treatment method and the subsequent correlating changes in cerebral glucose metabolic rates has been published (Baxter et al., 1992, Schwartz, et al., 1996) and will be discussed in chapter 10.

6.5 Cognitive Behaviour Therapy

Similar to the other treatment methods, few controlled studies have been documented in the scientific literature as to the efficacy of cognitive behaviour therapy in the treatment of trichotillomania. Again, mostly single case studies are described.

Furthermore, cognitive techniques have been utilized mostly in combination with other behavioural techniques (Keuthen et al., 1998). Techniques such as thought stopping (Taylor, 1963), positive imagery (McLaughlin and Nay, 1975), cognitive desensitization to anxiety-provoking triggers (Bornstein et al., 1978), rational emotive therapy and self-instructional training (Bernard et al., 1983) have been described as having positive outcomes.

Ottens (1981; 1982), proposed a cognitive-behaviour treatment programme including rational restructuring, self-talk, self instruction, covert assertion and anticipatory strategies for controlling pulling behaviour. Similar programmes were proposed by Rothbaum (1990) and Gluhoski (1995). Rothbaum (1990) proposed a comprehensive treatment package including habit reversal training, muscle relaxation and breathing control, thought-stopping, cognitive restructuring, guided self-dialogue, role play and relapse prevention. The programme spans nine sessions as described in table 6.2.

Table 6.2 Cognitive Behaviour Therapy model for trichotillomania

Session 1: **Session 2:** **Session 3:**	Information gathering, This includes, response description, response detection (awareness training), identifying response precursors (early warning), identifying habit prone situations, and self monitoring.
Session 4:	Habit reversal training (as described in previous table)
Session 5:	Deep muscle relaxation training
Session 6:	Differential relaxation plus breathing retraining
Session 7:	Thought stopping
Session 8:	Cognitive restructuring
Session 9:	Guided self dialogue Role playing and covert modelling Relapse prevention

A study by Rothbaum and Ninan, (1992), compared the efficacy of cognitive behaviour therapy with pharmacotherapy (clomipramine) and placebo control in 14 patients. The patients were randomised across the three study conditions for a 9 week period. After the trial with cognitive behaviour therapy 71% of the patients (n=5) in this group were categorized as responders. Of the group randomized to the clomipramine treatment, 67% (n=4) of the completers were categorized as responders. None of the patients randomized to the placebo group showed an improvement. While both the drug and the cognitive-behaviour therapy groups were superior to placebo at the end of the treatment time, the patients in the cognitive-behaviour therapy group maintained gains at a 3 month follow-up, while those in the clomipramine group tended to relapse. However, the small sample size was seen as a problem. Rothbaum et al., (1999) concluded that cognitive behaviour therapy with some component of habit reversal and clomipramine are the only treatments reported to be effective in

controlled studies of trichotillomania. A detailed description of Cognitive Behaviour Therapy will be discussed in Chapter 8.

6.5.1 Cognitive Behaviour Therapy and the treatment of the obsessive- compulsive spectrum of disorders

Various studies have indicated that both pharmacological and cognitive behaviour treatment are similarly effective, in the treatment of unipolar major depression (Elkin, Shea, Watkins, Imber, Sotsky, et al., 1989), as well as panic and agoraphobia (Marks, Gray, Cohen, Hill, Mawson, Ramm and Stern, 1983). Others, using PET have demonstrated similar patterns in mood disorders (Baxter, 1991), anxiety disorders (Reiman, Raichel, Butler, Herscovitch and Robins, 1984; Reiman, Raichle, Robins, Minton, Fusselman, et al., 1989), and OCD (Baxter et al., 1992).

The possibility of both SRI's and cognitive behaviour modification treatments having the same neural affect on an organism could be possible. The research undertaken by Montarolo, Goelet, Castellucci, Morgan, Kandel and Schacher (1986) and Kandel (1989) demonstrated that in lower life forms such as the sea slug *Alypsia,* it is changes at the synaptic level that use serotonin that seem to mediate learned changes in stimulus response behaviour. Direct applications of serotonin at these synapses can produce the same lasting changes in synaptic function and behaviour as seen with behaviour modification in *Alypsia*.

As was discussed previously in chapter 4, Schwartz (1998) describes the neuroanatomical aspects of cognitive behavioural therapy response in OCD in the context of recent advances in the knowledge of cortical-basal ganglia physiology. Schwartz (1998) argues that if OCD symptoms are related to a malfunction in cortical circuit activation and, in particular to an >error-detection= mechanism in the orbital cortex, then activation of an alternative circuit through the focused performance of a familiar alternative behaviour might over time ameliorate the discomfort related to the faulty brain mechanism. By shifting to another task as a systematic response to OCD symptoms, patients learn new adaptive responses to intrusive OCD thoughts and urges, and thereby change the metabolic activity of the cortico-striate circuits. The fact that the metabolic activity in these circuits seems to change in a manner which is significantly related to changes in symptom expression underscores the possibility that there is a causal relationship between the change in behaviour and the change in brain function.

In a study by Schwartz, et al., (1996), nine patients with OCD were studied with positron emission tomography before and after 10 weeks of cognitive behaviour treatment. Results indicated that the responders demonstrated a significant bi-lateral decrease in caudate glucose metabolic rates that were greater than those seen in poor responders to treatment. They concluded that in the light of their findings, it should not be surprising that the deconditioning of OCD symptoms accomplished through cognitive behavioural therapy might be accompanied by a change in function in the head of the caudate nucleus, and in the cortico striato-thalamic system of which it is a key element.

6.6 General treatment outcome

Keuthen et al, (1998) are optimistic regarding treatment outcome for trichotillomania in general. These researchers conducted a retrospective survey, using multiple measures of assessment in a large group of patients who suffered from trichotillomania. They examined the reported improvement in hair pulling, self esteem, psycho-social functioning, depression and anxiety. Sixty three patients participated in their study. Thirty three patients (52%) rated themselves as treatment responders. All subjects received part or all of their treatment at the speciality clinic of the researchers, who were confident that adequate trials of each treatment modality were attempted in most cases. Fifty-seven patients (90%) received behavioural treatment, 46 patients (73%) received medication treatment, and 41 patients (65%) received both medication and behavioural treatment. Twenty-seven patients (59%) were given clomipramine, 27 patients (59%) fluoxetine, 23 patients (50%) paroxetine, 11 patients (24%) venlafaxine, 10 patients (22%) setraline, 10 patients (22%) flavoxamine and 7 patients (15%) lithium carbonate. In addition 23 patients (37%) were treated with hypnosis, 31 (49%) were treated with psychotherapy, and 21 patients (33%) participated in a support group. Statistical significant improvement was found in all the areas controlled for.

The findings of the afore mentioned researchers are in direct contrast to earlier reports indicating limited treatment efficacy. These authors suggest that while research investigations into better treatments for trichotillomania continue, their results suggest

that current state-of-the-art treatment methods benefit a substantial proportion of patients with trichotillomania who seek treatment.

CHAPTER SEVEN

THEORY OF COGNITIVE BEHAVIOUR THERAPY

Cognitive Behaviour Therapy is the field of applied psychology that is unified by a belief in the central role played by mediating between knowledge structures or thinking processes in explaining, and changing human behaviour. While acknowledging a reciprocal interaction between cognition and emotion, many diverse

orientations within cognitive therapy tend towards viewing cognition from the point of view of its contributory role in influencing emotion (Blackburn and Twaddle, 1996).

7.1 Historical background

The philosophical origins of cognitive behaviour therapy can be traced back to Stoic philosophers, particularly Zeno of Citium (fourth century BC), Chrysippus, Cicero, Seneca, Epictetus and Marcus Aurelius. Epictetus wrote in *The Enchridion:* AMen are disturbed not by things but by the views which they take of them≅. Like Stoicism, Eastern philosophies such as Taoism and Buddhism have emphasised that human emotions are based on ideas (Beck, Rush, Shaw and Emery, 1979). Control of most intense feelings may be achieved by changing one=s ideas,

Freud initially presented the concept that symptoms and affect are based on unconscious ideas, (Freud, 1953). The Individual Psychology of Alfred Adler emphasized the importance of understanding the patient within the framework of his own conscious experiences. Adler considered therapy as an attempt to unravel how a person perceives and experiences the world, (Adler, 1958).

Philosophical emphasis on conscious subjective experiences stems from the works of Kant, Heidegger, and Husserl. This phenomenological movement has substantially influenced the development of modern psychology. The application of the phenomenological approach to specific pathological states is exemplified by the

works of Jaspers (1968), Binswanger (1958), and Straus (1966). The influence of developmental psychologists such as Piaget (1950, 1960) is also apparent in the formulation of cognitive psychotherapy.

Developments in behavioural psychology have also emphasized the importance of the patient=s cognitions. Bowers (1973) has argued for an interactional model of subject and environmental events and against the situationalism of classical behavioural approaches. Increasing emphasis on cognitive restructuring or modifying cognitions is reflected in the work of Lazarus (1972) who is of the opinion that the bulk of psychotherapeutic endeavours may be said to centre on the correction of misconceptions.

American psychotherapists have outlined more specifically how the therapist might modify cognitions in a systematic manner during psychotherapy. Kelly (1955) developed personal construct therapy to alter the patient=s ongoing conscious daily experience. In >fixed role= therapy, the patient assumes a role based on assumptions about the world or himself which are not congruent with his usual beliefs. In this new role, the patient is brought face to face with assumptions he had been making about himself and his interaction with others. Kelly referred to these underlying assumptions or beliefs as >personal constructs=.

Berne (1961, 1964) and Frank (1961) added different methods and conceptualizations to therapies designed to alter the ongoing conscious experience or cognitions of the patients.

Ellis (1973) provided a major impetus in the historical development of cognitive-behavioural therapies. He linked the environmental or activating event (A) to the emotional consequence by the intervening belief (B). Thus, his Rational Emotive Psychotherapy aims at making the patient aware of his irrational beliefs and the inappropriate emotional consequence of these beliefs. Rational Emotive Psychotherapy is designed to modify these underlying irrational beliefs. Other techniques to raise irrational beliefs into awareness and to modify them have been presented by Maultsby (1975); his so called Rational Behaviour Therapy.

Recent contributions to the development of cognitive therapy by behaviourally orientated writers (Mahoney, 1974; Meichenbaum, 1977; Goldfried and Davison 1976; and Kazdin and Wilson 1978) have provided a firmer empirical and theoretical basis for further growth in this field.

However, it was Beck=s (1979) model that placed Cognitive Behaviour Therapy thoroughly on the map. This heuristic cognitive model was developed as a reaction to the theoretical excesses and practice limitations of classical psychoanalysis and to the rigidly restrictive nature of radical behaviourism

7.2 Cognitive theory of personality

According to Corsini and Wedding (1989), cognitive behaviour therapy emphasizes the role of cognitive processing in emotion and behaviour. An individual=s emotional

and behavioural responses to a situation are largely determined by how that individual perceives, interprets, and assigns meaning to that event. Defining cognition is a difficult task as the term cognition is used variably as a label for an intra psychic structure, a process, or an outcome. It includes such phenomena as perceptions, deployment of attention, labelling, coding and categorising stimuli, memory and retrieval, thinking reasoning, judging, problem solving, values, philosophies, beliefs standards, rules, expectations, imaging, and conditioned linkages between stimuli both stored and conscious (Granvold, 1994). In an attempt to provide an organising framework for the various cognitive constructs, Ingram (1983) has devised a useful taxonomic system in which cognition is divided into four interrelated but conceptually distinct major elements namely:

1) Cognitive structure which refers to the way in which information is organised and internally represented.

2) Cognitive propositions which refer to the content stored in the cognitive structures. Stored content may be knowledge either received from external sources, or internally generated.

3) Cognitive operations which serves as the mechanism for information processing and involves selective attention to available stimuli, perception, encoding, storage and retrieval.

4) Cognitive products which are the results of information processing and exists as thoughts, self-verbalisations and explicitly held outcomes such as beliefs, opinions, attitudes, values, judgments and conclusions.

Corsini et al., (1989) continue by stating that cognitive therapy views personality as shaped by central values, or super-ordinate schemes. Kendall and Ingram (1987) note that the concept of cognitive structure and cognitive propositions are together usually termed as schemata. Schemata are inflexible general rules or silent assumptions (beliefs, attitudes, concepts that (1) develop as enduring concepts from past (early) experiences; (2) form the basis for screening, discriminating, weighing and coding stimuli and (3), form the basis for categorising, evaluating experiences, making judgements and distorting reality situations (Rush and Beck, 1978). The individual's perceptual filters, views of self, others and the world, and cohesive factors that form the bases of appraising and judging are all based on schemata. Schemata as memory structures fulfil the function of matching objects or situations to previous knowledge and guiding the search for more information when there is no match (Winfrey and Goldfried, 1986). Making the connection between current stimuli and one=s unique psychosocial history involves schemata. In this manner, current phenomena become integrated into a stored matrix of associations. Schemata can be identified as etiologically accountable for such problems as distortion in thought processes, maladaptive emotional episodes and faulty, exaggerated or unrealistic expectations of self, others or environmental conditions (Granvold, 1994). Furthermore, schemata may be adaptive or dysfunctional (Corsini et al., 1989).

Beck, Epstein and Harrison (1983), stated that certain clusters of personality attributes or cognitive structures are related to certain types of emotional response. They described sociotrophy or social dependency and autonomy as two personality dimensions present in depression and possibly other disorders. Whereas the

sociotrophic personality becomes depressed after the disruption of relationships, the autonomic personality becomes depressed when they cannot reach their self-set goals.

Improvement in the problem areas can be achieved by targeting underlying schemata for change, and effectively modifying them.

7.3 Methodological assumptions

Cognitive therapy came to be associated with a set of explicit assumptions, or guiding principles, which have become the defining characteristic of the approach. According to Blacburn et al., 1996, there are broadly speaking eight of them;

- **The centrality of the cognitive conceptualisation -** The models became conceptually driven in that, rather than being a shotgun technique-orientated approach with no theme or focus, they stressed the importance of a clear treatment conceptualisation guiding a series of organised and focused treatment strategies.

- **The phenomenological emphasis -** This approach to psychopathology naturally led to the patient=s idiosyncratic subjective experience becoming the central focus of the therapeutic exchange. This became one of the most distinctive early aspects of cognitive therapy. Seeing the >world through the patient=s eyes= naturally meant relying heavily on his own reports of his experience, and taking it at face value. The point of variation across therapists

and conceptual models was the focus that each put on the different aspects of that experience (content, process, structure).

X **The collaborative nature of the therapeutic relationship** - The emphasis on phenomenology requires collaborations inviting the patient and therapist working in an atmosphere of negotiation - a direct descendant of Kelly=s (1955) notion of patient and therapist as Apersonal scientists=. Beck (1979) and his colleagues coined the phrase >collaborative empiricism=, which encapsulated the idea of a team approach in which the patient provides the raw data to be investigated with the therapists guidance. The objective of such a relationship is to develop a milieu in which specific cognitive change techniques can be applied in the most efficient manner. Beck=s model originally viewed difficulties in the therapeutic relationship, such as transference, as technical problems to be identified and examined in the same fashion as any other cognitive behavioural data. The emphasis was on minimising its occurrence in therapy. This stance was common in all the models of the time with more attention being given to technique than to the relationship between therapist and patient *per se* (Meichenbaum (1985); Rehm (1977).

X **Active involvement of the patient** - With a collaborative type of relationship the process of therapy evolved into a highly interactive one. The model heavily emphasized actively engaging the client to devise and experiment with strategies for cognitive and behavioural change. Both therapist and patient

came to have a role in selecting therapeutic targets and negotiating how such targets should be approached. This was, and still is, a more or less unique aspect of cognitive therapy. A study by Vallis, Shaw and McCabe (1988) is of interest here. They compared therapist competency ratings of cognitive therapists and general non-specific therapists, using a checklist of therapist behaviours. They observed among the most competent cognitive therapists a greater frequency of brief questions necessitating >yes/no= answers, and of interruptions. These are classed as >errors= in communication on this scale, but in cognitive therapy such questions and interruptions are regarded not as errors but as a critical part of the active collaboration between patient and therapist.

X **The use of Socratic questioning and guided discovery -** The type of questioning used within cognitive therapy became known as Socratic dialogue. Rather than interpreting the patient=s thoughts and actions, the therapist's role is to raise questions about thought, feelings and actions, thereby encouraging the patient to think for her/himself: - a process of guided discovery. This was in sharp contrast to communicative interpretations. Apart from running the risk of >mind reading= and of misunderstanding the patient, the interpretations were considered to risk putting her/him in a compromising position- in that it is simpler to agree than to disagree, or to seem ungrateful or difficult. The Socratic questioning format of cognitive therapy allowed the patient to maintain integrity and the therapist to gather the most accurate data - accuracy being an important prerequisite for developing hypotheses, the building blocks

of cognitive conceptualisation (Vallis et al., 1988).

X **Explicitness of the therapist** - From the idea of negotiation or collaboration followed the requirement that the therapist share with the patient explicitly the model, his or her own working hypotheses and ideas on conceptualisation. It also entailed admitting mistakes and agreeing to disagree. There was to be no place for >private= therapist models within the cognitive therapy paradigm, as this would sabotage the collaborative stance.

X **The emphasis on empiricism** - The cognitive behaviour models have always been heavily empirical, creating and testing out the working hypotheses emerging from the collaborative guided discovery process via a number of techniques, broadly categorised as cognitive (focussed primarily on modifying thoughts, images and beliefs) and behavioural (focussed primarily on modifying overt behaviours). The categories are not mutually exclusive. For example, a behavioural technique such as assertiveness training has cognitive components to it, can be used to accomplish cognitive changes such as adjustment in expectancies regarding the consequences of assertion, as well as changes in interpersonal behaviour itself (Vallis et al., 1988)

X **The outward focus -** The empirical active aspect of cognitive therapy was designed to facilitate the generalisation of in-session therapeutic change : the so called >homework= of cognitive therapy. Such generalisation activities are explicitly determined, monitored and evaluated. It has become a general rule

that cognitive therapy does not focus selectively and entirely on the in-session interaction. Much attention is given to functioning outside the therapy context. Several studies suggest that compliance with homework assignments is related to better outcome (Vallis et al., 1988).

7.4 Cognitions as a target for change

The focus of cognitive therapy is the modification of mediational factors that are identified as operative in the client=s personal and interpersonal distress. Targets for change include both cognitive outcomes and processing. Beliefs, expectations and meaning attached to life events may be the objects of exploration and change. Several interrelated cognitive variables can be targeted for change, including perception, expectancies, attributions, information processing (which includes cognitive distortions) and beliefs.

Perception has been identified as an active process rather than the passive receptivity of stimuli. Prior knowledge stored within the individual converges with external sensation in the construction of perception. This stored information influences perception with regard to selective attention, meaning of stimuli and memory. Perceptual selectivity may account for maladaptive functioning. In depression, for example, negative input is screened in and positive input is screened out (Beck and Emery, 1985). Perceptual errors may arise from evaluative beliefs, values and stored prior experiences. For example, individuals who have experienced physical abuse and other forms of aggressive behaviour may be less reactive to aggression than those

who have experienced passivity. They may be so desensitised to aggression that they unconsciously ignore (screen out) specific acts of aggression. Perceptual error may also result from factors related to the individual=s current physical and emotional state. Fatigue and emotional arousal frequently influence perception. One who is in an extreme emotional state is strongly predisposed to perceptual error. People=s subjective expectancies that they can significantly influence life events (outcomes) have been the focus of much research.

According to Rotter (1966), the concept of locus of control can be identified as the degree to which people view themselves as capable of producing a specific outcome. The view that an event is contingent on one=s own behaviour is termed as internal locus of control, and the belief that life events are controlled by outside forces, is termed as external locus of control. People with an internal locus of control seem to be better adjusted to life whereas a sense of externality appears to coincide with maladjustment and disturbance. Seligman (1975) identified the concept of learned helplessness as characteristic of depressed persons. They see themselves as unable to control significant life events. They view success and failure as independent of their own skill and influence.

Attributions are the beliefs or statements a person uses to explain why a behaviour, condition or event has occurred. To resolve causal ambiguity, people tend to seek singular, definitive, simplistic explanations drawing on contextual information or beliefs held regarding self, others and the world. Such explanations may be fraught

with error and may result in negative views and conclusions and corresponding maladaptive emotional responses (Granvold, 1994).

Locus of control may have an influence on causal attributions. People with a internal locus of control will view an achievement as a product of their own efforts whereas a person with an external locus of control may view outcome as caused by such factors as luck, chance or fate. Negative attributions may take the form of blaming where the person blames another for the outcome of an event.

Abramson, Seligman and Teasdale (1978), categorize attributions into three dimensions; stable-unstable, internal-external and specific global. Outcomes may be attributed to factors that are more changeable (unstable) or less changeable (stable). E.g. AI lost the game because I do not play so well= (stable) as opposed to > I lost the game because I had an off day≅ (unstable).Internal-external dimensions involves the attribution of outcomes to oneself or to outside factors - other people or environmental conditions. Negative outcomes may be attributed to oneself, whereas positive potential outcomes are attributed to outside forces. Global versus specific attributions refer to the representativeness of the causal explanation concluded. A negative attribution with global meaning has more far reaching implications and is correspondingly less responsive to change than a more specific negative attribution. For example, a person who struggles marginally with science may take the view that he/she does not have academic abilities (global), or may hold the view that science is uncharacteristically difficult for him/her (specific).

According to Tataryn, Nadel and Jacobs (1989), information processing has been identified as the cognitive scientist=s central dogma. Stimulation received at the periphery is coded by receptors, represented in some spatiotemporal set of brain activities and then processed, transferred and stored in the service of specific ends or needs. There is evidence that errors in information processing partially cause, or at least sustain, many forms of psychological dysfunction (Miller and Porter, 1988). Distortions in this process can occur in many ways. Freeman (1990) noted that these distortions can occur in many combinations. The following is a description of the most common cognitive distortions;

Arbitrary inference- refers to the process of drawing a specific conclusion in the absence of evidence to support the conclusion or when the evidence is contrary to the conclusion. Freeman (1983) describes two types of arbitrary inference, namely mind reading (AI know he thinks I am ugly≅), and negative prediction (AI just know that there will be problems with this job≅).

Selective abstraction **-** consists on focusing on a detail taken out of context, ignoring other more salient features of the situation and conceptualising the whole experience on the basis of this fragment.

Overgeneralization **-** refers to the pattern of drawing a general rule or conclusion on the basis of one or more isolated incidents and applying the concept across the board to related and unrelated situations.

Magnification and minimization **-** are reflected in errors in evaluating the significance or magnitude of an event that are so gross as to constitute a distortion.

Personalization **-** is the act of relating a negative event or situation to the self without adequate causal evidence to make the connection. There are two forms of personalization. In the first instance people reach an arbitrary conclusion that they have caused a negative event, (AIf I did not fall asleep I could have saved the child from drowning≅). In the second form of personalization, a person reaches the arbitrary conclusion that they are the object of a negative event and are therefore causally connected to it, (AIt is raining especially to make me late for work≅).

Absolutistic, dichotomous thinking **-** is manifested in the tendency to place all experiences in opposite categories, for example, flawless or defective, immaculate or filthy, saint or sinner. In describing himself the person selects the extreme negative categorization.

7.5 Cognitive behaviour intervention methods

An early objective in treatment is to develop client awareness of the cognitive model and of the impact that their cognitive processes have on their functioning. This goal can be achieved through didactic presentation or through the use of written material. Clear understanding can best be achieved by applying cognitive methods to a client's concern or problem. The therapeutic relation is formed as the client and therapist explore cognitive, emotional and social or environmental factors that could be

relevant to the client=s problem or dysfunction. Cognitive variables addressed include thoughts, perception, attributions, information processing, expectancies, beliefs and motivation. Emotion is assessed in terms of frequency, intensity and duration and factors associated with the emotional episodes are explored (trigger events, contextual factors and reinforcing consequences).Behavioural competencies and skills as well as deficits are identified. Social/environmental factors such as opportunity, resource limitations and so on are considered. This first session should include the use of relevant tests and measures as well as a thorough interview. The outcome of this exploration is the collaborative formulation of a treatment plan, which includes a series of testable hypotheses.

The testing process requires that clients perform homework. They may be expected to track their thoughts or thought patterns, record the restructuring of their dysfunctional thinking, track their negative emotion and corresponding cognitive, behavioural, and environmental factors, and perform and record the performance of targeted behaviours or tasks. The homework is used in treatment sessions to validate the hypotheses, to modify existing strategies, and to generate new directions in treatment. Cognitive intervention comprises three main areas;

7.5.1. Cognitive restructuring

Cognitive restructuring is an intervention method in which dysfunctional schemata and thinking patterns are identified. These are then appraised as both ill-formed (having little or no logical support or evidence) and as interfering with the pursuit and

attainment of goals (producing undesirable emotional or behavioural consequences). Methods are then applied to modify them. The exposure, challenge, and disputation of faulty cognitions are accomplished through the implementation of a logical-empirical method of scientific questioning, in which supportive evidence is sought to validate or invalidate them. The restructuring of faulty perceptions, faulty attributions, information processing errors, and schematic errors can be accomplished through exposing the distortion (client awareness) and guiding the client in observing, evaluating, disputing and modifying the cognitive distortion (Ellis, 1977). Homework assignments are employed to promote the application and habituation of adaptive thinking and speaking.

The following guidelines are offered (Granvold, 1994) for use in the formulation of an intervention strategy:

X Expose the belief(s) that are hypothesised to be operative in a given situation.

X Gain the historical meaning of the belief.

X Determine the strength of the belief now and in the past.

X Determine the way in which the belief has been used and the perceived consequences of maintaining the belief, both past and present.

X Determine the matrix of beliefs associated with the target belief.

X Establish the cognitive, emotional, behavioural and social consequences of maintaining the belief.

X Guide the client in a search for the evidence to support the belief.

X Guide the client in a search for evidence that may compete with or refute the target belief.

X Train the client to monitor and record (1) faulty thinking associated with the belief and (2) emotional, behavioural and social consequences of the faulty thinking.

X Assign homework to transfer this training to the client=s daily life.

X Check the client=s homework and guide the repetitious challenging of the faulty schemata.

Cognitive restructuring can be used in conjunction with other cognitive methods (thought stopping, covert sensitization, guided imagery, and behavioural methods (behaviour rehearsals, skills training, deep muscle, or other, relaxation). It is highly desirable to promote specific behaviour changes along with cognitive restructuring assignments. (For example the depressed client can be instructed to increase the performance of pleasurable activities or those involving mastery).

7.5.2. Problem solving

Problem solving has been classified alternatively as a behavioural procedure and a cognitive-behaviour procedure. D=Zurilla and Goldfried (1971, p. 108) define problem solving as *Aa behavioural process whether overt or covert in nature which (a) makes available a variety of potentially effective response alternatives for dealing with the problematic situation and (b) increases the probability of selecting the most effective response from among these various alternatives≅.* Problem solving deficiencies have been found to be associated with a variety of personal and

interpersonal problems, and training in problem solving has been found to improve the functioning of various clinical populations.

There are several approaches to problem solving, but the most widely accepted is the five-step model of D=Zurilla and Goldfried (1971);

(1) *Problem orientation* - which relates to the cognitive set of the individual in relation to problem solving and coping with life situations. Problem orientation factors can be problem perception, causal attributions, problem appraisals, beliefs about personal control, and values concerning the commitment of time and effort to problem solving.

(2) *Problem definition and formulation* - involves an assessment of the problem and the establishment of an achievable goal.

(3) *Generation of alternative solutions* - involves brainstorming to generate alternative solutions to the problem.

(4) *Decision making* - when the alternatives are evaluated and a solution is selected for implementation.

(5) *Implementation and verification of solutions* - is designed to determine the effectiveness of the problem solving effort and, if the solution is found to be lacking, to promote modification of the original solution or a return to step 3 to select another possible alternative solution.

The goal of problem solving is to treat life problems that are antecedents of maladaptive responses and are considered to be causally related to these responses. Examples of problem solving issues are as follows. (Granvold, 1994);

X How can I make better use of my time?

X How can my husband and I do more novel activities?

X How can I reduce the disruptive behaviour of my children at the table?

X How can I get my aging father to take better care of himself?

X How can I reduce the stress of my job?

The effectiveness of problem solving can be extended through the use of such adjuncts as cognitive restructuring and training in social skills, assertiveness, self-control techniques and coping skills.

7.5.3. Self-instruction training

Self instruction involves the use of self verbalization to guide the performance of a task, skill or problem solving process. Drawing on the work of Luria (1961) and Vygotski (1962), Meichenbaum and Goodman (1971) developed the method to treat hyperactive, impulsive children. Before performing tasks automatically, young children verbalize instructions. As they develop skill and begin to act automatically, the verbalization becomes sub vocalized.

Meichenbaum (1975) recognised that verbal mediation could be inserted between a stimulus and response to prevent maladaptive behaviour and performance deficiency. In this manner, the stimulus-response chain can be interrupted, and the maladaptive respose can be inhibited. The anticipated result is an increased likelihood that an adaptive response will follow a given stimulus.

Self -instruction training provides the client with step by step self statements (including verbal self-reinforcement and coping statements) for the development of competency in the performance of targeted tasks and skills. A five step procedure is used to instruct the client (Meichenbaum, 1977);

(1) The therapist models performance of the task while talking out loud (cognitive modelling).
(2) Clients perform the same task while receiving verbal guidance from the therapist (overt, external guidance).
(3) Clients perform the task while instructing themselves out loud (overt self-guidance).
(4) Clients perform the task while whispering instructions to themselves (faded, overt self- guidance).
(5) Clients perform the task while instructing themselves privately (covert self-instruction).

The self-statements modelled by the therapist and rehearsed by the client may incorporate error correction, self praise and coping statements. Initial self instruction training should begin with simple tasks, followed by more complex skills such as problem solving. In the same fashion, complex tasks should be broken down into manageable incremental steps to allow for response chaining and successive approximation. Homework may be assigned in which the client is to practice the rehearsal or self instruction while performing tasks.

The verbalization modelled by the therapist and rehearsed by the client includes several performance relevant skills (Granvold, 1994):

X *Problem definition* - ≅What is it that I am trying to do?≅

X *Statements regarding task completion* - AI am going to ask her for a date≅.

X *Guidance of performance by self instruction* - ARelax, approach her comfortably, and make the invitation.≅

X *Coping self statements and error correcting options* - AI can still approach her even if I feel some anxiety - that is okay≅.

X *Self-reinforcement* - AThere, I did it≅.

Self instruction has been used to treat a wide range of academic and clinical problems in various populations. Effective results have been reported in improving the self control of hyperactive, impulsive children, reducing children=s aggressiveness, developing social skills in social isolates, enhancing memory recall in children, improving creativity in college students, reducing test anxiety; improving problem solving skills in the elderly, and reducing psychotic speech in adult schizophrenics. It has been modified to include such components as operant procedures (for example, social and token reinforcement), relaxation training, stimulus-modelling films and tapes and covert modelling procedures.

7.6 Research Hypothesis

The hypothesis of this study follows from 3 tenets; Firstly that trichotillomania has a neurobiological basis as was indicated by functional neuroimaging studies (Grachev, 1997). Secondly that Cognitive Behaviour Therapy has been demonstrated to be effective in the treatment of the obsessive-compulsive spectrum of disorders (Rothbaum and Ninan, 1992; Hollander et al., 1996). Thirdly that, as with pharmacological intervention, Cognitive Behaviour Therapy has been shown to effect changes on a neurobiological level which is detectable by neuroimaging (Baxter et al., 1992 ; Schwarts et al., 1996).

On the basis of the above literature review, the following hypotheses are put forward:

1. It is postulated that as with PET, SPECT will be able to determine a neuroanatomical area in the brain of patients suffering from trichotillomania.

2. It is postulated that by making use of a Cognitive Behaviour Therapy intervention programme, without medication, symptom relief in trichotillomania will be achieved.

3. It is further postulated that by making use of SPECT scanning, a detectable difference in the patterns of cerebral blood flow (neuronal activity) between baseline and that following a 9-12 week Cognitive Behavioral Treatment, in trichotillomania, would be demonstrated.

CHAPTER EIGHT

SUBJECTS, PROCEDURE AND METHODS

8.1 Study design

The present study took the form of an experimental design, using within subject comparisons, before and after Cognitive Behaviour therapy intervention.

8.2 Subjects

8.2.1 Criteria for selection

Subjects were included in the study provided;

1. They met the diagnostic criteria for trichotillomania as set out in the DSM IV (1994), refer to chapter three (3.1)
2. They did not suffer from any other Axis 1 illness as set out in the DSM IV (1994), as to keep the variables to a minimum.
3. They were female and not pregnant (since the radio-active isotope in ^{99m}Tc-HMPOA which is used for the SPECT scanning is not recommended while pregnant). It was decided to exclude males from the present study due to the asymmetry present in the male brain as well as difficulty in recruiting male subjects due to the small numbers of males suffering from trichotillomania.
4. They were over 21 years of age (legally this age allows them to sign the consent form independently)
5. They were not on any medication while participating in the study, so that any changes before and after the SPECT can be attributed to Cognitive Behaviour Therapy).

8.2.2 Subject sample

Subjects were recruited from the Trichotillomania Support Group of South Africa, as well as from referrals from private practices. The Trichotillomania Support Group is a self help group that comprises of trichotillomania sufferers. They meet approximately once a month to discuss and support members on the issue of trichotillomania. The promote awareness of the illness by giving talks on national television and by publishing articles in local popular magazines. They have approximately 50 members. All subjects came from the Witwatersrand area, which is a geographical area with a radius of approximately 60km around Johannesburg. Twelve subjects participated in the study. A comprehensive description of the sample is provided in chapter 10 (10.1 and 10.2)

8.3 Procedures

The research was conducted in the following order;

1. Ethical clearance was obtained (Appendix A1)
2. The study was explained to each participant by the investigator, at an initial meeting. Informed consent was obtained from each subject. (Appendix A2)
3. Once the consent form was handed back to the investigator, a semi-structured interview was conducted by the investigator and a psychiatrist to obtain a general background history of the patient, to determine whether the subject met the inclusion criteria, as well as to rate the subject's severity of

trichotillomania, using the Psychiatric Institute Trichotillomania Scale (PITS). (Appendix A3) The subject's level of anxiety was determined by a separate rating scale (The Hamilton Anxiety Rating Scale) (Appendix A4) on the day of the scans.

4. The Temperament and Character Inventory (TCI) (Appendix A5) were completed.
5. The Hamilton Anxiety Rating Scale (Appendix A4) was completed and the first scan was obtained
6. A 12-16 weeks Cognitive Behaviour Therapy programme (Table 9.1) was conducted.
7. On completion of the programme a second scan was obtained, the Hamilton Anxiety Rating Scale and the PITS were completed again.

8.3.1 Ethical clearance

Prior to the study, ethical clearance from the University of the Witwatersrand; - Committee for Research on Human Subjects was obtained. A clearance certificate with the following protocol number was issued:

M 960118 (Appendix A1).

8.3.2 Informed consent

Before commencement of the study, a detailed explanation of the nature and extent of the study was provided to each participant. The patient was informed about the different questionnaires that were to be completed. They were given a brief overview of what the instrument measured, as well as the reason for incorporating the questionnaire into the study. A written information sheet and consent form, (Appendix A2), was handed to the patient to study in their own time. Once the consent form was signed an appointment was made for obtaining the first scan.

8.3.3 Rating scales

8.3.3.1 Rating the severity of trichotillomania symptoms

The Pennsylvania Institute Trichotillomania Scale (PITS), (Winchel, Joneds,Stanley, Molcho and Stanley, 1992), was used to assess the level of trichotillomania, (Appendix A3). The scale rates six descriptive categories of the illness namely; number of sites, severity of hair loss, duration of pulling sessions per day, ability to resist the urge to pull, interference of activities and level of distress, using a rating between 0 and 7. The score sheet provides descriptions of behaviours that serve as guidelines for scoring each category. The guidelines for a semi-structured interview as suggested in the PITS manual were followed to obtain severity ratings. The maximum score that can be obtained is 42 points.

The questionnaire was administered pre and post intervention. The pre and post scores were compared to see if a significant difference exists by making use of Student=s paired t testing as well as Wilcoxon=s matched pairs signed rank testing (one tailed).

8.3.3.2 Rating the level of anxiety

The Hamilton Rating Scale for Anxiety (HAMS-A), (Hamilton, 1959), was used to obtain the level of anxiety (Appendix A4). This scale is the most commonly used measure for anxiety (Schweizer and Rickels, 1997) and is generally acknowledged to be weighted towards the somatic symptoms of anxiety. It comprises 14 items that can be rated between 0-4 points each, 0 indicating none and 4 indicating severe, grossly disabling symptoms. The maximum score that can be obtained is 56 points. The questionnaire was completed by the subject and the researchers together, prior to therapy and after the therapy has been completed. The sums of the pre and post scores were compared using Student=s paired t-testing as well as Wilcoxon=s matched pairs signed rank testing (one tailed).

8.3.3.3 The Temperament and Character Inventory -125 (TCI -125)

Not much data is available regarding personality and character styles in trichotillomania. As the TCI-125 was constructed from a biopsychosocial model the researcher wanted to explore whether general trends of personality and character traits in trichotillomania could be deducted from this scale. The Biosocial Theory of Personality proposed by Clonninger (1986) provides a framework to integrate a

diverse range of possible predictor variabilities including clinical, personality and biological measures. These inherited temperamental traits can be assessed by a self-report questionnaire (Joyce, Mulder and Clonninger, 1994).

A booklet (Appendix A5) comprising 125 questions which requires the subject to answer either yes or no was given to the subjects to complete in their own time. The TCI comprises a battery designed to assess differences between people in seven basic dimensions of temperament and character. Temperament refers to automatic emotional responses to experience that are moderately heritable and stable throughout life; the four measured temperament dimensions are Novelty Seeking, Harm Avoidance, Reward Dependence and Persistence. According to this framework the genetically determined dimensions of temperament can be linked to neurochemical systems. In contrast, character refers to self-concepts and individual differences in goals and values, which influence voluntary choices, intentions and the meaning of what is experienced in life. Differences in character are moderately influenced by sociocultural learning and mature in progressive steps throughout life. The three measured character dimensions are Self-Directedness, Cooperativeness and Self-Transcendence. These aspects of personality interact with each another to motivate adaptation to life experiences and influence susceptibility to emotional and behavioural disorders.

The TCI includes a neurobiological based operant learning model to guide the rational development of descriptors for temperament (Clonninger, 1987; 1991). Initially it was hypothesized that the temperament systems in the brain were functionally organized

as independently varying systems for the activation, maintenance and inhibition of behaviour in response to specific classes of stimuli. Behavioural activation involved the activation of behaviour in response to novelty and signals of reward or relief of punishment; accordingly individual differences in such activities were called Novelty Seeking. Behavioural inhibition occurred in response to signals of punishment or non-reward, so individual differences in inhabitability were called Harm Avoidance. Behaviour that was previously rewarded was later maintained for a while without continued reinforcement, and individual differences in such maintenance were called Reward Dependence. The temperament dimensions were designed to assess individual differences in associative conditioning, whereas the character dimensions were designed to assess individual differences in conceptual learning. This character distinction corresponds to the differentiation of procedural learning, which involves differences in habits and skills, and propositional learning, which involves differences in goals and values. More specifically, at the level of individual dimensions, each temperament dimension was hypothesized to be regulated neurochemically by a complex distributed network of brain connections. Monoaminergic cell bodies in dense nuclei in the brainstem send projections throughout the brain that allow widespread but specific modulation of responses to stimuli (Clonninger, 1994). Validity and reliability for this scale have been demonstrated (Svrakic, Whitehead, Przybeck and Clonninger, 1993).

The results were scored, according to the instructions set out in the TCI manual (Clonninger, Przybeck, Svrakic and Wetzel (1994). Scores were compiled for each of

the 7 dimensions and expressed as a percentage. The interaction between high and low scores was described.

8.3.4 Acquisition of the first or baseline ^{99m}Tc-HMPOA SPECT scan.

SPECT is a dynamic brain imaging technique that grew out of the evolution of increasingly more sophisticated approaches to understanding the pathophysiology and biology of neurological and psychiatric disorders over the past few decades. According to Schukit (1992), the scanner used in SPECT is a modification of approaches already available in many radiology departments. It detects the number of photo emissions released when a gamma ray collides with a crystal, usually sodium iodide, in a region of interest (ROI). A computer is used to analyse the intensity of the energy of the ray as it hits the crystal as well as its trajectory and calculates the original source of the energy. The number of crystal activations in a given amount of time is proportional to activity at any given point in the region of interest, and is described in terms of a pixel. Earlier methods used a single camera that rotated around the head, whereas the newer more sensitive approaches incorporate information from multiple cameras: both methods feed the data into a computer to generate a three dimensional or tomographic image.

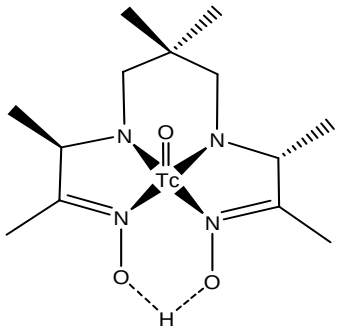


Figure 8 **^{99m}Tc- HMPAO (Ceretec)**

The SPECT approach can use several radio pharmaceuticals, such as a labelled Xenon gas, or an amphetamine labelled with iodine. The third radio pharmaceutical that can be used is a compound (hexamethylpropyleneamine oxime) labelled with a form of Technicium, abbreviated as ^{99m}Tc- HMPAO and marketed as Ceretec. HMPAO can be stored for months (Figure 8), can be given in larger doses, easily crosses over from the blood to the brain and imaging may begin as early as 15 minutes post injection. Ceretec is approved by the Food and Drug administration (Heertum, 1992).

Similar to applications in other radiological procedures, SPECT may assist the practitioner in confirming a suspected diagnosis. SPECT may help to determine the cause of a disorder and may be able to guide therapy by imaging alterations in brain function as therapeutic management progresses (Devous, 1992). As far as can be determined, no SPECT studies have been undertaken to investigate trichotillomania.

Conceptualising the similarities and differences between trichotillomania and the other obsessive-compulsive spectrum of disorders in terms of the aforementioned could contribute to the further understanding of these illnesses. Furthermore it could provide a basis from which more specific neural striatal pathways involved in these

illnesses can be investigated and described with a view to novel treatment methodologies.

A 10 ml of Potassium Perchlorate dissolved in water was given to the subject approximately 15 minutes prior to the scan. This minimizes Pertechnotate (Tracer) uptake in salivatory glands and thereby reduces background noise due to free Technicium. All subjects received an intravenous injection of 740 Mbq of ^{99m} Tc-HMPAO (Ceretec). The scan was acquired under resting conditions in a dark room, with minimal sensory stimulation. Image acquisition began 10 to 20 minutes after the injection of the radiotracer. Patients were placed in a supine position, with head immobilized on a head rest and secured with Velcro front and/or chin straps. The studies were performed on an Elscint dual-head, rotating Gamma camera (Helix) equipped with low energy, high resolution collimators, in the department of Nuclear Medicine at the Johannesburg General Hospital. Data was collected in 120 projections, on a step and shoot mode, through 180 degree rotation per head using 128 x 128 matrix resolution. Using a preset programme, reconstruction was carried out with a Metz 308 filter on a 64 x 64 matrix and a slice thickness of 1 pixel (0.44 cm). The superior parietal areas were compared by placing the regions of interest at the superior transaxial slice where the superior cingulate gyrus fully appeared. The raw data of the SPECT were processed both by visual assessment and semi quantitative SPECT analysis. The images obtained from the ^{99m} Tc-HMPAO brain scan before and after cognitive behaviour therapy were visually interpreted by a brain expert nuclear physician (Prof. MDT. Vangu) in the Department of Nuclear Medicine at the Johannesburg General Hospital.

8.3.5 Cognitive Behavior Therapy programme (CBT)

A programme (Table 8) incorporating the techniques of Cognitive Behaviour Therapy was followed. The programme was based on the guidelines set out by Rothbaum and Nina (1992).

Therapy was conducted by the researcher (Clinical Psychologist). Patients were seen once a week for approximately an hour. The first session as well as session 7 (Autogenic relaxation and guided imagery) lasted for an hour and a half. The programme was strictly adhered to.

Table 8 Cognitive Behaviour Therapy treatment programme

Session	Focus
1	a) Treatment outline and education concerning trichotillomania b) Information gathering c) Awareness training
2	Habit reversal training a) Competing response b) Response delay
3	a) Rationale for targeting anxiety and stress in the treatment of trichotillomania b) Relaxation training - Progressive deep muscle relaxation c) Breathing control
4	a) Rationale for learning theory b) Beck=s list of distorted thinking
5	Thought stopping Reality testing
6	Cognitive restructuring
7	a) Autogenic relaxation

	b) Guided imagery
8 +	Monitoring and reinforcement of all previous sessions
Final	a) Relapse prevention b) Termination

8.3.6 Acquisition of the second ^{99m}Tc-HMPAO SPECT scan

A second scan was taken from all patients in exactly the same manner as described previously. Prior to the scan a Hamilton Anxiety Rating scale and a Psychiatric Institute Trichotillomania Scale (PITS) were completed by the examiner and the patient.

8.3.7 Statistical Analysis

Statistical analysis of the SPECT data was conducted by a statistician in the Department of Nuclear Medicine at the University of the Witwatersrand. Parametric (Student's paired t-testing) were obtained.

As a comparative measure statistical analysis was also undertaken by an independent statistician in the Department of Biostatistics at the University of the Witwatersrand. Both parametric (Student=s paired t-testing) and non-parametric (Wilcoxon=s matched pairs signed rank test, one tailed) were used to determine the level of difference before and after the intervention for the SPECT scans, the Hamilton Anxiety Rating Scale and the Pennsylvania Institute Trichotillomania Scale.

CHAPTER NINE

RESULTS

9.1 Subjects

Twelve unmedicated female subjects meeting the inclusion criteria consented to participate in the study. The demographics of this group matches the description in the literature. As can be seen from Table 9.1 the average age of the subjects was 32,35 years. Nine (75%) of them had 12 years of formal education. One (8,3%) received 13 years of formal education, one (8,3%) had 15 years of education , while two (16,6%) had 16 years of formal education. Although a wide spectrum of careers were practiced, 11 (91,6%) of the participants reported that their primary level of stress was employment related. Most of the participants seemed to have an over-involvement in

their work, taking excessive responsibility, even when it is not entirely part of their job-description.

Table 9.1 Subjects demographic data

Subject number	Age	Level of education	Type of employment
1	55 years 6 months	Matric*	Risk management – banking
2	40 years	Matric	Secretarial
3	29 years 6 months	Matric	Graphic artist – Own company
4	39 years 9 months	Matric	Insurance Broker
5	23 years 1 month	Matric	Sales Assistant
6	33 years 6 months	Matric	Insurance Broker
7	25 years 6 months	Matric -One year secretarial course.	Housewife Part-time administrative duties
8	38 years 4 months	B.A. (Fine Arts)	TV Producer
9	37 years 10 months	B. Llb.	Administrative Officer
10	22 years 7 months	B.Sc (Hon). Physics	Research Scientist
11	22 years 1 month	Matric	Administrative Officer
12	21 years	Matric	Administrative Officer

*** Matric is equivalent of 12 years of formal schooling**

The average age of onset of the illness was 12,25 years. Two (16,6%) of the participants started pulling at the age of 15 years, one (8,3%) at the age of 13 years, six (50%) at the age of 12 years, one (8,3%) at the age of 13 years, two (16,6%) at the age of 11 years and one (8,3%) at the age of 10 years (Table 9.2). This is a similar pattern to that which Ratner (1989) describes as late onset, is more common in females and tends to last for decades, as has been described in chapter 2.

Table 9.2 Subject's illness history

Subject Nr.	Age at onset of TTM	Previous intervention	Familial history of mental disorder
1	12 years	Hypnotherapy	Son - head banging
2	12 years	Support group	Sister=s daughter – TTM
3	12 years	Psycho-dynamic therapy	
4	12 years	Psychiatric Psycho-dynamic therapy Hypnotherapy Support group	
5	15 years	Psychiatric	
6	12 years	Support group	
7	12 years	Psychiatric	Mother - possibly TTM
8	15 years		Sister – TTM
9	11 years	Psychiatric	Maternal aunt - Tourette=s - syndrome Mother -possibly TTM Son - skin-picking and ADHD
10	10 years	Psychiatric	Mother - Depression and drinking

		CBT	problem
11	11 years	None	Mother - Depression and finally suicide
12	13 years	Support group	Father - possibly OCD Cousin on father=s side – TTM

Only one of the subjects has never received any intervention for her illness. Five (41,6%) subjects received medication prescribed by psychiatrists. None of these subjects reported continual benefit from this type of intervention. Two (16,6%) subjects received psycho-dynamic therapy and two (16,6%) subjects received hypnotherapy, with neither intervention providing relief of symptoms. Four (33,3%) of the subjects were members of the same support group, who met approximately once every three months. These members found the support group useful and positive although it did not effect any change in their symptoms. One (8,3%) subject started with cognitive behaviour therapy but discontinued after two sessions due to therapist-patient incompatibility.

As far as familial history is concerned, three patients (25%) reported instances of diagnosed trichotillomania in their families. Two (16,6%) subjects reported that their mother might be suffering from trichotillomania, as they seem to pull their hair. However, they were never formally diagnosed as suffering from this illness. One subject (8,3%) reported that her son demonstrated head banging behaviour until the age of 5 years, while one subject (8,3%) reported that her son picks his skin and suffers from ADHD. This same subject reported that her mother=s sister had been diagnosed with Tourette=s Syndrome. Two (16,6%) of the subjects reported that their mothers suffered from depression. The one=s mother also has a drinking problem,

while the other subject=s mother finally committed suicide. One (8,3%) of the subjects reported that her father might possibly suffer from OCD as he engages in excessive list making, and is excessively tidy to the point where it causes difficulty within the family. All of these familial illnesses are described in the literature as part of the obsessive-compulsive spectrum of disorders and as having a high incidence rate in the families of trichotillomania sufferers as was described in chapter 3 (Christenson, McKenzie and Reeve, 1992b ; Swedo, 1993; Hollander et al., 1995).

In terms of sites from which hair was pulled (Table 9.3), the patients in this study demonstrated similar hair pulling behaviour to that which is described in the literature (Muller et al., Winkelman, 1972; Winchell, 1992). Four (33,33%) of patients pulled their hair mainly from one large site on the scalp. Seven (58,3%) pulled on multiple sites on their scalps. Five (41,66) patients pulled from their pubic area. Two (16,66%) patients pulled from their eyebrows, while only one (8,33%) patient pulled her eyelashes. Two (16,66%) patients pulled hair from other parts of their bodies as well.

One patient reported that she used to pull the hair from her blankets, jerseys and from the dog when she was a child, while another patient reported that she used to pull the hair from her knees when she was still in primary school. One patient reported that she uses forceps to aid her with pulling hair from her eyebrows and legs.

Table 9.3 Hair pulling sites in present study

	SCALP		PUBIC HAIRS	EYE-BROWS	EYE-LASHES	REST OF BODY
Subject Nr.	Single large Area	Multiple Areas				

1	**X**					
2	**X**		**X**			
3	**X**					
4		**X**				**X**
5		**X**				
6		**X**				
7			**X**	**X**	**X**	
8		**X**				
9		**X**	**X**			
10		**X**	**X**	**X**		**X**
11	**X**		**X**			
12		**X**				

9.2 Results obtained from the Temperament and Character Inventory - 125 (TCI-125)

The results were scored, according to the instructions set out in the TCI manual (Clonninger, Przybeck, Svrakic and Wetzel (1994). Scores were compiled for each of the 7 dimensions (Table 9.4). The average score for each dimension was expressed as a percentage. The interaction between high and low scores was described.

Table 9.4 Results obtained from the Temperament and Character Inventory - 125 (TCI-125)

Sub-ject nr.	Novelty Seeking	Harm Avoidance	Reward dependence	Persistence	Self directed-ness	Coopera-tiveness	Self tran-Scendence
	20*	**20***	**15***	**5***	**25***	**25***	**15***
1	7	9	10	4	22	23	7
2	10	9	11	3	15	23	6
3	10	18	11	5	7	24	4
4	9	11	12	3	13	23	5

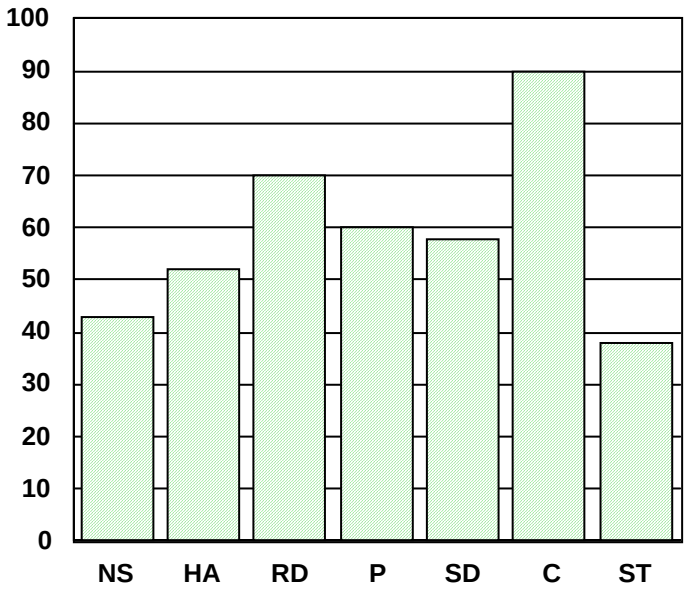
5	11	10	14	1	20	21	4
6	9	7	12	3	15	20	9
7	8	15	13	1	19	23	6
8	11	2	12	5	16	24	10
9	3	14	7	3	14	25	2
10	5	9	3	2	9	17	6
11	12	7	13	3	19	25	3
12	10	13	10	3	5	22	7
To-tal	**105**	124	128	36	174	270	69
%	43,75	51,66	71,11	60,00	58,00	90,00	38,33

*** Maximum points per category**

As far as character is concerned, the highest average score (90%) was obtained on the Cooperativeness subscale (Figure 9.1). Cooperativeness, according to Clonninger (1986) is based on the concept of the self as an integral part of humanity or society; from this self concept are derived feelings of community, compassion, conscience and charity.

Figure 9.1 **Representation of scores on the Temperament and Character Inventory (125)**

Abbreviations NS: Novelty Seeking; HA: Harm Avoidance; RD: Reward



Dependence; P: Persistence; SD: Self Directedness; C: Cooperativeness; SD: Self Transendence.

The lowest average score on the character scale (Figure 10.1) was obtained for Self Transcendence (38,33%), which describes the concept of self as an integral part of the universe and its source. From this self concept are derived feelings of mystical participation, religious faith, and unconditional equanimity and patience.

On the temperament scale the lowest average score (43,75%) was found on the Novelty Seeking scale and the highest average score (71,11%) was obtained on the Reward Dependence scale (Figure 9.1). Low scores on the Novelty Seeking scale are associated with indifferent, reflective, frugal and detached as well an orderly and regimented behaviour.

Trichotillomania has not yet been described in terms of character and temperament as defined by Clonninger (1986). According to Clonninger (1988) Novelty Seeking is believed to reflect individual differences in the brain=s behavioural activation system. Dopaminergic projections from the ventral tegmental area in the brainstem to the striatum (caudate and putamen) and other limbic and cortical sites are said to play a crucial role in the neuro-modulation of behavioural activation in response to novelty and to signals of reward or loss of punishment. Individuals who are low in Novelty Seeking are less likely to seek extraordinary stimulation, and have greater endurance of monotony and aversive stimulation. Furthermore Clonninger (1994) is of the opinion that mesolimbic dopaminergic activity would be a crucial indication for motor habits and skills.

Individuals high in Reward Dependence form strong social attachment, are warmly sentimental, attempt to be pleasing and ingratiating, and have greater dependency needs. Their ambition for rewards in the form of power, wealth, food, affection or recognition tends to be insatiable in extreme cases so that they might be described as addicted or >dependent= on rewards (Clonninger, 1986).

It is postulated that individuals who are highly reward dependent are more strongly influenced by positive and negative reinforcement, i.e. they are generally more susceptible to conditioning that is contingent on reward or non-punishment. Hence it is postulated that fixedness in reward seeking behaviour is negatively correlated with variation in basal noradrenergic activity in the central reward system. In other words, low basal noradrenergic activity is associated with more intense responses to rewards and hence better maintenance (resistance to extinction) of rewarded behaviour (Clonninger, 1986).

Furthermore Reward Dependence is said to reflect individual differences in the brain=s system for modulation of conditioned signals of reward, particularly social signals. Clonninger (1994) hypothesizes that Reward Dependence reflects individual differences in postsynaptic sensitivity of neurons in the frontal cortex to noradrenergic projections from the locus ceruleus in the brainstem. High Reward Dependence is associated with low basal or resting levels of prefrontal activation, which is predicted to produce increased sensitivity to noradrenergically regulated orienting responses. Tempero-parietal activity is expected to be particularly crucial for cooperative

behaviour (an area which demonstrates hyper perfusion in trichotillomania). Coherence of activity in homologous parts of the two sides of the brain is also thought to be particularly crucial for the experience of creative and self-forgetful states associated with Self-Transcendence (Clonninger 1994 a). As will be discussed in the following section, the baseline SPECT indicated that a significant difference between the two hemispheres exists especially in the right superior parietal lobes. This could be a possible explanation for the low scores obtained on the Self Transcendence scale.

9.3 Results of the comparison of the difference between the right and left superior parietal area at baseline SPECT acquisition

Considering the average overall scores between the left and right parietal regions, a highly significant increase ^{99m}Tc-HMPAO count density was noted in the right superior parietal region as compared to the left superior parietal region ($p < 0.0003$), (Table 9.5).

The average global right hemisphere count density also showed a significantly increased count of ^{99m}Tc-HMPAO when compared to its left counterpart ($p<0,01$). This finding is more likely due to the inclusion of the parietal area. Although comparisons were made in the frontal lobe area as well as in the basal ganglia area, no significant difference was found in this study in these regions, which differs from

*f*MRI and PET studies where significant differences were indicated. This is probably due to the heightened sensitivity of PET and *f*MRI.

Table 9.5 **Results of the comparison of the average difference between the right and left superior parietal area at baseline SPECT acquisition**

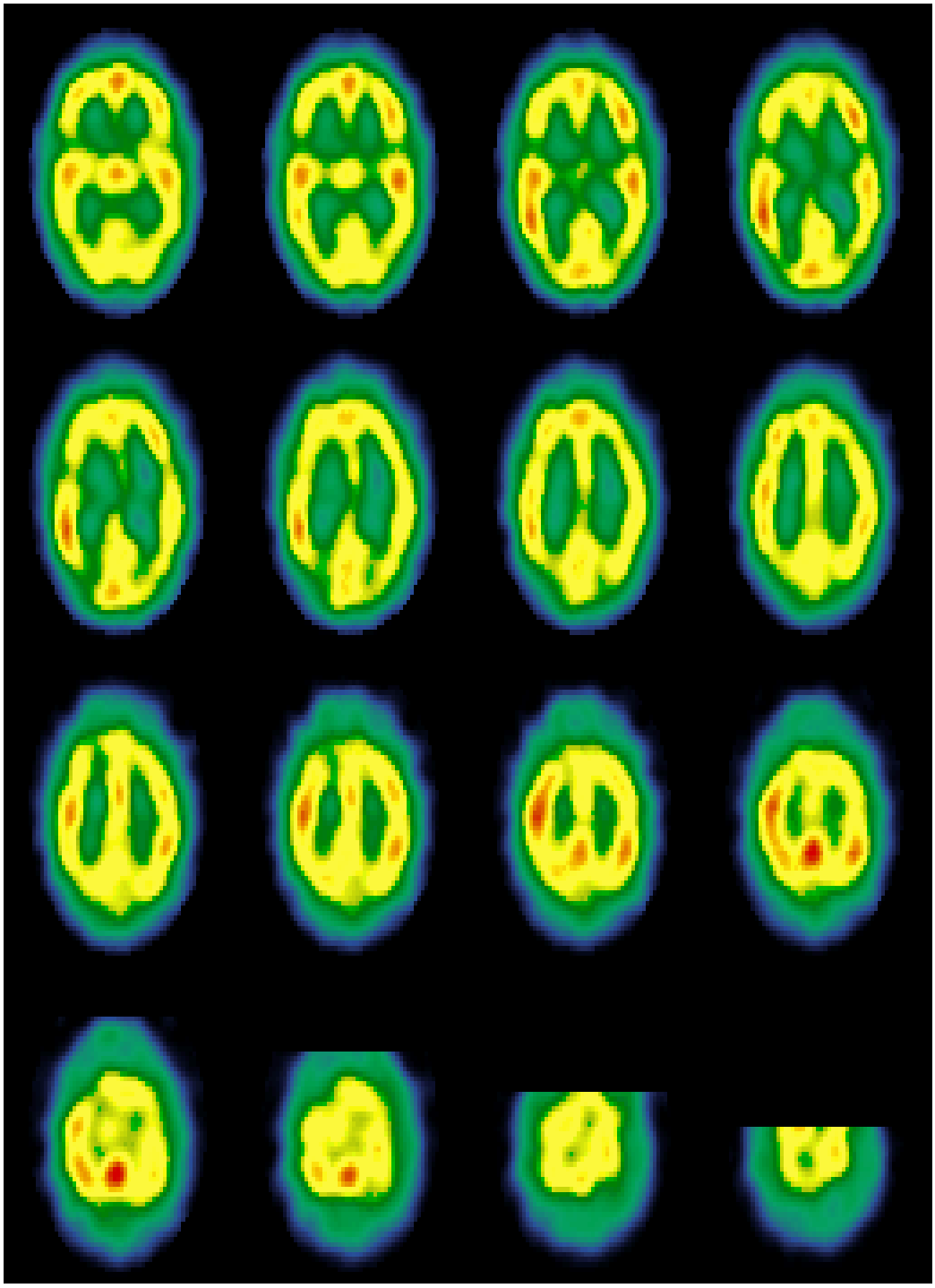
Subject Number	RIGHT Counts per Pixel*	LEFT Counts per Pixel	DIFFERENCE
1	3430	3175	255
2	3304	3146	158
3	3379	3303	76
4	3571	3373	198
5	3360	3271	89
6	3620	3523	97
7	3386	3270	116
8	3176	2941	235
9	3106	3126	-20
10	3131	3085	46
11	3463	3399	64
12	3444	3247	197
Student=s paired t - test : p = 0,0003			

Wilcox matched-pairs signed-ranks test : p = 0.0005 (one tailed)

*** A pixel is the number of released photo emissions in the Region Of Interest (ROI) detected by the Sodium Iodide crystal over a period of time, in this instance 20 minutes.**

Figure 9.2 gives an example of the visual compilation of the SPECT data at 16 different levels obtained from an individual subject. The hyperactivity is indicated by the red colour.

Figure 9.2 Regional ^{99m}Tc-HMPAO SPECT in trichotillomania demonstrating hyperperfusion in the right parietal region



9.4 Second analysis - ^{99m}Tc-HMPAO SPECT after Cognitive Behaviour Therapy

9.4.1 Comparison between the right and left hemisphere, before and after treatment

a. ***Group that responded to treatment***

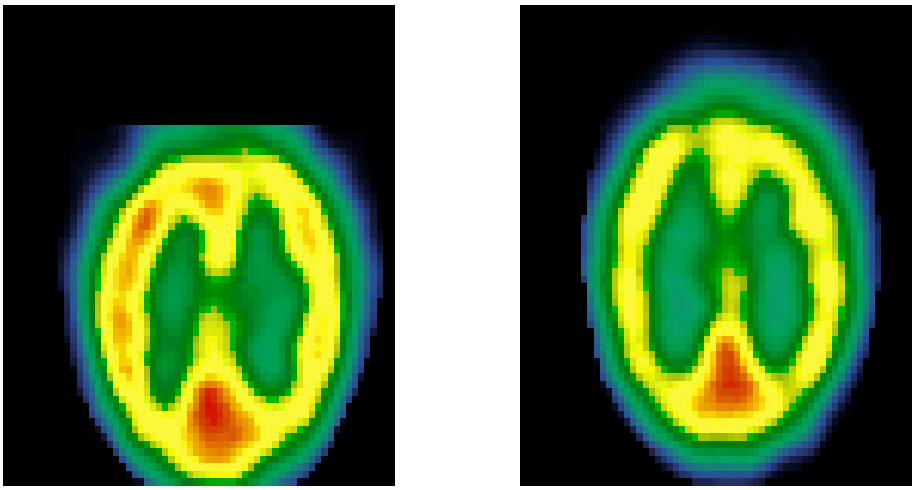
Subjects who were able to stop pulling their hair altogether as well as patients who decreased hair pulling behaviour substantially were considered as responders. Subjects who did not stop pulling at all were considered as non-responders. This observation was made by the researcher who was blind to the results of the SPECT scanning.

The results obtained from the ^{99m}Tc-HMPAO SPECT after the Cognitive Behaviour Therapy indicated a significant reduction in metabolic perfusion of the right superior parietal lobe as compared to the left (Table 9.6). Whereas the baseline SPECT data showed a significantly increased count of ^{99m}Tc-HMPAO SPECT in the right superior parietal region as compared to the left ($p< 0,001$), the pattern was equalised after CBT, such that there was no difference between the right and left superior parietal areas in those patients who responded to therapy.

Table9.6 **Second analysis. Group that responded. Comparison of the differences between the two hemispheres, using only one pair of ROI=s, before and after treatment.**

Before treatment					**After treatment**		
Subject nr	**Right Hemisphere * count per pixel**	**Left Hemisphere * count per pixel**	**Difference**		**Right Hemisphere * count per pixel**	**Left Hemisphere * count per pixel**	**Difference**
1	3430	3175	255		3408	3424	-16
2	3304	3146	158		3261	3254	7
3	3379	3303	76		3296	3287	9
4	3571	3373	198		3393	3361	32
5	3360	3271	89		3236	3230	6
6	3620	3523	97		3522	3497	25
7	3386	3270	116		2946	2967	-21
8	3176	2941	235		3007	3009	-2
9	3106	3126	-20		3022	3029	-7
Average difference : 133.8 T=average (sd/sqrt (n)) : 4.62 p<0.05					**Average difference : 3.7 T=average (sd/sqrt (n)) P=No Significance**		

An example of hyperperfusion (red) on the right and equalization on the left (red disappears) in one of the subjects who responded to treatment are presented in Figure 9.3.



(a) **(b)**

Figure 9.3 **Difference between regional uptake before (a) and after (b) Cognitive Behaviour Therapy in a responder**

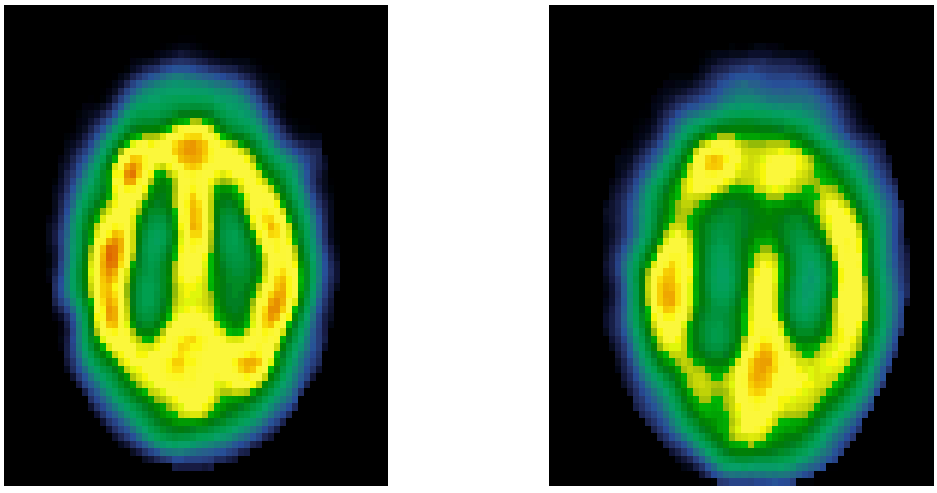
b) Group of non-responders

As can be seen from the Table 9.7 no significant difference between the first and second SPECT was achieved in the non-responding group.

Table 9.7 **Second analysis. Group that did not respond. Comparison of the differences between the two hemispheres, using only one pair of ROI=s, before and after treatment.**

Before treatment					**After treatment**		
Subject nr	**Right hemisphere**	**Left Hemisphere**	**Difference**		**Right hemisphere**	**Left hemisphere**	**Difference**
10	3131	3085	46		3295	3057	238
11	3463	3399	64		3604	3523	81
12	3444	3247	197		3138	2926	212
Average difference : 102.3 **T=average (sd/sqrt (n)) : 2.15** **p = No Difference**					**Average difference : 177.0** **T=average (sd/sqrt (n)) : 3.64** **p= No Difference**		

As can be seen from Figure 9.4 the hyperperfusion was not resolved in the region of interest after Cognitive Behaviour Therapy.



(a) **(b)**

Figure 9.4 **No significant difference in levels of hyperperfusion between hemispheres in non-responders, before (a) and after (b) Cognitive Behaviour Therapy**

The present study suggests that the increased count density of ^{99m}Tc-HMPAO in the right superior parietal area might be a state related disturbance in physiology in this disorder, as it resolves with successful treatment.

9.4.2 Comparison of the same hemisphere before and after treatment in responders

When comparing the difference between the right hemisphere before treatment to the right hemisphere after treatment a significant difference ($p=0.05$) was indicated in the patients that responded (Table 9.8). No difference was demonstrated in the non-responders. No significant difference was indicating comparing the left hemisphere before treatment to the left hemisphere after treatment in responders as was expected since this area does not seem to be involved in trichotillomania.

Table 9.8 **Second analysis. Comparison of relative counts in the same hemisphere before treatment with those in the same hemisphere after treatment - Group that responded**

BEFORE TREATMENT				AFTER TREATMENT		
Subject nr.	Right	Right	Difference	Left	Left	] Difference
1	3430	3408	22	3175	3424	-249
2	3304	3261	43	3146	3254	-108
3	3379	3296	83	3303	3287	16
4	3571	3393	178	3373	3361	12
5	3360	3236	124	3271	3230	41
6	3620	3522	98	3523	3497	26
7	3386	2946	440	3270	2967	303
8	3176	3007	169	2941	3009	-68
9	3106	3022	84	3126	3029	97
Average difference : 137.9				Average difference : 7.8		
t =average (sd/sqrt (n)) : 3.32				t = average (sd/sqrt (n)) : 0.15		
p > 0.05				p = No Significance		

9.5 Comparison of total scores before and after Cognitive Behaviour Therapy on the Psychiatric Institute Trichotillomania Scale (PITS)

As far as the Pennsylvania Institute Trichotillomania Scale is concerned (Table 9.9) a highly significant difference between the Total score before and after treatment was indicated (p=0,0004). Eleven subjects (91,66%) indicated improvement of symptoms on the PITS. The one subject (8,33%) that did not show any improvement did not complete the therapy due to drug abuse.

However, scrutinizing the data at a closer level produces many questions. For example, clinically it was found that participant number 4 made significant progress, as was also demonstrated on her second scan. However, according to the PITS, almost no difference before (29) and after (27) therapy occurred. A possible explanation for this type of discrepancy is posed by Rothbaum, Opdyke and Keuthen (1999), whose comments on the PITS are: *>Unfortunately, the anchors for some items can be somewhat arbitrary; for example, one non-scalp site is rated as less severe than one scalp site=*. This implies that if somebody pulls out all of their eye lashes, she scores 1 point on the Sites subscale whereas if a person pulls out half or more of her scalp hair (as long as it is in one place), she scores two points. Similarly, two people can have the same score where one person pulls out 50% of the hair on her head and another pulls out 50% of her eyelashes. Severity of symptoms should not really be compared in this way.

Table 9.9 **Comparison of total scores before and after Cognitive Behaviour Therapy on the Psychiatric Institute Trichotillomania Scale (PITS)**

Nr	Sites		Severity		Duration		Resistance		Interference		Distress		TOTAL	
	B*	A*	B	A	B	A	B	A	B	A	B	A	B	A
1	6	0	5	0	4	0	6	1	3	0	7	1	31	2
2	6	0	4	0	3	0	6	0	2	0	3	1	24	1
3	2	0	6	0	5	0	6	1	2	0	5	2	26	3
4	5	5	5	5	5	4	6	5	3	3	5	5	29	27
5	6	3	6	2	6	3	6	5	0	0	4	3	28	16
6	5	0	6	0	4	0	6	2	1	0	4	3	26	5
7	3	0	5	1	2	1	7	2	1	0	4	2	22	6
8	4	2	3	1	4	1	5	2	1	0	4	2	21	8
9	6	7	3	4	6	7	6	6	0	1	6	4	27	29
10	7	4	4	2	7	2	7	4	3	3	6	6	34	21
11	2	2	4	4	4	1	6	4	0	0	4	1	20	12
12	6	2	4	3	3	3	5	6	3	2	4	4	25	20
Student=s paired t-test : p = 0,0004 Wicoxon's matched pairs signed rank test : p = 0.0002 (one tailed)														

***B = Before** ***A = After**

No significant correlation between symptom severity as concluded from this scale and hyper-perfusion as demonstrated by the SPECT was found. However a trend was observed. The non-correlation was probably due to two outliers. One outlier was from

the subject who developed major depression, while the other (who did not finish treatment), started abusing drugs. Swedo et al., (1991) describe a similar pattern of non-comparability between severity and perfusion of four out of six sites in their study. The two sites which they found did correlate using PET, were the cerebellum and the caudate nucleus (both of which did not demonstrate significant changes in the present study).

9.6 Comparison of total scores before and after Cognitive Behaviour Therapy on the Hamilton Anxiety Rating Scale

When comparing the Hamilton Anxiety Scale scores (Table 9.10) before treatment to those after treatment a significant difference was detected (p=0,0001). As addressing anxiety is part of the cognitive behaviour programme, this was expected. A decrease of anxiety was reported by all the subjects.

Comparing the level of anxiety as measured by this scale to metabolic hyper-perfusion on the first SPECT scans did not indicate significant correlation (p=0,2696). Since no correlation between anxiety and symptom severity as indicated by the SPECT scan was found prior to treatment, calculating for statistical correlation between the second scale and the second SPECT scan was not undertaken. A similar result was described by Swedo et al. (1991) using the Spielberger Anxiety Rating Scale. These researchers postulated that their finding might be due to the self rating nature of the scale as well as the small sample size. Further research is needed.

Table 9.10 Comparison of anxiety rating before and after Cognitive Behaviour Therapy

Subject number	Before	After
1	37	9
2	23	3
3	18	7
4	24	22
5	29	18
6	18	9
7	19	8
8	17	7
9	31	28
10	48	33
11	29	6
12	34	22
Student=s paired t-test - p=0.0001 ; Wilcoxon=s matched pairs signed rank test p= 0.0002 (0ne tailed)		

CHAPTER 10

DISCUSSION

Traditionally diagnoses of mental disorders are based on symptom clusters rather that underlying brain function. The chances are that if patients experience problems with their feelings such as anxiety or depression, with their thoughts, such as with intrusive thoughts in OCD, or with their behaviour, such as self mutilation or Trichotillomania, a brain scan would not be ordered, thus it would not be known whether any areas or neuro-circuits of the patient's brain are not working or are hyper or hypoactive. Using functional imaging such as SPECT for identifying as well as following the course of treatment and tracking changes in brain systems can lead to more effective management of the patient, utilization of resources (currently one brain scan is more cost effective that two months of psychotherapy) and de-stigmatization of mental disorders. Since the field of functional brain imagery is still relatively new as well as under-utilized especially in the field of psychotherapy, the results of this study contributes to the growing body of scientific literature in the field of mental disorders in general and in Trichotillomania specifically.

As far as could be determined this is the only study using SPECT to investigate functional localization in trichotillomania. However, similar patterns of cerebral blood flow in trichotillomanic patients were found as demonstrated by Swedo et al., (1991), using PET. As was already mentioned these researchers demonstrated global cerebral glucose hypermetabolism and increased normalized regional metabolic rates in the right superior parietal region and the right and the left cerebella. As with the present study these authors did not find significant differences in the frontal lobe areas. Furthermore, the results of the present study are also in keeping with the subsequent

findings of Grachev et al., (1997), who demonstrated enlarged right cuneal volume as well as significantly reduced left inferior frontal gyrus volumes in comparison to normal controls.

The parietal lobes, which are strategically situated between the frontal, occipital and temporal lobes, are closely related in function to each of these regions of the brain (Walsh, 1994). Partly as a result of this, a greater variety of clinical manifestations is likely to result from disease of the parietal lobe than from disturbance of any other part of the hemispheres. According to Grachev et al., (1997), the physiology of the cuneal cortex as a part of the visual association cortex (i.e. the peristriate cortex ; Brodmann=s areas 18 and 19) and its roles in behaviour is not completely understood. The association areas act as obligatory relays for the intercortical transfer of sensory information from primary areas towards other parts of the neocortex. PET techniques have shown that exposure to white light increases the metabolic activity only of the primary visual cortex, whereas presentation of a more complex visual scene led to additional metabolic activation of the peristriate cortex (Phelps, 1981; Petersen, Fox, Snyder and Raichle, 1990; Corbetta, Niezen, Dobmeyer, Snyman and Peterson, 1991) Neurons in the peristriate cortex have firing properties similar to those of neurons in the primary visual cortex. However, these neurons show regional specialization for analyzing more complex aspects of visual information such as motion and form (Petersen et al., 1990; Van Essen and Maunsell, 1983) and are apparently involved in more complex aspects of visual information processing and movement analysis, which interact with other components of mental content, including the individuals' past experience (Damasio and Damasio, 1983; Damasio, Tranel and Damasio, 1990).

Lesions of the peristriate induce exceptionally specific disturbances of movement perception or visual dysfunction and may lead to the interruption in the relay of visual input to the parietal and frontal heteromodal areas, causing visual -somatosensory and visual motor disconnections (Mesulam, 1985; Lockwood, Moulton, Picon-Niero, Vanderloeg and Ochipa., 1996). Grachev et al., (1997) put forward the hypothesis that enlargement of the right cuneal cortex in trichotillomania, may reflect the complex interactions between the visual and sensorimotor cortices when the relay mechanism is not working in the normal way and significantly more neuronal stimuli are able to get to the targets generating the repetitive or compulsive behaviour seen in trichotillomania.

Furthermore, it is well known that the right hemisphere is dominant in determining the spatial distribution of attention, for the extra personal space, for the paralinguistic aspects of communication, for the expression of emotions and for affective behaviour (Mesulam, 1985; Grady, 1996; Dweyer et al., 1981, Delis et al, 1983 Brumbach, Staton and Wilson, 1984; Posner and Petersen, 1990). However, the neuroanatomical basis of the asymmetrical brain organization and its role in behaviour, including trichotillomania, has not been well established yet.

Of substantial importance is the fact that the current study demonstrates a neurophysiological substrate and impact of psychotherapy. The results as indicated by the difference in SPECT data, as well as score reductions on symptom scales, before and after treatment suggest that Cognitive Behaviour Therapy is effective in the treatment of trichotillomania. Similar results indicating that Cognitive Behaviour

Therapy impacts on neurophysiological functions has also been demonstrated in an OCD population (Baxter et al., 1992; and Schwartz et al., 1996).The studies completed by Baxter et al., (1992) and Schwartz et. al., (1996) compared the effect of CBT versus serotonin re-uptake inhibitors on brain function in OCD and concluded that in both instances systematic changes in cerebral glucose metabolic rate were obtained and that a prefrontal cortico-striato-thalamic brain system is implicated in mediation of symptoms of OCD. These authors state that the >serotonin hypothesis= of OCD rests most firmly on evidence that chemically diverse drugs that are strong serotonin re-uptake inhibitors are effective in the treatment of OCD, while similar agents that affect other neurotransmitters are ineffective.

As a possible explanation for the reason why behaviour therapy could produce brain function changes similar to neurochemically specific drugs in OCD, Baxter et al., (1992) postulates that serotonin might be responsible. Even in lower animals such as the sea slug *Aplysia* it is changes at synapses that use serotonin that seems to mediate learned changes in stimulus-response behaviour. Further direct application of serotonin at these synapses can produce the same lasting changes in synaptic function and behaviour as seen with behaviour modification in *Aplysia* (Montarolo et al., 1986 ; Kandel, 1989).

There has been increased recent interest in examining the ways in which individual differences in behaviour can be tied to individual differences in brain structure and function (Berenbaum, 1996; Cummings 1993). Currently cortico-striatal pathways have been implicated in procedural learning and attention, and some authors consider

that the obsessive-compulsive spectrum of disorders represent an impairment in the neural mechanisms underlying procedural learning (Rauch, Savage and Brown, 1995) as well as declarative learning (March, et al., 2004).

Electron microscopy studies confirm the strong links between the inferior prefrontal cortex and nucleus accumbens (Haber et al., 1995), between the cuneal cortex (anterior-parietal area) and inferior prefrontal cortex through the system of indirect parietal and temporal cortex pathways. This is consistent with the finding of Grachev (1997), who suggests that the enlargement of the right cuneal cortex in trichotillomania may reflect the complex interactions between the visual and sensorimotor cortices when the relay mechanism is not working in the normal manner and significantly more neuronal stimuli are able to get to their targets, generating the repetitive or compulsive behaviour seen in trichotillomania. The projections from the inferior prefrontal cortex also terminate within the dorso lateral head of the caudate nucleus, which has been shown to be compromised in OCD (Saxena et al., 1998). Similarly the tics in Tourette syndrome seem to be the product of core disturbances in the structure and function of the striatum that predispose an individual to impairments in learning and to the expression of fragmented motor and vocal behaviours. These predispositions to the tic behaviour may be released from regulatory influences of the prefrontal cortex (March et al., 2004)

Schwartz (1998), drawing from the extensive literature on the behavioural neurobiology of the basal ganglia, proposed a reason why Cognitive Behavioural Treatment is effective in OCD. According to Schwartz (1998), given that this

extensive data demonstrating that the basal ganglia are involved in the functional modulation of cortical circuitry which is involved in the expression of previously learned behavioural arrays, and the apparent involvement of the circuitry in the expression of OCD symptoms, it seems reasonable to teach patients with OCD a technique intended to activate alternative circuits within the vast array of circuitry contained in the cortico-striate system. Schwartz (1998) further states that if OCD symptoms are related to a malfunction in cortical circuit activation and, in particular, to an >error-detection= mechanism in the orbital cortex, then activation of an alternative circuit through the focused performance of a familiar alternative behaviour might over time ameliorate the discomfort related to the faulty brain mechanism.

As has been discussed in previous chapters, trichotillomania, OCD as well as Tourette syndrome can be conceptualized in terms of the obsessive-compulsive spectrum of disorders. It is understood that whereas the symptomatology, neurophysiology and intervention strategies overlap, specific differences between these conditions are also present. However, although it appears that the basal ganglia and the striatum are possibly involved in all of these pathologies, dysfunction in different parts of the striatum has been suggested for each of the illnesses. Recent findings furthermore suggest that the heterogeneous symptoms of OCD might each have its underpinning in a different part of the striatum (Mataix-Cols, et al., 2004). Considering the heterogeneous nature of trichotillomania it is postulated that, as in OCD, closely related but different parts of the striatum may be involved in trichotillomania. This needs further investigation.

In the light of the above it is thus highly probable that the reasons for Cognitive Behaviour Therapy being successful in the treatment of trichotillomania are similar to that of the other obsessive-compulsive spectrum of disorders. Similarly, teaching the trichotillomanic patient alternative behaviour, such as awareness and habit reversal techniques, might over time activate alternative circuitry, which might lead to a reduction in the pulling behaviour.

CHAPTER 11

CONCLUSIONS AND RECOMMENDATIONS

11.1 Conclusion and hypothesis testing

As far as the first part of the hypothesis of this study is concerned a highly significant increase in ^{99m}TC- HMPAO count density was indicated in the right superior parietal region as compared to the same region in the left parietal area in this sample of trichotillomania sufferers. As far as can be determined this is the only study that has used SPECT as a means of functional imaging in trichotillomania. One study utilizing PET has described similar results prior to this study. Subsequently similar results using *f*MRI were published. In the present study, SPECT did not indicate cerebellar involvement as was described in the PET study or reduced activity in the inferior frontal lobes as described by the *f*MRI study. This discrepancy is most probably due to lowered resolution and sensitivity of the SPECT technique.

As far as the second part of the of the hypothesis is concerned, this study demonstrated that by making use of Cognitive Behaviour Therapy improvements in symptoms can be achieved. Significant reduction in symptom severity was indicated on the Hamilton Anxiety Rating Scale as well as the on the Psychiatric Institute Trichotillomania Scale. Furthermore the results of this study demonstrated a neurophysiological substrate and impact of psychotherapy since significant changes in right superior parietal metabolic perfusion after effective Cognitive Behaviour Therapy in trichotillomania was demonstrated. These changes were not seen in patients who did not respond to treatment. Whilst this study did not compare Cognitive Behaviour Therapy to pharmacotherapy, this finding adds to the growing body of evidence that Cognitive Behaviour Therapy is an effective intervention and might be useful in patients who suffer from drug intolerance or who are non compliant with medication. Drawing on comparisons between other obsessive-compulsive spectrum disorders, the observed neurophysiological changes brought about by Cognitive Behaviour Therapy could possibly be ascribed to new learning and thus to activation of different striatal pathways as was described in chapters 4 and 10.

A statistically significant reduction of symptom severity was indicated after Cognitive Behaviour Therapy on the Psychiatric Institute Trichotillomania Scale. However using this scale to rate symptom severity proved problematic since this scale does not distinguish sufficiently between different hair pulling behaviour as , for example, pulling one hair from the eyelashes scores the same as pulling from one site on the

scalp (which could mean pulling many of hairs). Although a trend was observed, no significant correlation between symptom severity as indicated by this scale and hyper-perfusion as indicated by SPECT was found possibly due to the small sample size.

As far as the Temperament and Character Inventory is concerned, the present study found that people suffering from trichotillomania obtained their highest scores on the Character Inventory part of the scale on the Cooperative scale and their lowest score on the Self Transcendence scale. On the Temperament part of the scale patients scored highest on the Reward Dependence scale and lowest on the Novelty Seeking scale. As was described in chapters 9 and 11 people with this type of profile might be described as addicted to rewards and might have a greater endurance toward monotony and aversive stimulation. Differences between the present study and that of Lochner et al, (2005) are probably due to the small sample size in the current study.

As with the Psychiatric Institute Trichotillomania Scale a significant reduction in scores was found after Cognitive Behaviour Therapy on the Hamilton Anxiety Rating Scale in this study. This was expected as relaxation training is part of the programme. No significant correlation was found between the baseline SPECT and symptom severity as measured on this scale.

The third part of the hypothesis was confirmed, with results obtained from the ^{99m}Tc-HMPAO SPECT after the Cognitive Behaviour Therapy indicating a significant reduction in metabolic perfusion of the right superior parietal lobe as compared to the same area on the left in the responder group. The present study suggests that the

increased count density of ^{99m}Tc-HMPAO SPECT in this area might be a state related disturbance in physiology in this disorder as it resolves with successful treatment.

11.2 Weaknesses of the study and future research

Although significant results were obtained as far as the SPECT components of the study are concerned, the small sample size might have been a weakness in terms of comparisons between severity of symptoms as indicated by the rating scales and the hyper perfusion as indicated by SPECT. As a trend of correlation was observed between the Psychiatric Institute Trichotillomania Scale and the SPECT further investigations using larger sample sizes might be useful.

Using the Psychiatric Institute Trichotillomania scale further weakened the present study due to it=s unsophisticated nature. For future research it would be recommended that this scale is refined or another scale is drawn up to accommodate the heterogeneous nature of trichotillomania.

It is further recommended that functional imaging techniques are incorporated to determine whether neuroanatomical differences exist between the different symptoms of trichotillomania. Since recent research making use of refined imaging techniques have indicated neuroanatomical differences in other obsessive-compulsive spectrum of disorders this might well be the case in trichotillomania.

As far as the author is aware, no functional imaging studies of males suffering from trichotillomania exist and thus need investigation.

As faulty learning patterns might be involved in trichotillomania and the other obsessive-compulsive spectrum of disorders, it might be worthwhile to determine whether neuro-imaging patterns in early onset trichotillomania differ from that of later onset.

Replication of the neuroimaging part of the study using larger sample sizes comparing hyper-perfusion patterns with different anxiety rating scales are needed to determine whether the non-correlation of the present study as well as that of previous research are due to the nature of the specific rating scales or whether the assumed role of anxiety in trichotillomania needs to be reinvestigated.

Finally, although the >trancelike= state during hair pulling episodes was noted in the present study and briefly mentioned in previous literature, no in-depth investigation has been attempted but would contribute to the understanding of trichotillomania.

REFERENCES

Abramson, L.Y**.,** Seligman, M.E.P. and Teasdale, J.D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology.* **87** : 49-74.

Adam, B.S. and Kashani, J.H. (1990). Trichotillomania in children and adolescents: review of the literature and case report. *Child Psychiatry and Human Development.* **20** (3) : 159-168.

Adler, A. (1958). What life should mean to you. New York : Capricorn

Alexander, G.E., Crutcher, M.D. and De Long, M.R. (1990). Basal ganglia-thalamo-cortical circuits : Parallel substrates for motor, oculomotor, “prefrontal” and “limbic” functions. *Progressive Brain Research.* **85** : 119-146.

Alexander, G.I., De Long, M.R. and Strick, P.L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*. **9** : 357-381.

Allen, G.I. and Tsukahara, N. (1974). Cerebro-cerebellar communication systems. *Physiology Review*. **54** : 769-775.

American Psychiatric Association . (1994). Diagnostic and Statistical Manual of Mental Disorders, 4th Edition. Washington, DC: American Psychiatric Association.

Anderson, F.N.and Dean, H.C. (1956). Some aspects of child guidance clinic intake policy and practices : a study of 500 cases at the Los Angeles Guidance Clinic. *Public Health Monograms.* **42** : 1-16.

Azrin, N.H. and Nunn, R.G. (1973a). Habit reversal : A method of eliminating nervous habits and tics. *Behavior Research Therapy*. **11** : 619-628.

Azrin, N.H. and Nunn, R.G. (1977). Habit control in a day. New York: Simon and Schuster.

Azrin, N.H., Nunn, R.G. and Frantz, S.E. (1980). Treatment of hair pulling (trichotillomania): A comparative study of habit reversal and negative practice training. *Journal of Behavior Therapy and Experimental Psychiatry*. **11** : 13-20.

Barahal, H.S. (1940). Psychopathology of hair-plucking. (Trichotillomania). *Psychoanalytic Review*. **27** : 291-310.

Baxter, L.R., (1991). PET studies of cerebral function in major depression and OCD: the emerging prefrontal cortex consensus. *Ann. Clinical Psychiatry*. **3** : 103-109

Baxter, L.R., Schwartz, J.M., Bergman, K.S., Szuba, M.P., Guze, B.H., Mazziotta, J.C., Alazraki, A., Selin, C.E., Ferng, H.K., Munford, P. and Phelps, M.E. (1992). Caudate glucose metabolic rate changes with both drug and behavior therapy for OCD. *Archives of General Psychiatry*. **49** : 681-689.

Bayer, C.A. (1972). Self monitoring and mild aversion treatment of trichotillomania. *Journal of Behaviour Therapy and Experimental Psychiatry*. **3** :139-141.

Beck, A.T. (1978). Depression inventory. PA : Center for Cognitive Therapy.

Beck, A.T. and Emery, G. (1985). Anxiety disorders and phobias : A cognitive perspective. New York : Basic Books.

Beck, A.T., Epstein, H. and Harrison, R. (1983). Cognitions, attitudes and personality dimensions in depression. *British Journal of Psychotherapy,* **1:** 1-16.

Beck, A.T., Rush, J., Shaw, B.F. and Emery, G. (1979). Cognitive therapy of depression. New York : Guilford Press.

Benton, A.L. (1974). The visual retention test (4th ed.). New York : The Psychological Corporation.

Benton, A.L. and Hamsher, K. (1976). Multilingual aphasic examination. Iowa City : University of Iowa.

Benton, A.L., Hamsher, K., Varnay, N.R. and Spreen, O. (1983). Contributions to neuropsychological assessment. New York : Oxford University Press.

Berenbaum, S. (1996). Are variations in cognitive abilities related to variations in brain organization? *Journal of the International Neuropsychology Society.* **2** : A67

Berg, C. (1936). Unconscious significance of hair. *International Journal of Psychoanalysis.* **17** : 73-78.

Bern, E. (1961). Transactional analysis in psychotherapy. : A systematic individual and social psychiatry. New York : Grove Press.

Bern, E. (1964). Games people play. New York : Grove Press.

Bernard, M.E., Kratochwill, T.R. and Keefauver, L.W. (1983). The effects of rational-emotive therapy and self instructional training of chronic hair-pulling. *Cognitive Therapy Research.* **7** : 273-280.

Binswanger, L. (1958). In : A new dimension in psychology and psychiatry. (eds. R. May, E. Angel and H.F. Ellenberger). New York : Basic Books.

Black, D.W. and Blum, N. (1992). Trichotillomania treated with clomipramine and a topical steroid. *American Journal of Psychiatry.* **149** : 842-843.

Blackburn, I. and Twaddle, V. (1996). Cognitive therapy in action. London : Souvenir Press (Educational & Academic)Ltd.

Blount, R.L. and Finch, A.J. Jr. (1987). Reducing trichotillomania in a three year old girl. *Child and family behavioral therapy.* **9** : 65-72.

Bordnick, P.S., Thyer, B.A. and Ritchie, B.W. (1994). Feather picking disorders and trichotillomania : an avian model of human psychopathology. *Journal of Behaviour Therapy and Experimental Psychiatry.* **25(3)** : 189 - 196.

Bornstein, P.H. and Rychtarik, R.G. (1978). Multi-component behavioral treatment of trichotillomania : a case study. *Behaviour Research and Therapy*. **16** : 217-220.

Bowers, H.S. (1973). Situationism in psychology : An analysis and critique. *Psychological Review,* **80** : 307-336.

Brigandt, I. (2005).The instincr concept of the early Konrad Lorenz. *Journal of the History of Biology*. **38 (3)** :571.

Brumback, R.A., Staton, R.D. and Wilson, H. (1984). Right cerebral hemispheric dysfunction. *Archives of Neurology*. **41** : 248-249.

Burns, D.D. (1990). The Feeling Good Handbook. New York : William Morrow and Company.

Butters, N., Soeldner, C. and Fedio, P. (1972). Comparison of parietal and frontal lobe spatial deficits in man: extra personal vs. personal (egocentric) space. *Perceptual and Motor Skills.* **34** : 27-34.

Cautela, J.R. (1967). Covert sensitization. *Psychological Reports.* **20** : 459-468.

Christenson, G.A. (1995). Trichotillomania - from prevalence to comorbidity. *Psychiatric Times.* **12 (9)** : 44 – 48.

Christenson, G.A., Chernoff-Clementz, E. And Clementz, B.A. (1992a). Personality and clinical characteristics in patients with trichotillomania. *Journal of Clinical Psychiatry*. **53 (11)** : 407-413.

Christenson, G.A. and Crow, S.J. (1996). The characterization and treatment of trichotillomania. *Journal of Clinical Psychiatry*. **57 (suppl. 8)** : 42-49.

Christenson, G.A., Mackenzie, T.B., Mitchell, J.E. and Callies, A.L. (1991b). A placebo controlled double-blind cross-over study of fluoxetine in trichotillomania. *American Journal of Psychiatry*. **148** : 1566-1571.

Christenson, G.A., MacKenzie, T.B. and Reeve E. (1992b). Familial trichotillomania. [Letter]. *American Journal of Psychiatry*. **149** : 283.

Christenson, G.A., MacKenzie, T.B. and Mitchell, J.E., (1991a). Characteristics of 60 adult chronic hair pullers. *American Journal of Psychiatry*. **148** : 365 - 370.

Christenson, G.A., Popkin, M.K., Mackenzie, T.B. and Realmuto, G.M. (1991c). Lithium treatment of chronic hair-pulling. *Journal of Clinical Psychiatry*. **52** : 116-120.

Christenson, G.A., Pyle, R.L. and Mitchell, J.E. (1991d). Estimated life prevalence of trichotillomania in college students. *Journal of Clinical Psychiatry*. **52** : 415-417.

Christenson, G.A., Ritzvedt, S.L. and Mackenzie, T.B. (1993). Identification of trichotillomania cue profiles. *Behaviour Research and Therapy*. **31** : 315-320.

Clonninger, C.R. (1986). A unified biosocial theory of personality and its role in the development of anxiety states. *Psychiatric Developments.* **3** : 167-226.

Clonninger, C.R. (1987). A systematic method of clinical description and classification of personality variants. A proposal. *Archives of General Psychiatry.* **44** : 573-588.

Clonninger, C.R. (1988). A unified biosocial theory of personality and its role in the development of anxiety states. *Psychiatric Developments.* **2** : 83-120.

Clonninger, C.R. (1994). Temperament and personality. *Current Opinions in Neurobiology*. **4** : 266-273.

Clonninger, C.R, Przybeck, T.R. and Svrakic, D.M.(1991). The tridimentional personality questionnaire: US normative data. *Psychological Reports.* **69** :1047-1057.

Clonninger, C.R., Przybeck, T.R., Svrakic, D.M. and Wetzel, R.D. (1994). The Temperament and Character Inventory. A guide to Its Development and Use. Washington University press. Missouri.

Cooper, S.J. and Dourish, C.T. (eds). (1990). Neurobiology of stereotyped behaviour. Oxford University Press. New York.

Corbetta, M., Miezen, F.M., Dobmeyer, S., Shulman, G.L. and Petersen, S.E., (1991). Selective and divided attention during visual discrimination of shape, color and speed. Functional anatomy by positron emission tomography. *Journal of Neuroscience.* **11** : 2383-2402.

Cordle, J, and Long, C. (1980). The use of operant self-control procedures in the treatment of compulsive hair-pulling. *Journal of Behaviour Therapy and Experimental Psychiatry.* **11** : 127-130.

Corsini, R.J. and Wedding, D, (1989). Current psychotherapies. Illinois : F.E. Peacock Publishers Inc.

Cummings, J.L.(1993). Frontal-subcortical circuits and human behaviour. *Archives of neurology.* **50** : 873-880.

Damasio, A.R. and Damasio, H. (1983). Localization of lesions in achromatopsia and prosopagnosia. In : A. Kertesz (ed.). *Localization in Neuropsychology.* Academic Press: New York, 417-428.

Damasio, A.R., Tranel, D., Damasio, H. (1990). Individuals with sociopathic behaviour caused by frontal damage fail to respond autonomically to social stimuli. *Behavioural Brain Research.* **41** : 81-94.

Davis, C.S. (1991). Parrot psychology and behavior problems. *Veterinary Clinics of North America Small Animal Practices.* **21** : 1281-1288.

Davis, M., Robbins-Eschelman, E. and McKay, M. (1995).The relaxation and stress reduction workbook. (4th ed.). New Harbinger Publication Inc.: California.

Dawber, R. (1985). Self-induced hair loss. *Seminar for Dermatology*. **4** : 53-57.

Dean, J.T., Nelson, E. and Moss, L. (1992). Pathologic hair-pulling : A review of the literature and case reports. *Comprehensive Psychiatry*. **33** (2) : 84-91.

DeFilippis, N.A. and McCampbell, E. (1979). The booklet category test. Psychological Assessment Resources Inc. : Odessa F.

Delis, D.C., Kramer, J.H., Kaplan, E. and Ober, B.A. (1983). California verbal learning test form II. The Psychological Corporation : San Antonio, Texas.

De Long, M.R. and Georgopoulos, A.P. (1981). In G.E. Alexander, M.R. De Long and P.L. Strick. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*. **9** : 357-381.

Devinski, O. (1992). Behavioral Neurology. London : Edward Arnold.

Devous, M.D. (1992). Comparison of SPECT applications in neurology and psychiatry. *Journal of Clinical Psychiatry.* **53 (11: suppl)** : 13-19.

Displacement activity. (2006). In *Encyclopaedia Britannica.* Retrieved September 11, 2006 from Encyclopaedia Britannica Premium Service: *http://www.britannica.com/eb/article-9030642*

Dodman, N.H.(1994). Pharmacological treatment of behaviour problems in cats. *Veterinary International.* **4** : 13-20.

Dwyer, J.H. and Rinn, W.E. (1981). The role of the right hemisphere in contextual inference. *Neuropsychology.* **19** : 479-482.

D=Zurilla, T.J. and Goldfried, M.R. (1971). Problem solving and behavior modification. *Journal of Abnormal Psychology.* **78** : 107-126.

Elkin, I., Shea, M.T., Watkins, J.T., Imber, S.D., Sotsky, S.M., Collins, J.F., Glass, D.R., Pilkonis, P.A., Leber, W.R., Docherty, J.P., Fiester, S.J. and Parloff, M.B. (1989). National institute of mental health treatment of depression collaborative research program: general effectiveness of treatments. *Archives of General Psychiatry*. **46** : 971-982.

Ellis, A. (1973). Humanistic psychotherapy : The rational-emotive approach. New York : Mc Graw-Hill.

Ellis, A. (1977). The basic clinical theory of rational-emotive therapy. In : Handbook of Rational-emotive therapy. (eds. A. Ellis and R. Grieger). New York : Springer.

Encyclopaedia Britannica (2006). See 'displacement activity'

Evers, R.A.F. and Van De Wettering, B.J.M. (1994). A treatment model for motor tics based on a specific tension reduction technique. *Journal of Behaviour Therapy and Experimental Psychiatry.* **25(3)** : 255-260.

Everts, E.V. and Thatch, W.T. (1969). Motor mechanism of the CNS : cerebro-cerebellar interrelations. *Annual Review of Physiology*. **31** : 451-498.

Ezra 9:3. The Holy Bible. (1984). International Version. Hodder and Stroughton. (6thEd.). International Bible Society. New York. 49.

Fabri, R. and Dye, A.J. (1974). Hypnotic treatment of trichotillomania. *International Journal of Clinical and Experimental Hypnosis.* **22** : 210-215.

Feldman, M.J. and Drasgow, J.A. (1951). A visual-verbal test for schizophrenia. *Psychiatry Quarterly*. **25 (suppl)** : 55-64.

Flowers, K.A. and Robertson, C. (1985). The effect of Parkinson=s disease on the ability to maintain a mental set. *Journal of Neurology, Neurosurgery and Psychiatry.* **48** : 517-529.

Frank. J. (1961). Persuation and healing. Baltimore : John Hopkins Press.

Freeman, A. (1983). Cognitive therapy : an overview. In : Cognitive therapy with couples and groups. (ed. A. Freeman). New York : Plenium.

Freeman, A. (1990). Cognitive therapy. In : Handbook of comparative treatments for adult disorders. (eds. A.S. Bellack and M.Hersen). New York : Wiley.

Freud, S. (1953/1991). The interpretation of dreams. London : Penguin Books.

Friman, P.C., Finney, J.W. and Christophersen, E.R. (1984). Behavioral treatment of trichotillomania: An evaluative review. *Behaviour Therapy*. **15** : 249-265.

Galski, T. (1983). Hair pulling. (Trichotillomania). *Psychoanalytic Review*. **70 (3)** : 331-346.

Gardner, G.C. (1978). Hypnotherapy in the management of childhood diseases. *Journal of Pediatrics.* **92** : 838-849.

Gluhoski,V.L. (1995). A cognitive approach for treating trichotillomania. *Journal of Psychotherapy and Practice and Research.* **4** : 277-285.

Goldfried, M.R. and Davison, G.C. (1976). Clinical behaviour therapy. New York : Holt, Rinehart and Winston.

Graber, J. and Arndt, W.B. (1993). Trichotillomania. *Comprehensive Psychiatry.* **34 (5)** 340-346.

Grachev, I.D. (1997). MRI- based morphogenic topographic parcellation of human neocortex in trichotillomania. *Psychiatry and Clinical Neurosciences.* **51** : 315-324.

Grady, C.L. (1996). The roles of medial temporal and prefrontal cortex in encoding and recognition of visual stimuli. *Journal of the International Neuropsychological Society.* **2** : A 45.

Granvold, D.K. (1994). Cognitive and behavioral treatment. Methods and applications. California : Brooks/Cole Publishing Company.

Greenberg, H.R. (1969). Transactions of hair pulling symbiosis. *Psychiatric Quarterly.* **43** : 662-674.

Greenberg, H.R. and Sarner, C.A. (1965). Trichotillomania, symptom and syndrome. *Archives of General Psychiatry.* **12** : 482-489.

Gridlinger, H.M. and Ramsay, E. (1991). Compulsive feather picking in birds. *Archives of General Psychiatry.* **48** : 857.

Gronwall, D.M.A. and Sampson, H. (1974). The psychological effects of concussion. Auckland : University Press.

Gupta, S. and Freimer, M. (1993). Trichotillomania, clomipramine, topical steroids (letter). *American Journal of Psychiatry.* **150** : 524.

Haber, S.N., Kunishio, K., Mizobuchi, M. and Lyndt-Balta, E. (1995). The orbital and medial pre-frontal circuits through the primate basal ganglia. *Journal of Neuroscience.* **15**: 4851-4867.

Hajcak, G., Franklin, M.E., Simons, R.F. and Keuthen, N.J. (2006). Hairpulling and skin picking in relation to affective distress and obsessive-compulsive Symptoms. *Journal of Psychopathology and Behavioral Assessment.* **28 (3)** : 179-187

Hallopeau, F.M.M. (1889). Alopecie par grottage (trichomanie au trichotillomanie). *Annals of Dermatology et de Syphilsgraphie.* **10** : 440 - 441.

Halstead, W.C. (1947). Brain and intelligence. Chicago : University of Chicago Press.

Hamilton, M. (1959). The assessment of anxiety states by rating. *British Journal of Medical Psychology.* **32** : 50-55.

Harrison, G.L. (1986). Disorders in the integument. In : Clinical avian medicine and surgery : including aviculture. (eds. G.L. Harrison and L. Harrison). (509-524). Philadelphia, PA : W.B. Saunders.

Heaton, R. K. (1981). Wisconsin card sorting test (WCST). Psychological assessment Resources : Odessa Florida.

Hollander, E. (1993*a*). Obsessive-compulsive related disorders. Washington DC. American Psychiatric Press.

Hollander, E. (1993b). Obsessive compulsive spectrum disorders : an overview. *Psychiatric Annals.* **23 :** 355-358.

Hollander, E. (1998). Treatment of obsessive-compulsive spectrum disorders with SSRI=s. *British Journal of Psychiatry*. **172 (suppl. 35)** : 7-12.

Hollander, E., DeCaria, C.M. and Nitescu, A. (1992). Serotonergic functions in OCD: behavioural and endocrine responses to m-chlorophenylpiperazine and fenfluramine in patients and healthy volunteers. *Archives of General Psychiatry*. **49** : 21-28.

Hollander, E., Kwon, J.H., Stein, D., Broatch, J., Rowland, C. and Himelein, R. (1996). Obsessive-compulsive and spectrum disorders: Overview and quality of life issues. *Journal of Clinical Psychiatry.* **57 (suppl. 8).** : 3-6.

Hollander, E., Liebowitz, M.R., Winchel, R.(1989). Treatment of body dysmorphic disorder with serotonin reuptake blockers. *American Journal of Psychiatry.* **146** : 768-770.

Hollander, E. McElroy, S.L., Hudson, J.I., et al. (1995). Body dysmorphic disorder : an obsessive-compulsive spectrum disorder, a form of affective disorder , or both? *Journal of Clinical Psychiatry.* **56 (suppl. 4)** :41-51.

Hollander, E. Stein, D.J. and DeCaria, C.M. (1993). Impulsivity and compulsivity : symptoms and diagnoses. *International Psychiatry Today.* **1** :11-12.

Hollander, E. and Wong, C.M. (1995). Obsessive compulsive spectrum disorders. *Journal of Clinical Psychiatry.* **56 (suppl 4)** : 3-6.

Hynes, J.V. (1982). Hypnotic treatment of five adult cases of trichotillomania. *Australian Journal of Clinical and Experimental hypnosis.* **10** : 109-116.

Iancu, I. Weizman, A., Kindler, S. Sasson, Y. And Zohar, J. (1996). Serotonergic drugs in trichotillomania :Treatment results in 12 patients. *Journal of Nervous and Mental Disease.* **184** : 641-644.

Iglauer, F. and Rasim, R. (1993). Treatment of Psychogenic feather picking in psittacine birds with a dopamine antagonist. *Journal of small animal practice.* **34** : 564-566.

Ingle, D. and Crews, D. (1985). Vertebrate neurotology: Definition and paradigms. *Annual Review of Neuroscience*. **8** : 457-494.

Ingram, R.E. (1983). Content and process distinctions in depressive self-schemata. In : Depression and schemata. Symposium presented at the meeting of the American Psychological Association. (L.B Alloy : Chair). Anaheim, CA.

Jacobs, B.J. and Fornal, C.A. (1995). Serotonin and behaviour : a general hypothesis. In: Psychopharmacology: The fourth generation of progress. (eds. F.E. Bloom, and D.J. Kupfer). New York : Raven Press.

Jacobson, E. (1929). *Progressive Relaxation.* Chicago: University of Chicago Press.

Jaspers, K. (1968). General psychopathology. Chicago : University of Chicago Press.

Jenike, M.A. (1989). Obsessive-compulsive and related disorders: a hidden epidemic (editorial). *New England Journal of Medicine.* **321** : 539-541.

Jillson, O.F. (1983). Alopecia II : trichotillomania (trichotillohabitus). *Cutis.* **31** : 383 - 389.

Johnson, C.A. (1987). Chronic feather picking : a different approach to treatment, in : Proceedings of the First International Conference of Zoology and Avian Medicine, Turtle Bay HI.

Joyce, P.R., Mulder, R.T. and Clonninger, C.R. (1994). Temperament predicts clomipramine and desipramine response in major depression. *Journal of Affective Disorders.* **30**:35-46.

Kandel, E.R. (1989). Genes, nerve cells, and the remembrance of things past. *Journal of Neuropsychiatry*. **1** : 103-125.

Kaplan, A. (1992). Trichotillomania U.S. prevalence at 2%, experts report data at national conference. *Psychiatric Times.* **9(12: 1) :** 33-34.

Kaplan, E.F., Goodglass, H. and Weintraub, S. (1983). The Boston naming test (2nd ed.). Philadelphia : Lea and Febiger.

Kaplan, H.I.. and Sadock, B.J. (1991). Synopsis of Psychiatry. Behavioral Sciences. Clinical Psychiatry. (6^{th} ed.). Baltimore : Williams and Wilkens.

Kaplan, L. (1991). Female perversions. New York: Doubleday Dell.

Kazdin, A.E. and Wilson, G.T. (1978). Evaluation of behaviour therapy: Issues, evidence and research strategies. Ballinger : Cambridge, California.

Kelly, G. (1955). The psychology of personal constructs. New York : Norton.

Kendall, P.C. and Ingram, R. (1987). The future for cognitive assessment of anxiety : Let=s get specific. In : Anxiety and stress disorders. (eds. L. Michelson and L.M. Ascher). New York : Guilford Press.

Kennedy, K.A. and Draper, D.D. (1991). Common psittacine behavioral problems. *Iowa State University Veterinarian.* **53** : 21-25.

Kenny, D.E. (1994). Use of naltrexone for treatment of psychogenically induced dermatoses in five zoo animals. *Journal of the American Vet. Med. Association.* **205** : 1021-1023.

Keuthen, N.J., Makris, N., Schlerf, J.F., Martis, B., Savage, C.R., McMullin, K., Seidman, L.J., Schmahmann, J.D., Kennedy, D.N., Hodge, S.M. and Rauch, S.L. (2007). Evidence for reduced cerebellar volumes in trichotillomania. *Biological Psychiatry*. **61** ; 374-381.

Keuthen, N.J., O=Sullivan, R.L., Goodchild, P., Rodriques, D., Jenike, M.A. and Baer,L. (1998). Retrospective review of treatment outcome for 63 patients with trichotillomania. *American Journal of Psychiatry*. **155 (4)** : 560 -561.

Keuthen, N.J., O'Sullivan, R.L., Ricciardi, J.N., Shera, D., Savage, C.R., Borgmaa, A.S., et al, (1995). The Massachusetts General Hospital (MGH) hairpulling scale: 1, development and factor analyses. *Psychotherapy and Psychosomatics.* **64** : 141-145.

Keuthen, N.J., O=Sullivan, R.L. and Sprich-Buckminster, S. (1998). Trichotillomania : Current issues in conceptualization and treatment. *Psychotherapy and Psychosomatics.* **67** : 202 - 213.

Keuthen, N.J., Savage, C.R., O=Sullivan, R.L., Brown, H.D., Shera, D.M., Cyr, P., Jenike, M.A. and Baer, L. (1996). Neuropsychological functioning in trichotillomania. *Biological Psychiatry.* **39** : 747-749.

Keuthen, N.J., Stein, D.J. and Christenson, M.D. (2001). Help for hair pullers. California : New Harbinger Publication Inc.

King, R.A., Scahill, L., Vitulano, L.A., Schwab-Stone, M., Tercyak, K.P.and Riddle, M.A.(1995). Childhood trichotillomania : Clinical phenomenology, co morbidity, and family genetics. *Journal of American Academy of Child and Adolescent Psychiatry.* **34** : 1451-1459.

Klerman, G.L. (1990). Treatment of recurrent unipolar major depressive disorder: commentary on the Pittsburgh study. *Archives of General Psychiatry*. **47** : 1158-1162.

Koran, L.M., Ringold, A. and Hewlett, W. (1992). Fluoxetine for trichotillomania : An open clinical trial. *Psychopharmacology Bulletin.* **28** : 145-149.

Krishnan, K.R., Davidson, J.R. and Guajardo, C. (1985). Trichotillomania : A review. *Comprehensive Psychiatry*. **26 :** 123-128.

Kunkle, G. (1995). Therapy for feline dermatoses. *Veterinary Clinics of North American Small Animal Practice.* **25** : 981-1005.

Lazarus, A. (1972). Behaviour therapy and beyond. New York : McGraw-Hill.

Leckman, J.F. (2002). Tourette=s syndrome. *Lancet*. **360** : 1577-1586.

Leckman, J.F., Walker, D.E., Goodman, W.K., Pauls, D.L. and Cohen, D.J. (1994). AJust right≅ perceptions associated with compulsive behavior in Tourette=s syndrome. *American Journal of Psychiatry.* **151(5)** : 675-680.

Leonard, H., Swedo, S., Rapoport, J.L., et al. (1988). Treatment of childhood OCD with clomipramine and desmethyl-imipramine : a double-blind crossover comparison. *Psychopharmacological Bulletin.* **24** : 93-95.

Lenane, M.C., Swedo, S.E., Rapoport, J.L., Leonard, H., Sceery, W. and Guroff, J.J. (1992). Rates of OCD in first degree relatives of patients with trichotillomania : a research note. *Journal of Child Psychology and Psychiatry*. **5** : 925-933.

Levine, B.A. (1976). Treatment of trichotillomania by covert-sensitization. *Journal of Behaviour Therapy and Experimental Psychiatry*. **7** : 75-76.

Lloyd, G.E.R.(1983). In: Hippocratic Writings. (trans.: Chadwick, J.M Mann, W.N., Lonie, I.M., et al.). Harmondsworth, UK, Penguin Books. 100-103.

Lockwood, J.H., Moulton, D.O., Picon-Nieto, L., Vanderloeg, R.D. and Ochipa, C. (1996). Visual associative agnosia : A traumatic brain injury case without typical focal anatomic finding. *Journal of the International Neuropsychological Society*. **2** : A 17.

Lochner, C., Seedat, S., Du Toit, P.L., Nel, D.G., Niehaus, D.J.H., Sandler, R. and Stein, D.J. (2005). Obsessive-compulsive disorder and trichotillomania: a phenomenological comparison. *BMC Psychiatry*. **5 (2)** : 1471-244X/5/2.

Lorenz, K.Z. *The Foundations of Etiology*. (1981). New York: Springer-Verlag.

Luria, A. (1961). The role of speech in the regulation of normal and abnormal behaviors. New York : Liveright.

Luria, A.R. (1973). The working brain. An introduction to neuropsychology. UK : Penguin Books.

Luthe, W. (1969). Autogenic therapy. New York : Grune and Stratton.

Lutzker, J.R. and Lamazor, E.A. (1985). Behavioral pediatrics : research, treatment, recommendations. *Progressive Behaviour Modification*. **19** : 231-234.

Mahoney, M.J. (1974). Cognition and behaviour modification. Cambridge : Ballinger.

Mahr, G. (1993). Fenfluramine and trichotillomania. *Psychosomatics.* **34** : 284.

Mannino, F.V. and Delgado, R.A. (1969). Trichotillomania in children : a review. *American Journal of Psychiatry*. **126** :505-511.

Mansueto, C.S. (1990). Typography and Phenomenology of Trichotillomania. Paper presented at the annual convention of the Association for the Advancement of Behaviour Therapy. San Francisco, C.A.

Manzo, P.A., Caviness, V.S. and Jenike, M.A. (1997). Reduced basal ganglia volumes in trichotillomania measured via morphometric magnetic resonance Imaging. *Society of Biological Psychiatry*. **42** : 39-45.

March, R., Alexander, G.M., Packard, M.G., Zhu, H., Wingard, J.C., Quackenbush, G. and Peterson, B.S. (2004). Habit Learning in Tourette syndrome. *Archives of General Psychiatry*. **61** : 1259-1268.

Marks, J.M., Gray, S., Cohen, D., Hill, R., Mawson, D., Ramm, E. and Stern, R.S. (1983). Imipramin and brief therapist-aided exposure in agoraphobics having self-exposure homework. *Archives of General Psychiatry*. **40** : 153-162.

Martin, A., Pigott, T.A., Lalonde, F.M., Dalton, I., Dubbert.B. and Murphy, D.L. (1993). Lack of evidence for Huntington=s disease-like cognitive dysfunction in OCD**.** *Biological Psychiatry*. **33** : 345 -353.

Mataix-Cols, D., Wooderson, S., Lawrence, N., Brammer, M.J., Speckens, A. and Phillips, M.L. (2004). Distinct neural correlates of washing, checking and hoarding symptom dimensions in OCD. *Archives of General psychiatry*. **61** : 565-576.

Matthews, C.G. and Klove, H. (1964). Instruction Manual for the adult neuropsychology test battery. Madison, WI : University of Wisconsin Medical School.

Maultsby, M.C. (1975). Help yourself to happiness through rational self counseling. Boston : Esplanade Books.

Mavissakalian, M. and Michelson, L. (1986). Agoraphobia: relative and combined effectiveness of therapist -assisted in vivo exposure and imipramine. *Journal of Clinical Psychiatry*. **47** :117-122.

McDonald, W.M., Husain, M., Doraiswamy, P.M., Figiel,G., Boyko, O. and Krishnan. (1991).A magnetic resonance imaging study of age-related changes in human putamen nuclei.. *Neuro reports* . **2** : 41-44.

Mc Elroy, S.L., (1995)

McLauchlin, J.G. and Nay, W.R. (1975). Treatment of trichotillomania using positive coverants and response cost : a case report. *Behaviour Therapy.* **6** : 87-91.

Meichenbaum, D. (1974). Self-instructional training : a cognitive prosthesis for the aged. *Human Development.* **17** : 273-280.

Meichenbaum, D. (1975). Theoretical and treatment implications of development research on verbal control of behaviour. *Canadian Psychological Review.* **16** : 22-27.

Meichenbaum, D.B. (1977). Cognitive behaviour modification : An integrative approach. New York : Plenum.

Meichenbaum, D.B. (1985). Stress inoculation training. New York : Pergamon Press.

Meichenbaum, D. and Goodman, J. (1971). Training impulsive children to talk to themselves : A means of developing self control. *Journal of Abnormal Psychology*, **77** : 115-126.

Merchant, S.R. (1994). Diagnosis of feline skin diseases based on cutaneous reaction patterns. *Compendium of Continuing Education for Practicing Veterinarians.* **16** : 163-172.

Mesulam, M.M.. (1985). Patterns in behavioural anatomy: Association areas, the limbic system and hemispheric specialization. In: Principles of Behavioural Neurology. (ed. M.M. Mesulam). Philadelphia : F.A. Davis Co. : 1-70.

Miller. D.T. and Porter, C.A. (1988). Errors and biases in the attribution process. In : Social cognition and clinical psychology. (ed. L.J. Abramson). New York : Guilford Press.

Milner, B. (1965). Visually-guided maze learning in man: Effects of bilateral hippocampal, bilateral frontal, and unilateral cerebral lesions. *Neoropsychologia.* **3** : 317-338.

Miltenberger, R.G. and Fuqua, R.W. (1985). A comparison of contingent vs. Non-contingent competing response practice in the treatment of nervous habits. *Journal of Behaviour Therapy and Experimental Psychiatry.* **16** :195-200.

Minichiello, W.E., O=Sullivan, R.L., Osgood-Hynes, D. and Baer, L. (1994). Trichotillomania: Clinical Aspects and Treatment Strategies. *Harvard Rev Psychiatry.* **1(6)** : 336-344.

Money, J., Alexander, D and Walker, H.T. (1965). A standardized road map test of direction sense. Baltimore : John Hopkins University Press.

Monroe, J.T. and Abse, D.W. (1963). Psychopathology of Trichotillomania and trichophagy. *Psychiatry*. **26** : 95-103.

Montarolo, P.G., Goelet, P., Castellucci, V.F., Morgan, J., Kandel, E.R. and Schacher, S. (1986). A critical period for macromolecular synthesis in long-term heterosynaptic facilitation in *Alypsia*. *Science*. **23** : 1249-1253.

Moon-Fanelli, A.A. and Dodman, N.H. (1998). Description and development of compulsive tail-chasing in terriers and response to clomipramine treatment. *Journal of American Veterinary Medicine Association.* **212** : 1252-1257.

Moss, M.B., Albert, M.S., Butters, N. and Payne, M. (1986). Differential patterns of memory loss among patients with Alzheimer's disease, Huntington's disease, and alcoholic Korsakoff's syndrome. *Archives of Neurology*. **43** : 239-246.

Muller, G.H., Kirk, R.W. and Scott, D.W. (1989). *Small animal dermatology. 4th ed.* Philadelphia. PA. WB Saunders.

Muller, S.A. (1987). Trichotillomania. *Dermatological Clinic*. **5** : 595-601.

Muller, S.A. and Winkelman, R.K. (1972). Trichotillomania: a clinicopathologic study of 24 cases. *Archives of Dermatology*. **105** : 353-340.

Ninan, P.T., Mansueto, C., Rothbaum, B., O=Sullivan, R. and Nemeroff, (n.d). Challenges in the classification and treatment of trichotillomania. (n.pub.).

Ninan, P.T., Rothbaum, B.O., and Stipetic, M. (1992). CSF 5HIAA as a predictor of treatment response in trichotillomania. *Psychopharmacological Bulletin.* **28** : 451-455.

Oranje, A.P., Peereboom-Wynia, J.D.R. and DeRaemaecker, D.M.J. (1986). Trichotillomania in childhood. *Journal of the American Academy of Dermatology*. **15** : 614-619.

O=Sullivan, R.L., Keuthen, N.J., Hayday, C.F., Ricciardi,, J.N., Buttolph, M.L., Jenike, M.A. and Baer, L. (1995). The MGH Hair pulling Scale II : reliability and validity. *Psychotherapy Psychosomatics.* **64** : 146 -148.

O'Sullivan, R.L., Keuthen, N.J. and Rodriques, D. (1998)Venlafaxine treatment of trichotillomania : an open series of ten cases. *CNS Spectrums.* **3(10)** : 56-53.

O'Sullivan. R.L., Rauch, S., Breiter, H.C., Grachev, I.D., Baer, L., Kennedy, D.N., Keuthen, N.J., Savage, C.R., Manzo, P.A., Caviness, V.S. and Jenike, M.A. (1997), Reduced basal ganglia volumes in trichotillomania measured via morhometric magnetic resonance imaging. *Biological Psychiatry*. **42** : 39-45.

Osterrieth, P. and Rey, A. (1944). Le test de copie d=une figure complex. *Archives of Psychology*. **30** : 205-221.

Ottens, A.J. (1981). Multifaceted treatment of compulsive hair pulling. *Journal of Behavioural Therapy and Experimental Psychiatry*. **12** : 77-80.

Ottens, A.J. (1982). A cognitive behavioral modification treatment of trichotillomania. *Journal of American College Health.* **31** : 78-81.

Pantelis, C., Barnes, T.R.E. and Nelson, H.E. (1992). Is the concept of frontal-subcortical dementia relevant to schizophrenia? *British Journal of Psychiatry*. **160** : 442-460.

Pazos, A., Cortes, R. and Palacios, J. (1985). Quantitative auto-radiographic mapping of serotonin receptors in rat brain: II. Serotonin II receptors. *Brain Research*. **346** : 231-249.

Pazos, A. and Palacois, J. Quantitative auto-radiographic mapping of serotonin receptors in rat brain : I. Serotonin I receptors. *Brain Research*. **346** : 205-230.

Petersen, S.E., Fox, P.T., Snyder, A.Z. and Raichle, M.E. (1990). Activation of extrastriate and frontal cortical areas by visual words and word-like stimuli. *Science.* **249** : 1041-1044.

Peterson, B.S., Riddle, M.A., Cohen, D.J., et al., (1993). Reduced basal ganglia volumes in Tourette=s syndrome using three-dimensional reconstruction techniques from magnetic resonance images. *Neurology* **43** : 941-949.

Peterson, B.S., Thomas, P., Kane, M.J., Scahill, L., Zhang, H., Bronen, R., King, R.A.,
Leckman, J.F. and Staib, L. (2003). Basal ganglia volumes in patients with Gilles de la Tourette syndrome. *Archives of Psychiatry.* **60** : 415-424.

Phelps, M.E., Kuhl, D.E. and Mazziotta, J.C. (1981). Metabolic mapping of the brain=s response to visual stimulation : Studies in humans. *Science.* **211** : 1445-1448.

Philippopoulus, G.S. (1961). A case of trichotillomania (hair pulling). *Acta Psychotherapy*. **9** : 304-312.

Piaget, J. (1950). Psychology of intelligence. (M.Piercy and DE Berlyne, trans). New York: Harcourt Brace and Co.

Piaget, J. (1960). The moral judgement of the child. (M.Gabain, trans.). Illinois : Free Press.

Pigott, T.A., L=Heuex, F., Grady, T.A., Bernstein, S., Dubbert, B., Rubenstein, C., Hill, J. and Murphy, (1992). Controlled comparison of clomipramine and fluoxetine in trichotillomania (abstracts of panels and posters) 31st Annual Meeting Am Coll

Neuropsychopharmacology, San Juan. In : N. Keuthen, et. al., 1998. Trichotillomania current issues in conceptualization and treatment. *Psychotherapy and Psychosomatics* . **67** : 202-213.

Pollard, C.A., Ibe, I.O., Krojanker, D.N., Kitchen, A.D., Bronson, S.S. and Flynn, T.M. (1991). Clomipramine treatment of trichotillomania : a follow-up report on four cases. *Journal of Clinical Psychiatry.* **52** : 128-130.

Posner, M.I. and Petersen, S.E. (1990). The attention system of the human brain. *Annual Review of Neuroscience.* **13** : 25-42.

Rapoport, J.L. and Wise, S.P. (1988). OCD: Evidence for basal ganglia dysfunction. *Psychopharmacology Bulletin*. **24 (3)** : 380-384.

Randall, W. (1988). Grooming reflexes in the cat : endocrine and pharmacological studies. *Annual New York Academy of Science.* **525** : 301-320.

Ratner, R.A. (1989). Trichotillomania. *Treatment of Psychiatric disorders* : *a taskforce report of the American Psychiatric association.* Washington, DC : American Psychiatric Association. 2481-2486.

Rauch, S.L., Savage, C.R. and Brown, H.D. (1995). A PET investigation of implicit and explicit sequence learning. *Human Brain Mapping.* **3** : 271-286.

Rauch, S.L., Wright, C.I., Savage, C.R., Martis, B., McMullin, K.G., Wedig, M., Gold, A. and Keuthen, N. (2007). Brain activation during implicit sequence learning in individuals with trichotillomania. *Psychiatry Research : Neuroimaging.* **!54** : 233-240.

Reading, P.J. (1991). Frontal lobe dysfunction in schizophrenia and Parkinson's disease, a meeting point for neurology, psychology and psychiatry : discussion paper. *Journal of the Royal Society of Medicine*. **84** : 349-353.

Reeve, E.A., Bernstein, G.A. and Christenson, G.A. (1992). Clinical characteristics and psychiatric comorbidity in children with trichotillomania. *Journal of the American Academy of Child and Adolescent Psychiatry.* **31** : 132-138.

Rehm, L.P. (1977). A self control model of depression. *Behavior Therapy,* **8,** 787-804.

Reiman, E.M., Raichel, M.E., Butler, F.K., Herscovitch, P. and Robins, E. (1984). A focal brain abnormality in panic disorder, as a severe form of anxiety. *Nature*. **310** : 683-685.

Reiman, E.M., Raichle, M.E., Robins, E., Butler, F.K., Herscovitch, P., Fox, P. and Perlmutter, J. (1986). The application of positron emission tomography to the study of panic disorder. *American Journal of Psychiatry.* **143** : 469-477.

Reiman, E.M., Raichle, M.E., Robins, E., Minton, M.A., Fusselman, M.J., Fox, P.T., Price, J.L. and Hackman, K.A. (1989). Neuroanatomical correlates of a lactate-induced anxiety attack. *Archives of General Psychiatry*. **46** : 493-500.

Reitan, R.M. (1959). Correlations between the trail making test and the Wechsler-Bellevue scale. *Perceptual and Motor Skills.* **9** : 127-130.

Reitan, R.M. and Davidson, L.A. (1974). Clinical Neuropsychology : Current status and applications. New York : Winston/Wiley.

Rettew, D.C., Cheslow, D.L., Rapoport, J.L., Leonard, H.L., Lenane, M.C., Black, B. and Swedo, S.E. (1991). Neuropsychological test performance in trichotillomania: A further link with OCD. *Journal of Anxiety Disorders*. **5** : 225-235.

Ristvedt, S.L. and Christenson, G.A. (1996). The use of pharmacological pain sensitization in the treatment of repetitive hair-pulling. *Behavioural Research and Therapy.* **34** : 647-648.

Robinson, D., Wu, H., Munne, R.A. (1995). Reduced caudate nucleus volume in OCD. *Archives of General Psychiatry*. **52** : 393-398.

Roland, P.E., Eriksson, L., Widen, L. and Stone-Elander, S. (1989). Changes in regional cerebral oxidative metabolism induced by tactile learning and recognition in man. *European Journal of Neuroscience*. **1** : 3-18.

Rosenthal, K. (1996). Diagnosis in the feather picking bird. In : The North American Veterinary Conference Proceedings. Orlando Fl. 741.

Rosskopf, W.J., Woerpel, R.W. and Reed-Blake. (1986). Feather picking in psittacine birds : a clinician=s approach to diagnosis and treatment. In : Proceedings of the Association of Avian Veterinarians. Miami, FL. 265-279.

Rotter, J.B. (1966). Generalized expectancies for internal versus external control of reinforcement *Psychological Monographs,* **80** : (609).

Rothbaum, B.O. (1990). Trichotillomania treatment programme. Presentation at the *Annual Meeting of the Association for the advancement of Behaviour Therapy*, SIG Section, San Francisco, CA.

Rothbaum, B.O. (1992). The behavioural treatment of trichotillomania. *Behavioural Psychotherapy*. **20** : 85-90.

Rothbaum, N.O. and Ninan, P. (1992). Treatment of trichotillomania: Behaviour therapy versus clomipramine. *Annual Meet Assoc Adv Behav Ther,* Boston.

Rothbaum, B.O. and Ninan, P.T. (1994). The assessment of trichotillomania. *Behaviour Research and Therapy*. **6** : 651 - 662.

Rothbaum, B.O., Shaw, L., Morris, R. and Ninan, P.T. (1993).Prevalence of trichotillomania in a college freshman population. [letter] . *Journal of Clinical Psychiatry.* **54** : 72.

Rowen, R. (1981). Hypnotic age regression in the treatment of a self-destructive habit : Trichotillomania. *The American Journal of Clinical Hypnosis.* **23(3)** : 195-197.

Rush, A.J. and Beck, A.T. (1978). Adults with affective disorders. In : Behavioral therapy in the psychiatric setting. (eds. M. Hersen and A.S. Bellack .). Baltimore : Williams and Wilkins.

Salkovskis, P.M. (1989). Cognitive Behavioural factors and the persistence of intrusive thoughts in obsessional problems. *Behaviour Research and Therapy.* **27(6)** ; 677-682.

Sawyer, S.S., Moon-Fanelli, A.A. and Dodman, N.D. Feline psychogenic alopecia : a survey. *Journal of the American Veterinary Medical Association* (in press).

Saxena,S., Brody, A.L., Schwartz, J. M. and Baxter, L.R. (1998). Neuroimaging and Frontal-subcortical circuitry in OCD. *British Journal of Psychiatry.* **173 (suppl. 35)** : 26-37.

Saykin, A.J., Gur, R.E., Mozley, P.D., Mozley, L.H., Resnick, S.M., Kesler, B. and Stafinicik, P. (1991). Neuropsychological functions in schizophrenia. Selective impairment in memory and learning. *Archives of General Psychiatry.* **48** : 618-624.

Schacter, M. (1961). Zum problem der kindlichen trichotillomania. *Praxis Kinderpsychol. Kinderpsychiatr.* **10** : 120-124.

Schlosser, S., Black, D.W., Blum, N. et al. (1994). The demography, phenomenology, and family history of 22 persons with compulsive hair pulling. *Annals of Clinical Psychiatry.* **6** : 147-152

Schnur, R.G. and Davidson, S. (1989). Trichotillomania in a ten year old boy : gender identity issues formulated in terms of individual and family issues. *Canadian Journal of Psychiatry.* **34** : 721-724.

Schuckit, M.A.(1992). An introduction and overview to clinical applications of neuroSPECT in psychiatry. *Journal of Clinical Psychiatry.* **53 (suppl)** : 3-6.

Schwartz, J.M. (1998). Neuroanatomical aspects of cognitive behaviour therapy response in OCD. *British Journal of Psychiatry.* **173 (suppl. 35)** : 38-44.

Schwartz, J.M., Stoessel, P.W., Baxter, L.R. Martin, K.M. and Phelps, M. (1996). Systematic changes in cerebral glucose metabolic rate after successful behaviour modification treatment of OCD. *Archives of General Psychiatry.* **53** : 109-113.

Schweizer, E. and Rickels, K. (1997). *Journal of Clinical Psychiatry*. **58** (suppl 11) : 30-38.

Seligman, M.E.P. (1975). Helplessness : On depression, development and death. San Francisco : W.H.Freeman.

Soubrie, P. (1986). Reconciling the role of central serotonin neurons in human and animal behavior. *Behavioral Brain Science*. **9** : 319-364.

Sperling, M. (1954). Use of hair as a bi-sexual symbol. *Psychoanalytic review*. **41** : 363-365.

Spielberger, C.D. Gorsuch, R.L. and Lushene, R.E. (1970). The state-trait anxiety inventory : Test manual for form X. Palo Alto : Consulting Psychologists Press.

Spreen, O. and Strauss, E. (1998). A compendium of neuropsychological tests. Oxford University Press. New York.

Stanley, M.A., Breckenridge, J. Swann, A, (1997a). Fluvoxamine treatment of trichotillomania, *Journal of Clinical Psychiatry*. **17** : 278-283.

Stanley, M.A., Hannay, H.J. and Breckenridge, J.K. (1997b). The Neuropsychology of Trichotillomania. *Journal of Anxiety Disorders.* **11(5)** : 473 - 488.

Stanley, M.A., Swann, A.C., Bowers, T.C., Davis, M.L. and Taylor, D.J. (1992). Comparison of clinical features in trichotillomania and OCD. *Behavioral Research and Therapy*. **1** :39-44.

Stedman=s Medical Dictionary, 24th Edition (1982). Baltimore, M.D., Williams and Wilkens.

Stein, D.J., Christenson, G.A. and Hollander, E. (1999). Trichotillomania. American Psychiatric Press. Washington DC.

Stein, D.J., Coetzer, R., Lee, M., Davids, B. and Bouwer, C. (1997). Magnetic resonance brain imaging in woman with obsessive-compulsive disorder and trichotillomania. *Psychiatry Research Neuroimaging.* **74** : 177-182.

Stein, D.J. Dodman, N.H. and Borchelt, P. (1994). Behavioural disorders in veterinary practice: relevance to psychiatry. *Comprehensive Psychiatry.* **35** : 275-285.

Stein, D.J. and Hollander, E. (1992a). Dermatology and conditions related to OCD. *Journal of the American Academy of Dermatology*. **26** : 237-242.

Stein, D.J. and Hollander, E. (1992b). Low-dose Pimozide augmentation of serotonin reuptake blockers in the treatment of trichotillomania. *Journal of Clinical Psychiatry.* **53 (4)** : 123-126.

Stein, D.L. Hollander, E. (1993). Impulsive aggression and OCD. *Psychiatric Annals*. **23** : 389-395.

Stein, D.J., Hollander, E. and Liebowitz, M.R. (1993). Neurobiology of impulsivity and the impulse control disorders. *Journal of Neuropsychiatry and Neuroscience*. **5** : 9-17.

Stein, D.J., Hollander, E., Simeon, D., Cohen, L., Islam, M.N. and Aronowitz, B. (1994). Neurological soft signs in female trichotillomania patients, OCD patients and healthy control subjects. *Journal of Neuroscience*. **6 (2)** : 184-187.

Stein, D.J., Hutt, C.S., Spitz, J.L. and Hollander, E. (1993). Compulsive picking and OCD. *The Academy of Psychosomatic Medicine*. 177-181.

Stein, D.J. Shoulberg, N., Helton, K. and Hollander, E. (1992). The neuroethological approach to OCD. *Comprehensive Psychiatry*. **33** : 274 - 281.

Stein, D.J., Simeone, D., Cohen, L.J., et al. (1995). Trichotillomania and OCD. *Journal of Clinical Psychiatry*. **4** : 28-34.

Stein, D.J., Spadaccini, E. and Hollander. E. (1995). Meta-analysis of pharmacotherapy trials for OCD. *International Clinical Psychopharmacology*. **10** : 11-18.

Stein, D.J., Van Heerden, B., Hugo, C., Van Kradenburg, J., Warwick, J., Zungu-Dirwayi, N. and Seedat, S. (2002). Functional brain-imaging and pharmacotherapy in trichotillomania. Single photon emission computer tomography before and after treatment with the selective serotonin inhibitor citalopram. *Progress in Neuro-Psychopharmachology and Biological Psychiatry*. **26** : 885-890.

Stein, D.J., Wessels, C., Carr et al. (1997). Hair pulling in a patient with Sydenham's Chorea. *American Journal of Psychiatry*. **154** : 1320.

Steinbush, H. (1981). Distribution of serotonin-immunoreactivity in the central nervous system of the rat. *Neuroscience*. **6** : 557-618.

Sticher, M., Abramovits, W. and Newcomer, V.D. : Trichotillomania in Adults. *Cutis.* **26** : 90-101.

Straus. E.W, (1966). Phenemenological Psychology : Selected Papers. New York : Basic Books.

Streichenwein, S.M. and Thornby, J.I. (1995). A long-term double-blind, placebo-controlled crossover trial of the efficacy of fluoxetine for trichotillomania. *American Journal of Psychiatry.* **152** : 192-196.

Stuart, A., Slater, J.M., Unwin, H.L. and Crossman, A.R. (1986). A semiquantitative analysis of 5-hydroxytryptamine-1 receptors in the primate brain. *Neuroscience*. **18** : 619-639.

Stuss, D.T., Kaplan, E.F., Benson, D.F. et al. (1982). Evidence for the involvement of orbitofrontal cortex in memory functions : An interference effect. *Journal of Comprehensive Physiological Psychology*. **96** : 913-925.

Svrakic, M.D., Whitehead, C., Przybeck, T.R. and Clonninger, C.R. (1993). Differential diagnosis of personality disorders by the seven factor model of temperament and character. *Archives of General Psychiatry.* **50** : 991-999.

Swedo, S.E. (1989). Rituals and releasers : an ethological model of OCDs. In: OCD in children and adolescents. (ed. J.L. Rapoport). Washington, DC: American Psychiatric Press. 269-288.

Swedo, S.E. (1993). Trichotillomania. *Psychiatric Annals*. **23** (7) : 402-407.

Swedo, S.E.(1993). Trichotillomania. *Child and Adolescent Psychiatric Clinics of North America.* **2 (4)** : 685-694.

Swedo, S.E. and Leonard, H.L. (1992). Trichotillomania. An obsessive-compulsive spectrum disorder? *Psychiatric Clinics of North America.* **15(4)** : 777-790.

Swedo, S.E. and Leonard H.L. (1992). Trichotillomania : an obsessive compulsive spectrum disorder? *Psychiatric Clinics of North America*. **15** (4) : 777-790.

Swedo, S.E., Leonard, H.L., Rapoport, J.L., Lenane, M.C., Goldberger, E.L. and Cheslow, D.L.(1989). A double-blind comparison of clomipramine and desipramine in the treatment of trichotillomania (hair pulling).*New England Journal of Medicine.* **321** : 497 - 501.

Swedo, S.E., Rapoport, J.L., Cheslow, D.L. (1989). High prevalence of obsessive-compulsive symptoms in patients with Sydenham's chorea. *American Journal of Psychiatry*. **146** : 246-249.

Swedo, S.E., Rapoport, J.L., Leonard, H.L., Lenane, M. and Cheslow, D. (1989).OCD in children and adolescents: clinical phenomenology of 70 consecutive cases. *Archives of General Psychiatry*. **46** : 335-341.

Swedo, S.E., Rapoport, J.L., Leonard, H.L., Shapiro, M.B., Rapoport, S.I. and Grady, C.L. (1991). Regional cerebral glucose metabolism of women with trichotillomania. *Archives of General Psychiatry*. **48** : 828-833.

Tahan, S.R. and Fogt, F. (1992). Trichotillomania : pathologic psychiatry? (Letter). *American Journal of Dermatopathology*. **14** : 582

Tataryn, D.J., Nadel, L and Jacobs, W.J. (1989). Cognitive therapy and cognitive science. In : Comprehensive handbook of cognitive therspy (eds. A.Freeman, K.M. Simon, L.E. Beutler and H. Arkowitz). New York : Plenum.

Taylor, J.G. (1963). A behavioral interpretation of obsessive compulsive neurosis. *Behavior Research.* **1** : 237-244.

Thoren, P., Asberg, M., Bertilsson, L., et al.(1980). Clomipramine treatment of OCD, II: biochemical aspects. *Archives of General Psychiatry*. **37** : 1289-1294.

Traber, J., Spencer, D.G., Glaser, T., et al. (1988). Actions of psychoactive drugs on ACTH- and novelty-induced behaviour in the rat. *Annual New York Academy of Science.* **525** : 270-280.

Trenerry, M.R., Crosson, B., DeBoe, J. and Leber, W.R. (1990). Visual search and attention test. Odessa Fl : Psychological Assessment Resources.

Vallis, T.M., Shaw, B. and McCabe, S. (1988). The relationship between therapist competence in cognitive therapy and general therapy skills. *Journal of Cognitive Psychotherapy* : *An International Quarterly*, **2** : 237-250.

Vandenburg, S.G. and Kuse, A.R. (1978). Mental rotations : A group test of three dimensional spatial visualization. *Perceptual and Motor Skills.* **47** : 599-604.

Van Essen, D.C. and Maunsell, J.H.R. (1983). Hierarchical organization and functional streams in the visual cortex. *Trends in Neuroscience.* **6** : 370-375.

Van Heertum, R.L. (1992).Brain imaging and psychiatry. *Journal of Clinical Psychiatry*. **53 (11: suppl)** : 13-19.

Vitulano, L.A., King, R.A., Scahill, L. and Cohen, D.J. (1992). Behavioral treatment of children and adolescents with trichotillomania. *Journal of the American Child and Adolescent Psychiatry*. **31** : 139-146.

Voith, V.L. and Borchelt, P.L. (1996). Social behavior of domestic cats. In: Readings in Companion Animal Behavior. (eds. V.L. Voith, P.L. Borchelt and N.J. Trenton). *Veterinary Learning Systems*. 248-257.

Vygotski. L. (1962). Thought and language. New York : Wiley.

Vythilingum, B., Warwick, J., Van Kradenburg, J., Hugo, C., Van Heerden, B. and Stein, D.J. (2002). SPECT scans in identical twins with trichotillomania. *Journal of Neuropsychiatry and Clinical Neuroscience*. **14(3)**. 340-342.

Wechsler, D. (1981). Wechsler Adult Intelligence Scale -Revised. Cleveland : The Psychological Corporation.

Weller, E.B., Weller, R.A. and Carr, S. (1989). Imipramine treatment of trichotillomania and co-existing depression in a seven-year-old. *Journal of the American Academy of Child and Adolescent Psychiatry.* **28** : 952-953.

Wepman, J. (1973). The auditory discrimination test. Chicago : Language Research Association Inc.

WHO - World Health Organization. (2002). *International Classification of Diseases.* Geneva.

Winchel, R.M. (1992). Trichotillomania: Presentation and treatment. *Psychiatric Annals.* **22(2)** : 84-89.

Winchel, R.M., Jones, J.S., Stanley, B., Molcho, A. and Stanley, M. (1992). Clinical characteristics of trichotillomania and its response to fluoxetine. *Journal of Clinical Psychiatry.* **53(9)** : 304-308.

Winfrey, L.L and Goldfried, M.R. (1986). Information processing and the human change process. In : Information processing approaches to clinical psychology. (eds. R. Ingram). New York : Academic Press.

Wolpe, J. (1958). *Psychotherapy by reciprocal inhibition.* Stanford, California : Stanford University Press.

Young, M.S. and Manning, T.O. (1984). Psychogenic Dermatosis (dog and cat). *Dermatology Reports.* **3** : 1-8.

Zaiden S.H. (1951). Skin, psychodynamic and psychopathological concepts. *Journal of Nervous and Mental Diseases.* **113** : 388-394.

Zohar, J., Kaplan, Z. and Benjamin, J. (1994). Compulsive exhibitionism successfully treated with fluvoxamine : a controlled case study. *Journal of Clinical Psychiatry.* **55** : 86-88.

Zohar, J, Mueller, E.A. and Insel, T.R. et al. (1987). Serotonergic responsivity in OCD : comparison of patients and healthy controls. *Archives of General Psychiatry.* **44** : 167-172.

A2 Information and consent form

SPECT and neuropsychological studies of the effects of Cognitive Behavior Therapy in trichotillomania (compulsive hair pulling).

We are currently conducting research on people with trichotillomanic symptoms (i.e. hair pulling), which involves brain imaging and Cognitive Behaviour Therapy. The imaging procedures is called SPECT (single photon emission computerized tomography). Cognitive Behaviour Therapy is a psychotherapeutic programme which teaches a patient / client to become aware of their problem and identify habit prone situations.
We are requesting people suffering from trichotillomania, to voluntarily participate in this stydy. All information will be considered strictly confidential and participants may withdraw from the study at ant time

The sequence of procedures is as follows:

1. Preparation

You will be asked not to eat or smoke after 12 o'clock midnight, prior to having the brain scan.

2. Brain imaging

A SPECT scan will be taken prior to the Cognitive Behaviour Therapy programme and another SPECT scan after the therapy.

Prior to the scanning you will be asked to read for a half an hour as to trigger trichotillomanic activity.

On the day of the scans, you need to arrive at 9.00 a.m. You will be asked to complete a few questionnaires to determine your level of anxiety and trichotillomania.

A radioactive dye will into a vein in your arm. This compound (99mTc-HMAPO/ Ceretec) can be given in large doses and is a substance used for a variety of purposes in radiology. It has been approved by the Food and Drug Administration (FDA), and the only known side effect, in the case of hypersensitivity, is an itchy skin rash, which disappears spontaneously within a few days.

You will then be asked to lie on your back while the scanner rotaes around your head taking pictures of your brain. The exposure to radioactivity is approximately that of doubly the amount needed for a chest X-Ray. So far no report of radiation being harmful for this type of study has been reported.

3. Neuropsychological testing

You will be asked to complete a battery of neuropsychological testing at the beginning and at the end of the study. Theses are pen and paper tests.

4. Cognitive Behaviour Therapy

After the first SPECT and neuropsychological testing and during the subsequent 9-12 weeks, between the scans, the Cognitive Behaviour Therapy programme will be followed. Weekly sessions will be conducted by the researcher.

5. Brain imaging

A second SPECT scan will be done following the same procedures as the first.

6. Risks

Possible risks of the study is that you will receive psychotherapeutic treatment regarding your illness

WRITTEN CONSENT

I, ……, have read the attached information sheet and consent to participate in this study. I consent to the SPECT scans being performed as well as completing the questionnaires and the neuropsychological tests, and participating in the psychotherapy.

I understand that the results will be used for research purposes and that all information will be treated as strictly confidential. My name will not be used in the study.

I understand that participation in this study is voluntary and that I may withdraw at any time of my own accord and that if I do, it will not affect the future care and attention which I will receive, and that I will be referred for alternative treatment if I so wish.

Signed : …….. Subject ……. Date

…….. Researcher ……Date

…… Witness ……Date

APPENDIX

A1 Ethics clearance certificate

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

Division of the Deputy Registrar (Research)

COMMITTEE FOR RESEARCH ON HUMAN SUBJECTS (MEDICAL)
Ref: R14/49 Gordon

CLEARANCE CERTIFICATE **PROTOCOL NUMBER** M 960118

PROJECT	A single-beam positron emission computerised tomography and neuro-psychological study of trichotillomania in terms of Cognitive Behaviour Therapy
INVESTIGATORS	Ms C Gordon
DEPARTMENT	Psychiatry, Medical School
DATE CONSIDERED	960126
DECISION OF THE COMMITTEE *	Approved unconditionally
DATE	960223

CHAIRMAN. .....(Professor P E Cleaton-Jones)

c c Supervisor: Dr M Berk
Dept of Psychiatry, Medical School

==========

DECLARATION OF INVESTIGATOR(S)

To be completed in duplicate and ONE COPY returned to the Secretary at Room 10001, 10th Floor, Senate House, University.

I/we fully understand the conditions under which I am/we are authorized to carry out the abovementioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee.

DATE.1996-03-30.....SIGNATURE .....

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES

A3 The Psychiatric Institute Trichotillomania Scale (PITS)

PSYCHIATRIC INSTITUTE TRICHOTILLOMANIA SCALE (Winchel, et al.)

SCORESHEET

DATE: ___/___/___ SUBJECT INITIALS: _____ SUBJECT NO.: _____ RATER(S) INITIALS: _____

	SITES	SEVERITY	DURATION	RESISTANCE	INTERFERENCE	DISTRESS
0	No sites	No loss	No time	No urge	No interference in functioning	No distress or thoughts about it
1	1 non-scalp site	Negligible loss (can't see loss even if site pointed out)	≤ 5 minutes per day	Always able to resist	Occasionally avoids 1 or 2 activities, creating no or minor inconvenience (e.g., avoids swimming)	Occasionally thinks about it, but isn't very concerned
2	1 scalp site	Mild loss (seen only if area pointed out)	> 5 min but ≤ 15 min	Almost always able to resist	Frequently avoids 1 or more minor activities, creating some inconvenience	Worries occasionally about hair pulling and/or its consequences
3	2 non-scalp sites	Moderate loss (loss visible to observer upon inspection) e.g. thin spots on scalp	> 15 min but ≤ 30 min	Able to resist 3/4 to almost always	Occasionally avoids 1 major life activity (such as work or dating)	Worries daily about hair pulling, but distress is only mild
4	2 sites including scalp	Loss of 50% of hair of brows or lashes or nearly bald spots on scalp or body part	> 30 min but ≤ 1 hr	Able to resist 1/2 to 3/4 the time	Occasionally avoids more than 1 major activity such as work or major social functions	Worries daily about hair pulling, and distress is moderately severe
5	3 sites	Loss of 75% of hair of brows or lashes or medium-sized bald spot on scalp or body part	> 1 hr but ≤ 2 hrs	Able to resist 1/4 to 1/2 the time	Frequently avoids 1 major activity such as work or major social functions (e.g. dating)	Worries occasionally that hair pulling may have major impact on life course (e.g., fears they may never be able to marry)
6	4 sites	Loss of almost all hair of brows or lashes or large areas of baldness on scalp or other body part	> 2 hrs but ≤ 3 hrs	Rarely able to resist	Frequently avoids more than 1 major activity such as work or major social functions	Worries frequently that hair pulling may have major impact on life course (e.g., fears they may never be able to marry)
7	5 or more sites	Total loss of hair of brows or lashes or almost total loss of scalp hair or other body part	> 3 hrs	Never able to resist	Almost always avoids 1 or more major activity such as work or major social functions	Has daily severe distress regarding hair pulling or its consequences

ITEM SCORES: ____ + ____ + ____ + ____ + ____ + ____ + ____ = TOTAL SCORE ____

A4 The Hamilton Anxiety Rating Scale

Hamilton Anxiety Rating Scale

Patient's Name _____ Date of First Report _____

Diagnosis _____ Date of This Report _____

Current Therapy _____

Instructions *This checklist is to assist the physician in evaluating each patient with respect to degree of anxiety and pathological condition. Please fill in the appropriate rating.*

0 *None*
1 *Mild*
2 *Moderate*
3 *Severe*
4 *Severe, grossly disabling*

Item		Rating	Item		Rating
Anxious Mood	Worries, anticipation of the worst, fearful anticipation, irritability.		Somatic (Sensory)	Tinnitus, blurring of vision, hot and cold flushes, feelings of weakness, picking sensation.	
Tension	Feelings of tension, fatigability, startle response, moved to tears easily, trembling, feelings of restlessness, inability to relax.		Cardiovascular Symptoms	Tachycardia, palpitations, pain in chest, throbbing of vessels, fainting feelings, missing beat.	
Fear	Of dark, of strangers, of being left alone, of animals, of traffic, of crowds.		Respiratory Symptoms	Pressure or constriction in chest, choking feelings, sighing, dyspnea.	
Insomnia	Difficulty in falling asleep, broken sleep, unsatisfying sleep and fatigue on waking, dreams, nightmares, night terrors.		Gastrointestinal Symptoms	Difficulty in swallowing, wind, abdominal pain, burning sensations, abdominal fullness, nausea, vomiting, borborygmi, looseness of bowels, loss of weight, constipation.	
Intellectual (Cognitive)	Difficulty in concentration, poor memory.		Genitourinary Symptoms	Frequency of micturition, urgency of micturition, amenorrhea, menorrhagia, development of frigidity, premature ejaculation, loss of libido, impotence.	
Depressed Mood	Loss of interest, lack of pleasure in hobbies, depression, early waking, diurnal swing.		Autonomic Symptoms	Dry mouth, flushing, pallor, tendency to sweat, giddiness, tension headache, raising of hair.	
Behavior at Interview	Fidgeting, restlessness or pacing, tremor of hands, furrowed brow, strained face, sighing or rapid respiration, facial pallor, swallowing, belching, brisk tendon jerks, dilated pupils, exophthalmos.		Somatic (Muscular)	Pains and aches, twitchings, stiffness, myoclonic jerks, grinding of teeth, unsteady voice, increased muscular tone.	
			Total Score		

Source. Reprinted with permission from Hamilton M: The assessment of anxiety states by rating. Br J Med Psychol 32:50–55, 1959.

A5 The Temperament and Character Inventory – 125 (TCI-125)

T C I - 125

In this booklet you will find statements people might use to describe their attitudes, opinions, interests, and other personal feelings.

Each statement can be answered TRUE or FALSE. Read the statement and decide which choice best describes you.

We would like you to fill out this questionnaire on your own using a pencil. When you are finished, please return the questionnaire.

HOW TO FILL OUT THIS QUESTIONNAIRE

To answer you only need to circle either "T" or "F" after each question. Here is an example:

EXAMPLE	TRUE	FALSE
I understand how to fill out this questionnaire.	T	F

(If you understand how to fill out this questionnaire, circle "T" to show that the statement is TRUE.)

****

Read each statement carefully, but don't spend too much time deciding on the answer.

Please answer every statement, even if you are not completely sure of the answer.

Remember there are no right or wrong answers -- just describe your own personal opinions and feelings.

Copyright © 1987, 1992 by C. R. Cloninger

Print your Name: _____Age _____D.O.B.___/___/___

Black ____ White ____ Hispanic ___ Other ___ SEX: M F

Occupation _____ Date _____

		TRUE	FALSE
1.	I often try new things just for fun or thrills, even if most people think it is a waste of time.	T	F
2.	I usually am confident that everything will go well, even in situations that worry most people.	T	F
3.	I often feel that I am the victim of circumstances.	T	F
4.	I can usually accept other people as they are, even when they are very different from me.	T	F
5.	I enjoy getting revenge on people who hurt me.	T	F
6.	Often I feel that my life has little purpose or meaning.	T	F
7.	I like to help find a solution to problems so that everyone comes out ahead.	T	F
8.	I could probably accomplish more than I do, but I don't see the point in pushing myself harder than is necessary to get by.	T	F
9.	I often feel tense and worried in unfamiliar situations, even when others feel there is little to worry about.	T	F
10.	I often do things based on how I feel at the moment without thinking about how they were done in the past.	T	F
11.	I usually do things my own way -- rather than giving in to the wishes of other people.	T	F
12.	I generally don't like people who have different ideas from me.	T	F
13.	I would do almost anything legal in order to become rich and famous, even if I would lose the trust of many old friends.	T	F
14.	I am much more reserved and controlled than most people.	T	F
15.	I like to discuss my experiences and feelings openly with friends instead of keeping them to myself.	T	F

		TRUE	FALSE
16.	I have less energy and get tired more quickly than most people.	T	F
17.	I seldom feel free to choose what I want to do.	T	F
18.	I often consider another person's feelings as much as my own.	T	F
19.	I often avoid meeting strangers because I lack confidence with people I do not know. ...	T	F
20.	I like to please other people as much as I can.	T	F
21.	I often wish that I was smarter than everyone else.	T	F
22.	I am usually so determined that I continued to work long after other people have given up. ..	T	F
23.	I often wait for someone else to provide a solution to my problems.	T	F
24.	I often spend money until I run out of cash or get into debt from using too much credit.	T	F
25.	Often I have unexpected flashes of insight or understanding while relaxing.	T	F
26.	I don't care very much whether other people like me or the way I do things.	T	F
27.	I usually try to get just what I want for myself because it is not possible to satisfy everyone anyway.	T	F
28.	I have no patience with people who don't accept my views.	T	F
29.	I sometimes feel so connected to nature that everything seems to be part of one living organism.	T	F
30.	When I have to meet a group of strangers, I am more shy than most people.	T	F
31.	I am more sentimental than most people.	T	F
32.	I seem to have a "sixth sense" that sometimes allows me to know what is going to happen.	T	F

		TRUE	FALSE
33.	When someone hurts me in any way, I usually try to get even.	T	F
34.	My attitudes are determined largely by influences outside my control.	T	F
35.	I often wish I was stronger than everyone else.	T	F
36.	I like to think about things for a long time before I make a decision.	T	F
37.	I am more hard-working than most people.	T	F
38.	I usually stay calm and secure in situations that most people would find physically dangerous.	T	F
39.	I do not think it is smart to help weak people who cannot help themselves.	T	F
40.	I cannot have any peace of mind if I treat other people unfairly, even if they are unfair to me.	T	F
41.	People will usually tell me how they feel. ...	T	F
42.	Sometimes I have felt like I was part of something with no limits or boundaries in time and space.	T	F
43.	I sometimes feel a spiritual connection to other people that I cannot explain in words.	T	F
44.	I like it when people can do whatever they want without strict rules and regulations.	T	F
45.	I would probably stay relaxed and outgoing when meeting a group of strangers, even if I were told they are unfriendly.	T	F
46.	Usually I am more worried than most people that something might go wrong in the future.	T	F
47.	I usually think about all the facts in detail before I make a decision.	T	F
48.	I often wish I had special powers like Superman.	T	F
49.	Other people control me too much.	T	F

		TRUE	FALSE
50.	I like to share what I have learned with other people.	T	F
51.	I am usually able to get other people to believe me, even when I know that what I am saying is exaggerated or untrue.	T	F
52.	Sometimes I have felt my life was being directed by a spiritual force greater than any human being.	T	F
53.	I have a reputation as someone who is very practical and does not act on emotion.	T	F
54.	I am strongly moved by sentimental appeals (like when asked to help crippled children)...	T	F
55.	I usually push myself harder than most people do because I want to do as well as I possibly can.	T	F
56.	I have so many faults that I don't like myself very much.	T	F
57.	I have too little time to look for long-term solutions for my problems.	T	F
58.	I often cannot deal with problems because I just don't know what to do.	T	F
59.	I prefer spending money rather than saving it.	T	F
60.	I can usually do a good job of stretching the truth to tell a funnier story or to play a joke on someone.	T	F
61.	If I am embarrassed or humiliated, I get over it very quickly.	T	F
62.	It is extremely difficult for me to adjust to changes in my usual way of doing things because I get so tense, tired, or worried.	T	F
63.	I usually demand very good practical reasons before I am willing to change my old ways of doing things.	T	F

	TRUE	FALSE
64. I nearly always stay relaxed and carefree, even when nearly everyone else is fearful.	T	F
65. I find sad songs and movies pretty boring. ...	T	F
66. Circumstances often force me to do things against my will.	T	F
67. I would rather be kind than to get revenge when someone hurts me.	T	F
68. I often become so fascinated with what I'm doing that I get lost in the moment - like I'm detached from time and place.	T	F
69. I do not think I have a real sense of purpose for my life.	T	F
70. I often feel tense and worried in unfamiliar situations, even when others feel there is no danger at all.	T	F
71. I often follow my instincts, hunches, or intuition without thinking through all the details.	T	F
72. Other people often think that I am too independent because I won't do what they want.	T	F
73. I often feel a strong spiritual or emotional connection with all the people around me.	T	F
74. I usually try to imagine myself "in other people's shoes", so I can really understand them.	T	F
75. Principles like fairness and honesty have little role in some aspects of my life.	T	F
76. I am better at saving money than most people.	T	F
77. Even when most people feel it is not important, I often insist on things being done in a strict and orderly way.	T	F
78. I feel very confident and sure of myself in almost all social situations.	T	F

	TRUE	FALSE
79. My friends find it hard to know my feelings because I seldom tell them about my private thoughts.	T	F
80. I like to imagine my enemies suffering.	T	F
81. I am more energetic and tire less quickly than most people.	T	F
82. I often stop what I am doing because I get worried, even when my friends tell me everything will go well.	T	F
83. I often wish I was more powerful than everyone else.	T	F
84. Members of a team rarely get their fair share.	T	F
85. I don't go out of my way to please other people.	T	F
86. I am not shy with strangers at all.	T	F
87. I spend most of my time doing things that seem necessary but not really important to me.	T	F
88. I don't think that religious or ethical principles about what is right and wrong should have much influence in business decisions.	T	F
89. I often try to put aside my own judgments so that I can better understand what other people are experiencing.	T	F
90. Many of my habits make it hard for me to accomplish worthwhile goals.	T	F
91. I have made real personal sacrifices in order to make the world a better place -- like trying to prevent war, poverty and injustice.	T	F
92. I prefer to wait for someone else to take the lead in getting things done.	T	F
93. I usually respect the opinions of others. ...	T	F
94. My behavior is strongly guided by certain goals that I have set for my life.	T	F

	TRUE	FALSE
95. It is usually foolish to promote the success of other people.	T	F
96. I usually like to stay cool and detached from other people.	T	F
97. I am more likely to cry at a sad movie than most people.	T	F
98. I recover more quickly than most people from minor illnesses or stress.	T	F
99. I often break rules and regulations when I think I can get away with it.	T	F
100. I need much more practice in developing good habits before I will be able to trust myself in many tempting situations.	T	F
101. I wish other people didn't talk as much as they do.	T	F
102. Everyone should be treated with dignity and respect, even if they seem to be unimportant or bad.	T	F
103. I like to make quick decisions so I can get on with what has to be done.	T	F
104. I am usually confident that I can easily do things that most people would consider dangerous (such as driving an automobile fast on a wet or icy road).	T	F
105. I like to explore new ways to do things.	T	F
106. I enjoy saving money more than spending it on entertainment or thrills.	T	F
107. I have had personal experiences in which I felt in contact with a divine and wonderful spiritual power.	T	F
108. I have had moments of great joy in which I suddenly had a clear, deep feeling of oneness with all that exists.	T	F
109. Most people seem more resourceful than I am. ..	T	F

		TRUE	FALSE
110.	I often feel like I am a part of the spiritual force on which all life depends.	T	F
111.	Even when I am with friends, I prefer not to "open up" very much.	T	F
112.	I think my natural responses now are usually consistent with my principles and long-term goals.	T	F
113.	I believe that all life depends on some spiritual order or power that cannot be completely explained.	T	F
114.	Often when I look at an ordinary thing, something wonderful happens -- I get the feeling that I am seeing it fresh for the first time.	T	F
115.	I usually feel tense and worried when I have to do something new and unfamiliar.	T	F
116.	I often push myself to the point of exhaustion or try to do more than I really can.	T	F
117.	My will power is too weak to overcome very strong temptations, even if I know I will suffer as a consequence.	T	F
118.	I hate to see anyone suffer.	T	F
119.	If I am feeling upset, I usually feel better around friends than when left alone.	T	F
120.	I wish I were better looking than everyone else.	T	F
121.	I love the blooming of flowers in the spring as much as seeing an old friend again.	T	F
122.	I usually look at a difficult situation as a challenge or opportunity.	T	F
123.	People involved with me have to learn how to do things my way.	T	F
124.	I usually feel much more confident and energetic than most people, even after minor illnesses or stress.	T	F
125.	When nothing new is happening, I usually start looking for something that is thrilling or exciting.	T	F

Revised 10-8-92

TCI Scoring Key

For all scales, positively scored items are not underlined (T = 1, F = 0) and negatively scored items are underlined (T = 0, F = 1).

Novelty Seeking

20 items: 1, 10, 14, 24, 36, 44, 47, 51, 53, 59, 60, 63, 71, 76, 77, 99, 103, 105, 106, 125

Harm Avoidance

20 items: 2, 9, 16, 19, 30, 38, 45, 46, 61, 62, 64, 70, 78, 81, 82, 86, 98, 104, 115, 124

Reward Dependence Scales

15 items: 11, 15, 20, 26, 31, 39, 54, 65, 72, 79, 85, 96, 97, 111, 119

Persistence

5 items: 8, 22, 37, 55, 116

Self-Directiveness

25 items: 3, 6, 17, 21, 23, 34, 35, 48, 49, 56, 57, 58, 66, 69, 83, 87, 90, 92, 94, 100, 109, 112, 117, 120, 122

Cooperativeness

25 items: 4, 5, 7, 12, 13, 18, 27, 28, 33, 40, 41, 50, 67, 74, 75, 80, 84, 88, 89, 93, 95, 101, 102, 118, 123

Self-Transcendence

15 items: 25, 29, 32, 42, 43, 52, 68, 73, 91, 107, 108, 110, 113, 114, 121