

Non-REM Dreaming in Relation to the Cyclic Alternating Pattern: An Exploratory Study

Danyal Wainstein

WNSDAN001

A minor dissertation submitted in partial fulfilment of the requirements for the award of the degree of Master of Arts in Psychological Research

Faculty of the Humanities

University of Cape Town

2013

COMPULSORY DECLARATION

This work has not been previously submitted in whole, or in part, for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation from the work, or works, of other people has been attributed, and has been cited and referenced.

Signature: _____

Date: _____

The financial assistance of the National Research Foundation (NRF) and the AW Mellon Foundation towards this research is hereby acknowledged. Opinions expressed and conclusions arrived at are those of the author and are not necessarily to be attributed to the NRF and the AW Mellon Foundation.

The copyright of this thesis vests in the author. No quotation from it or information derived from it is to be published without full acknowledgement of the source. The thesis is to be used for private study or non-commercial research purposes only.

Published by the University of Cape Town (UCT) in terms of the non-exclusive license granted to UCT by the author.

ACKNOWLEDGEMENTS

Professor Mark Solms, University of Cape Town: The supervisor of this work. I would like to sincerely thank Professor Solms for his invaluable advice and support throughout this project. He has been a constant source of inspiration and encouragement, for which I am very grateful. If I have managed to see further, it is only because I have been standing on the shoulders of a giant.

Dr Susan Malcolm-Smith, University of Cape Town: The co-supervisor of this work. I would like to sincerely thank Dr Malcolm-Smith for her invaluable advice and assistance with the analysis of my results, and for always being available to discuss the details of this project with me. Her keen guidance throughout has no doubt contributed greatly to the successful completion of this work.

Dr Pedro Wolf and Professor Colin Tredoux, University of Cape Town: I would like to thank both of these faculty staff for their instrumental advice regarding my data analysis.

The University of Cape Town Sleep Sciences Team: This project would not have been possible without the resources, both tangible and in terms of training and support, made available to me from this group of very talented young researchers. Thank you.

The AW Mellon Foundation, the NRF, and the University of Cape Town: I would like to thank all of these organisations and institutions for providing financial aid. Without this support the time and dedication required of this project would not have been possible.

TABLE OF CONTENTS

ACKNOWLEDGEMENTS	2
LIST OF FIGURES	8
LIST OF TABLES	9
LIST OF ABBREVIATIONS	10
ABSTRACT	12
INTRODUCTION	13
A Brief History of the Psychophysiology of Dreaming	13
The Phasic-Tonic Model of Sleep and Dreaming	14
The PGO Wave and Dreaming	15
The activation-synthesis model.....	17
REM sleep and Dreaming are Doubly Dissociable Processes	18
A Contemporary Understanding of Phasic and Tonic Sleep Processes	19
The Cyclic Alternating Pattern	20
The CAP Subtypes	21
CAP as a Marker of Sleep Instability.....	25
Mutually Antagonistic Arousal and Sleep Promoting Mechanisms.....	26
The Dynamic Organisation of NREM Sleep	28
Arousal and Anti-Arousal: Natural Elements of Sleep	28
Preliminary Evidence for an Association between Dreaming and CAP	31
Enhanced Brain Reactivity and Dream Recall.....	32
Arousal and Antiarousal Processes and Dreaming	34
Conclusion	35
SPECIFIC AIMS AND HYPOTHESES	37
METHODS	38
Design	38
Participants.....	39
Exclusion Criteria	39
Measurement and Materials	39
Dream Measures	39
The Multidimensional Dream Questionnaire (MDQ).....	40
Total Recall Count	41
Relation to Everyday Experience.....	41
Perceptual-Interaction Rating Scale.....	42

Awakenings	42
Assessing Homogeneity and the Inclusion/Exclusion Criteria.....	43
Wechsler Abbreviated Scale of Intelligence: Vocabulary subtest	43
Mini International Neuropsychiatric Interview.....	44
The Pittsburgh Sleep Quality Index	44
Measuring physiological sleep variables	44
Recording techniques, equipment and scoring.....	44
Sleep Stage and CAP Parameters.....	45
Procedure	46
Screening	46
Sleep Study	46
Sleep night protocol	47
Dream awakenings.....	47
Data Analysis.....	48
Balancing Type I and Type II error rates	48
Dream Measures	49
Sleep Stability in Relation to Dreaming.....	50
CAP Arousal Subtypes and Dreaming	51
RESULTS AND DISCUSSION	52
Participant Descriptives	52
Sleep Macrostructure	52
Dream Measures	54
The Multidimensional Dream Questionnaire	54
Reliability and Validity of the MDQ Subscales.....	55
Objective Dream Measures	56
Reliability Analysis.....	56
Types of dream recall.....	57
Dream Composite: A Global Measure of Dreaming	57
Reliability of the Dream Composite Measure.....	58
The Sleep Cycle and Dreaming	59
Dreaming in Relation to NREM Sleep Stability/ Instability.....	60
Types of Dream Recall	60
Light NREM sleep	60
Deep NREM sleep	61

Repeated Measures Analyses: Global Measures	62
Multivariate Analysis: Dream Quality	63
The Basic Emotions	63
Sleep Parameters for CAP and NCAP Conditions	65
Light NREM Sleep	65
Deep NREM sleep	65
The CAP Arousal Subtypes and Dreaming.....	66
Analysis of the Arousal Subtypes in Unstable NREM Sleep.....	66
CAP Parameters of Interest.....	66
Multilevel Model: Light NREM Sleep	70
Predictor and outcome variables	70
Dream reports.....	71
Calculation of the dependency in the data	71
Testing fixed parts of the RC model	71
The relationship between the A ₁ -A ₂ difference and dreaming.....	72
Sleep Parameters: A ₁ -A ₂ Difference.....	76
Sleep Cycle Distribution	76
Dream Measures: Light NREM.....	77
Global Dreaming.....	77
MDQ Subscales and Bizarreness	78
The basic emotions.	78
Dreaming during CAP Relative to REM and NCAP Sleep.....	80
Majority A ₂ , Light NCAP, and REM Awakenings.....	80
Majority A ₁ , Light NCAP, and REM Awakenings.....	81
Dream content variables.....	81
CAP parameters of interest: Deep NREM sleep	83
Stable NREM sleep, the Sleep Microstructure, and Dreaming	84
General Intercorrelations.....	84
GENERAL DISCUSSION	87
Synchronisation-type Microarousals and Dreaming.....	88
Homeostatic Sleep Processes and Dreaming.....	88
Homeostatic Features of Light NREM Sleep.....	93
Phasic Arousal Activity, Reward Processing, and Dreaming	93
The Dopaminergic Hypothesis of Dreaming	93
The basic emotions in dreaming	95

The reward activation model.....	95
Sleep Instability as a Gateway to Dreaming During SWS.....	96
Local Arousal Activity during Sleep	97
Activation of the Brainstem during SWS	100
A trait-like propensity for dreaming in SWS	100
Final Remarks on A ₁ Activity and Sleep Mentation	101
Desynchronised Microarousals and Dreaming	102
The Paradoxical Disconnection of Dreaming	106
Are Dreams the Guardians of Sleep?	108
Sleep State Misperception: A Consequence of Failed Dreaming?	108
Age-Related Changes in Sleep Fragmentation, Dopamine, and Dreaming	109
Dreaming as a ‘Gateway’ to REM Sleep	110
The Permeability of the Functional Disconnection.....	112
Dreaming: Interest Turned Inwards?	112
NREM Dreaming: Supplementary ML-DA Activation?	113
Additional Observations	115
Dreaming during Stable Sleep.....	115
Time of Night Effects	115
Limitations of the Present Study and Future Research	116
Concluding Remarks.....	117
REFERENCES	119
APPENDIX A	
Scoring Criteria for CAP and Non-CAP.....	141
CAP and non-CAP during non-REM sleep.....	141
CAP during REM sleep	142
APPENDIX B	
The Multidimensional Dream Questionnaire.....	143
APPENDIX C	
The Basic Emotions: Instructions for Participants.....	146
APPENDIX D	
Relation to Everyday Experience.....	148
APPENDIX E	
Perceptual-Interaction Rating Scale (PIRS): Instructions for External Raters	149
APPENDIX F	

Participant Consent and Information Form.....	157
APPENDIX G	
Principal Components Analysis of the Multidimensional Dream Questionnaire.....	161
Reliability Analysis of the Four Component Model.....	170
APPENDIX H	
Reliability Analysis for Types of Dream Recall.....	173
APPENDIX I	
Descriptive Statistics for the Qualitative Dream Measures.....	175
The Basic Emotions.....	176
APPENDIX J	
The Sleep Cycle and Dreaming.....	178
APPENDIX K	
Cyclic Alternating Pattern Scoring: Reliability Analysis.....	182
APPENDIX L	
Individual Dream Reports and Percentage Dreaming.....	184
Missing Data.....	185
APPENDIX M	
Arousal Subtypes Regression Model.....	186

LIST OF FIGURES

<i>Figure 1.</i> A graphic representation of the five macrostructural sleep stages from a single night of sleep.	21
<i>Figure 2.</i> Example of a cyclic alternating pattern (CAP) sequence (top) and non-CAP (bottom) in S2 NREM sleep.	23
<i>Figure 3.</i> Specimens of phase A subtypes: A ₁ (top), A ₂ (middle), and A ₃ (bottom).	24
<i>Figure 4.</i> Sleep hypnogram for a single experimental night.	54
<i>Figure 5.</i> The predicted relationship between dreaming and the A ₁ -A ₂ difference in light NREM sleep.	74
<i>Figure 6.</i> The empirical relationship between dreaming and the A ₁ -A ₂ difference ($n = 52$) in light NREM sleep.	74
<i>Figure 7.</i> Histogram of the distribution of the values for the A ₁ -A ₂ difference in light NREM sleep ($n = 52$).	75
<i>Figure M1.</i> Histogram of the standardised residuals for global dreaming (DC) regressed on the A ₁ -A ₂ difference.	187
<i>Figure M2.</i> Histogram of the standardised residuals for amount of dreaming regressed on the A ₁ -A ₂ difference.	187
<i>Figure M3.</i> Scatter plot of the standardised residuals and predicted values for DC regressed on the A ₁ -A ₂ difference.	188

LIST OF TABLES

Table 1. <i>Macrostructural Sleep Parameters</i>	53
Table 2. <i>Multidimensional Dream Questionnaire – Rotated Components Matrix^a</i>	56
Table 3. <i>Inter-rater Reliability for the Objective Dream Measures^a</i>	57
Table 4. <i>Dream Measures: General Intercorrelations</i>	59
Table 5. <i>Type of Dreaming for Pre-Awakening Conditions</i>	61
Table 6. <i>Quantitative and Qualitative Measures for Stable and Unstable NREM Sleep</i>	64
Table 7. <i>Pooled Averages - All NREM dream reports – Sleep Parameters</i>	66
Table 8. <i>Descriptives – CAP Measures for Light and Deep NREM Sleep</i>	68
Table 9. <i>CAP Measures: General Intercorrelations</i>	69
Table 10. <i>Descriptives for the Random Coefficients Regression Model</i>	70
Table 11. <i>A. OLS Regression for Fixed Effects: Prediction of DC from A₁-A₂ Difference</i>	72
Table 12. <i>Parameters for the A₁-A₂ Differences for Sleep Stage</i>	77
Table 13. <i>Dream Measures in Light NREM Sleep as a Function of the A₁-A₂ Difference</i>	79
Table 14. <i>Comparison of the A₁ and A₂ Majority Groups with Light NCAP and REM Sleep</i> .82	
Table 15. <i>Correlations Deep NREM, CAP Parameters and Dreaming^a</i>	83
Table 16. <i>CAP Parameters in Light NCAP Condition: General Intercorrelations^a</i>	85
Table 17. <i>CAP Parameters in Deep NCAP Condition: General Intercorrelations^a</i>	86
Table G1. <i>Pattern Matrix^a for Analysis 1 of the PCA</i>	163
Table G2. <i>Pattern Matrix^a for Analysis 3 of the PCA</i>	165
Table G3. <i>Structure Matrix for Analysis 3 of the PCA</i>	166
Table G4. <i>Pattern Matrix^a for Analysis 4 of the PCA</i>	168
Table G5. <i>Structure Matrix for Analysis 4 of the PCA</i>	169
Table G6. <i>Multidimensional Dream Questionnaire – Rotated Components Matrix^a</i>	171
Table G7. <i>Structure Matrix for Analysis 5 of the PCA</i>	172
Table H1. <i>Chi-Square Contingency Table for Absolute Inter-Rater Agreement – Types of Dreaming</i>	174
Table I1. <i>Dream Measures: General Intercorrelations</i>	175
Table I2. <i>The Basic Emotions and Overall Dream Recall: Intercorrelations</i>	177
Table J1. <i>Dream Quantity and Quality According to the Sleep Cycle</i>	181
Table K1. <i>Descriptives for CAP variables</i>	183
Table L1. <i>Number of Reports and Percentage Dreaming per Participant for each Preawakening Condition</i>	184

LIST OF ABBREVIATIONS

A₁	Arousal (Subtype) 1
A₂	Arousal (Subtype) 2
A₃	Arousal (Subtype) 3
AAT	Auditory Arousal Thresholds
ARAS	Ascending Reticular Activating System
AS	Ascending Slope
A-S	Activation-Synthesis
ASDA	American Sleep Disorders Association
BOLD	Blood oxygenation level dependent
CAP	Cyclic Alternating Pattern
DC	Dream Composite
DI	Dream Intensity
DS	Descending Slope
ECG	Electrocardiogram
EDF	European Data Format
EEG	Electroencephalogram
EMG	Electromyogram
EOG	Electrooculogram
ERP	Event Related Potential
fMRI	functional Magnetic Resonance Imaging
GABA	Gamma-aminobutyric Acid
ICC	Intraclass Correlation
KC	K-Complex
KMO	Kaiser-Meyer-Olkin
LDT	Laterodorsal Tegmental
LORETA	Low- Resolution Brain Electromagnetic Tomography
MANOVA	Multivariate Analysis of Variance
MEMA	Middle Ear Movement Activity
MINI	Mini International Neuropsychiatric Inventory
ML-DA	Mesolimbic Dopamine
NCAP	Non Cyclic Alternating Pattern
NREM	Non Rapid Eye Movement
OLS	Ordinary Least Squares
PAT	Phases d'Activation Transitoire
PCA	Principle Component Analysis
PFC	Prefrontal Cortex
PGO	Ponto-geniculate-occipital
PIP	Periorbital Integrated Potential
PIRS	Perceptual-Interaction Rating Scale
PPT	Pedunculopontine Tegmentum
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index

RC	Random Coefficients
REM	Rapid Eye Movement
REM-M	Rapid Eye Movement Motility
REM-Q	Rapid Eye Movement Quiescence
S2	Stage 2
S3	Stage 3
S4	Stage 4
SE%	Sleep Efficiency Percentage
SL	Sequence Length
SOL	Sleep Onset Latency
SPT	Sleep Period Time
STW	Sleep-to-Wake
SWS	Slow Wave Sleep
TIB	Time In Bed
TIS	Time-in-Stage
TMS	Transcranial Magnetic Stimulation
TRC	Total Recall Count
TSA	Time Since Arousal
TST	Total Sleep Time
VIQ	Verbal IQ
vmPFC	Ventromesial Prefrontal Cortex
VLPO	Ventrolateral Preoptic
VTA	Ventral Tegmental Area
WASO	Waking After Sleep Onset

ABSTRACT

Dreaming is yet to be studied in relation to sleep microstructure. By endeavouring to study mentation in relation to the finer neurophysiological processes underlying the rhythmicity of the sleep cycles, dream science stands to benefit from the wealth of knowledge of these processes. While relationships between dreaming and certain of these processes have been identified in the literature, a comprehensive study of dreaming in relation to all of the recognized components of the sleep microstructure is completely lacking. With this in mind, the main aim of this study was to examine sleep microstructure in relation to dreaming and determine whether there is any relationship between dream recall and the various types of phasic arousal phenomena during NREM sleep, as systematised within the global framework of the *cyclic alternating pattern* (CAP). A group of healthy participants ($N = 22$), between the ages of 18 and 25 years, was recruited from the University of Cape Town to spend three nonconsecutive nights each in a sleep laboratory; the first night acted as an adaptation night, followed by two experimental nights. In total, 213 dream reports were collected from light and deep non-REM sleep, containing either CAP activity (unstable sleep) or no CAP activity (stable sleep); and additional 54 dream reports were collected from REM sleep. The awakenings from unstable sleep were further divided into three groups according to whether the last three minutes of sleep preceding each experimental awakening comprised: i) mostly the CAP A₁ arousal subtype; ii) mostly the A₂ arousal subtype; or iii) both A₁ and A₂ arousal subtypes. The main results showed that i) unstable deep NREM sleep yielded significantly more dream recall than stable deep NREM sleep; ii) light NREM sleep comprised mainly of A₂ arousal activity in the three minutes before waking yielded the most dream recall, on average, from NREM sleep; iii) A₁ arousal activity in light NREM sleep reduces dream recall; and that iv) the A₃ subtype had no consistent relationship with dream recall. These results indicate that phasic arousal activity is able to influence dream recall at certain points during the night, and that the various arousal subtypes are differentially related to dream recall. The implications of these findings for furthering our understanding the dream process are discussed.

Key words: antiarousal, arousal, cyclic alternating pattern, dreaming, NREM sleep, phasic activity, sleep microstructure.

INTRODUCTION

In contrast to the intimate relationship established between dreaming and rapid eye movement sleep (REM) in the 1950s, success determining the psychophysiological correlates of dreaming outside of REM has been variable and largely inconclusive. While the psychophysiological studies of the 1960s and 1970s led to a thorough investigation of finer within-sleep stage events in relation to dreaming, perhaps due to earlier biases and a lack of development in the neurophysiology of sleep, these initial studies focused almost entirely on presumed analogues of REM related processes in non-REM (NREM) sleep. In the last 30 years or so substantial developments in understanding and quantifying microstructural sleep processes have come to pass; NREM sleep is no longer considered to be a state of passive deactivation, but instead, is now understood to involve numerous dynamic processes which constantly interact to shape the typical sleep architecture. These dynamic processes comprise a range of arousal-related phenomena that essentially allow sleep to be both reversible and adaptable, and to allow its continuity when threatened by disruption. However, the earlier psychophysiological traditions did not endure to embrace these more recent developments in sleep microstructure, many of which are not directly related to REM sleep. With this in mind, this introduction will briefly look at the history of the early psychophysiological dream studies, after which a more contemporary understanding of transient within-sleep stage events will be reviewed. Various lines of evidence are considered that suggest that sleep microstructure may not only be valuable, but necessary, to understanding the occurrence of dreaming outside of REM sleep.

A Brief History of the Psychophysiology of Dreaming

Any review of the psychophysiology of dreaming must start with the discoveries of Aserinsky and Kleitman (1953, 1955), as they were the first to show that sleep comprises cyclical periods of activation characterised by a desynchronised electroencephalogram (EEG), the appearance of rapid conjugate eye movements, and an increased variability in autonomic functions. Following this discovery, interest was immediately directed to whether these periods of activation were accompanied by an equally active psychological state. This was indeed found to be the case. These periods of activation, later named REM (rapid eye movement) sleep, yielded detailed and vivid dream recall in 74% of awakenings, while only 9% produced such mental activity outside of these periods, during NREM sleep (Aserinsky & Kleitman, 1953). A later study by Dement and Kleitman (1957) replicated these results, with 80% of the awakenings from REM sleep yielding dream reports, compared with only 7% from NREM sleep.

Following these initial studies, Foulkes (1962) was the first to seriously question the almost exclusive assignment of dreams to REM sleep. Having noticed the discrepancy in recall figures for REM and NREM sleep in previous studies, Foulkes aimed to study the *qualitative* differences in dream content from different stages of sleep, in addition to the overall quantitative recall. The major methodological difference between his studies and those conducted previously was that subjects were asked to report *everything that had been passing through their mind* before waking, rather than to report whether or not they were *dreaming*. Using this approach, a substantial amount of mentation (74%) was found to accompany NREM sleep.

Over the course of the next decade, Foulkes's (1962) findings were replicated by a number of other studies (Foulkes & Vogel, 1965; Goodenough, Lewis, Shapiro & Sleser, 1965; Kamiya, 1961; Pivik & Foulkes, 1968; Rechtschaffen, 1973; Rechtschaffen, Verdone and Wheaton, 1963). The overall incidence of NREM mentation reported in the literature toward the end of the 1960s ranged from 23 – 74%. While NREM dreaming was generally found to be more thoughtlike, less dreamlike, less vivid, less emotional, less bizarre, and to have less active participation, at least 7 – 27% of it was comparable to, or even indistinguishable from, REM dreaming (Kamiya, 1961; Monroe, Rechtschaffen, Foulkes, & Jensen, 1965). Therefore, despite substantial differences in dream recall from REM and NREM sleep, this physiological dichotomy ultimately failed to account for similar types of mentation arising from sleep states characterised by vastly different EEG patterns. Researchers consequently began searching for physiological correlates of NREM dreaming that would allow this phenomenon to be better understood.

The Phasic-Tonic Model of Sleep and Dreaming

Originally, the *three-state model* considered NREM sleep, REM sleep and waking to be entirely discrete and homogenous states, each characterised by a distinctive pattern of electrophysiological and peripheral autonomic activity (Snyder, 1966). A few seminal articles in the 1960s altered this perception and led to the notion that the sleep stages were in fact heterogeneous. In particular, Moruzzi (1963) proposed that REM sleep comprised physiological features that were both tonic (longer lasting) and phasic (intermittent and intrusive) in nature¹. The dichotomization of the classic sleep stages according to *within-*

¹ Tonic components of REM sleep included a background of Theta waves of 5Hz (cycles per second) and a lack of tonus of the antigravity muscles. Alternatively, phasic phenomena occurred against this tonic background physiology and included the occurrence of (a) transient fluctuations in autonomic activity, (b) the presence of myoclonic twitches and fine motor activity, and (c) ponto-geniculo-occipital (PGO) spikes (Moruzzi, 1963).

stage events led to a revolutionised understanding of sleep; rather than merely a descriptive difference, Moruzzi (1963, 1964) postulated that the difference between REM and NREM sleep along tonic dimensions was one of degree and not of kind, and that the episodic intrusions of phasic events were essentially different from the tonic background upon which they were imposed (Pivik, 1991). Furthermore, based on the fact that widespread phasic activity (eye movements, muscle twitches, pupillary dilation, and other peripheral autonomic activity) tended to occur in clustered bursts, Moruzzi predicted that all phasic activity originated from a central generator. This hypothesis held parsimonious appeal, as it predicted that phasic events occurring both within and outside of REM sleep originated from the same physiological system. The search for this 'single generator' had previously been isolated to brainstem structures, and the ponto-geniculo-occipital (PGO) spike was considered to be the phenomenon most representative of this centralised activity.

Not surprisingly, the *tonic-phasic model* was rapidly applied to theories of dreaming and mental activity during sleep. In 1967, Aserinsky was the first to hypothesize that REM mentation may differ along phasic-tonic dimensions. He proposed that mental activity during periods of ocular motility (REM-M) would be fundamentally different to mentation from periods of ocular quiescence (REM-Q). Therefore, Aserinsky suggested that, like all other phasic phenomena, dreaming may also be related to PGO spike activity.

The PGO Wave and Dreaming

Ponto-geniculate-occipital waves are bursts of very high amplitude (200-300 μ V) spike potentials, of about 100ms duration, that originate in the pons and travel through the lateral geniculate body to the occipital cortex (Grosser & Siegal, 1971; Jouvet, Michel, & Courjon, 1959). Since it was discovered that the majority of PGO spikes occur in conjunction with eye movement activity during REM sleep in the cat (Jouvet et al., 1959), this phenomenon was of interest to dream researchers. Putting aside for the moment the intimate relationship between dreaming and REM sleep (where eye movement activity predominates), the PGO wave held special appeal for dream research because of its prominent appearance in the visual system (Michel, Rechtschaffen, & Vimont-Vicary, 1963)—consistent with the primarily visual nature of dreaming—as well the general increase in firing rates in many areas of the brain in conjunction with spike activity, which was congruent with the prediction that the brain is probably very active during dreaming (Rechtschaffen, 1973). In animal

Later on, Karacan, Goodenough, Shapiro, & Starker (1966) showed penile tumescence to be another tonic feature of REM sleep.

studies, isolated bursts of PGO activity were shown to occur outside of REM sleep, which implied that this phasic activity may also be responsible for NREM dreaming. Ultimately, PGO spike activity offered researchers the opportunity to account for dreaming across the sleep stages, and therein the psychophysiological parsimony that so many had sought.

However, investigations of hallucinatory quality, emotionality, quality of mentation, felt bodily experience and depth of sleep all failed to successfully differentiate phasic from tonic dream reports in REM and NREM sleep (Molinari & Foulkes, 1969; Pivik, 1970; Pivik, Halper, & Dement, 1969). Phasic REM dreams were, however, found to contain more hostility (Pivik, 1970; Watson, 1972), movement (Bosinelli, Cicogna, & Molinari, 1974), self-participation (Bosinelli et al., 1974; Molinari & Foulkes, 1969; Pivik, 1970), and overall dream intensity (Dement & Wolpert, 1958; Molinari & Foulkes, 1969) than dream reports from periods of ocular quiescence. On the other hand, tonic dream reports from REM sleep were reported to be more thoughtlike (Molinari & Foulkes, 1969) but this study failed to be adequately replicated (Foulkes & Pope, 1973; Medoff & Foulkes, 1971).

Additional indices of PGO activity used to try and distinguish phasic from tonic portions of REM and NREM sleep were *periorbital integrated potentials*, or PIPs,² and *middle ear muscle activity* (MEMA). These events were found to correlate with PGO activity in the cat, and on this basis were extrapolated as an index of PGO activity in the human. Awakenings following PIPs produced mentation that was more bizarre than periods of REM sleep not occupied by PIPs (Watson, 1972). However, Foulkes and Pope (1973) failed to find significantly more bizarreness in relation to eye movement activity during REM sleep, despite the posited concurrence of eye movements with PIPs. Similarly, MEMA activity failed to differentiate dream reports from both REM and NREM sleep along phasic and tonic dimensions, with results indicating that auditory imagery, emotionality, hallucinosis and clouding were *not* significantly different for reports following MEMA activity compared with reports following no MEMA activity. The only difference found along phasic-tonic dimensions was a nonsignificant trend towards more bizarreness and distortion from MEMA dream reports (Ogilvie, Hunt, Sawicki, Samahalskyi, 1982).

One of the most extensive studies of supposed PGO activity in relation to mentation during sleep was that undertaken by Pivik (1970), looking at the transient inhibition

² Essentially, PIPs are spike potentials recorded from the extra-ocular musculature during sleep (using surface electrodes); these potentials were used as an index because they were found to coincide with PGO activity in the cat (Gadea-Ciria & Jouvet, 1971; Michel et al., 1963; Rechtschaffen, Michel, & Metz, 1971).

throughout sleep of the *spinal H-reflex*. Suppression of the spinal H-reflex in the human was shown to be temporally correlated with indices of inferred PGO activity, such as bursts of eye movement activity, autonomic changes, and phasic EMG inhibition in REM sleep (Hodes & Dement, 1964; Pivik & Dement, 1970). Outside of REM sleep, animal studies showed that PGO spikes occurred in conjunction with phasic suppression of the H-reflex, but not of eye movement activity (Pivik & Dement, 1970). By studying H-reflex suppressions, Pivik aimed to study phasic PGO activity, both during and outside of REM sleep, to determine the extent to which this activity was responsible for dreaming.

Overall, Pivik (1970) found that neither Dreamlike Fantasy ratings nor visual imagery³ differed significantly for phasic and tonic conditions. Of fifteen dream content variables, only auditory imagery and hostility were found to be significantly increased as a function of phasic spinal H-reflex suppression. Based on these findings, Pivik concluded that the evidence provided little support for the hypothesis that phasic activity facilitates dreaming; “it is clear...from the measures that have been taken, that phasic activity is not the determinant of sleep mentation per se. Phasic activity does appear to facilitate recall...especially during slow wave sleep...but recall is relatively profuse under tonic conditions” (p. 244). Nevertheless, only a few years later a theory postulating that the PGO spike is the central determinant of dreaming re-emerged.

The activation-synthesis model. In the mid-1970s the neuromodulatory systems subserving the various states of consciousness were partially revealed. It was shown that the waking state is both cholinergically and aminergically (serotonin and noradrenaline) modulated; the initiation and development of NREM sleep is marked by a progressive decrease in the release of all three neuromodulators (including serotonin and noradrenaline), eventually culminating in the complete absence of aminergic firing, which in conjunction with a simultaneous increase in cholinergic activity results in the cholinergically modulated REM state.⁴ This aminergic-cholinergic interplay was termed the *reciprocal interaction model* (McCarley & Hobson, 1975). Furthermore, the cholinergic disinhibition responsible for REM sleep was considered to be the basis of PGO waves, and therefore, unsurprisingly,

³ According to the “PGO Activity = Dreaming” hypothesis, this finding is contradictory, considering the prevalence of PGO activity in the visual system.

⁴ Note that the mechanisms responsible for the sleep-wake and NREM-REM cycles have been developed substantially since this work (see the later section on mutually antagonistic arousal and sleep promoting mechanisms).

Hobson and McCarley (1977) shortly after introduced a theory of dreaming grounded in the principles of the reciprocal-interaction model.

Unlike other theories of PGO activity and dreaming, the *activation-synthesis model* (A-S) hypothesised that dreaming was an epiphenomenon of cholinergically modulated REM processes. According to the A-S model, the forebrain is *activated* by cholinergic brainstem mechanisms (responsible for REM sleep) that cause meaningless representations (such as thoughts, feelings and images) to be passively *synthesised*. In this way, the forebrain was said to make “the best of a bad job in producing even partially coherent dream imagery from the relatively noisy signals sent up from the brainstem” (Hobson & McCarley, 1977, p. 1347).

In spite of inconclusive evidence in favour of PGO determined dream activity, the A-S model was widely accepted. Nonetheless, many researchers continued to search for an explanation that accommodated the complex psychological organisation of the dream. Many also remained unconvinced by the claim that the involvement of PGO activity in dream genesis meant that the dream involved no higher cognitive processes. Regardless of this resistance to the reductionism of the A-S model, however, it would take another 20 years before Hobson and McCarley’s (1977) model of dreaming was seriously challenged.

REM sleep and Dreaming are Doubly Dissociable Processes

Human lesion studies have informed most of our knowledge of the neural correlates of dreaming by documenting the effects of brain injury on the dream process. In the most comprehensive clinico-anatomical lesion study to date, investigating the effects of brain injury on dreaming in 361 patients, damage to two brain regions were consistently found to be related to dream loss: the parieto-temporo-occipital (PTO) junction and the white matter of the ventromesial quadrant of the frontal lobes (Solms, 1997). Furthermore, a number of case studies have confirmed that dream cessation following posterior PTO lesions can occur concurrently with intact REM sleep (Benson & Greenberg, 1969; Bischof & Bassetti, 2004; Brown, 1972; Jus et al., 1973, Poza & Martí Massó, 2006); while patients with lesions to the pontine brainstem, resulting in the elimination of REM sleep, still report dreams (Solms, 1997). This constitutes substantial evidence that dreaming and REM sleep are *doubly dissociable* states (Solms, 2000). Additionally, the fact that dreaming ceases across *all sleep stages* after damage to the ventromesial frontal lobes or the PTO junction indicates that these regions are critical to dreaming during both REM and NREM sleep. Thus, while the physiological differences across sleep states may make secondary contributions to the dream process, the core dream process appears to involve critical frontal and posterior cortical regions regardless of the stage of sleep.

As a result of these findings the reductionist “REM equals dreaming” paradigm again lost credence. With the acceptance of NREM dreaming as a genuine phenomenon, underpinned by the same basic neurophysiological processes as REM dreaming, attention has once again shifted towards trying to understand how similar phenomenological states of consciousness can emerge from vastly different states of EEG-defined brain activity. Advances in sleep science have permitted the documentation of within-state events that exceed the narrow applications of the previous phasic-tonic model. Applying this knowledge to dream science may potentially provide a novel means of understanding dreaming as it relates to the sleep cycle.

A Contemporary Understanding of Phasic and Tonic Sleep Processes

Unlike the earlier psychophysiological studies that focused almost entirely on analogues of PGO activity, contemporary sleep science recognises that NREM sleep is laden with various other types of phasic phenomena; these *microstructural* events are involved in the development and maintenance of the widely recognised *macrostructural* sleep architecture (Halász, 1998; Figure 1). Indeed, behind the artificially distinct sleep stages that comprise the hypnogram is an ever changing dynamic process that is governed by ongoing fluctuations in sleep depth; therefore, paradoxically, one of the central characteristics of sleep is a rich spectrum of different degrees of arousal. Importantly, these continuous fluctuations in arousal are largely governed by input-dependent phasic changes that exist within a hierarchy—i.e., according to their level of arousing influence on sleep (Halász, 1998). These distinct phasic events represent sudden changes in ongoing activity, and are superimposed on the macrostructural sleep stages (tonic background). Furthermore, these events also occur spontaneously and can be elicited in the same form via a number of different (multimodal) stimuli (Halász, Terzano, Parrino, Bódizs, 2004).

The most elementary form of phasic arousal activity during sleep is the *K-complex* (KC). This phasic event provides substantive information processing while simultaneously inducing a level-setting sleep maintenance function, and therefore performs a dual purpose (Halász, 1998; Halász, 2005; Jahnke et al., 2012). Additionally, *les phases d'activation transitoire spontanées* (phases of spontaneous transitory activation) or PAT (Schieber, Muzet, & Ferriere, 1971) are more complex events that are further up in the hierarchy. These are the same phenomena that are now commonly referred to as *microarousals*. Both the original definition of PAT, as well as the more contemporary description of microarousals by the American Sleep Disorders Association (ASDA), recognise such events as rapid modifications of EEG frequency (shifts towards theta, alpha and/ or frequencies higher than

16 Hz) that are accompanied by an increase in EMG activity, cardiac variability, and body movements (Atlas Task Force, 1992). Integral to the scoring of these phasic events is arousal during sleep *without awakening* (Atlas Task Force, 1992; Schieber et al., 1971).

Microarousals (or PAT) are therefore a more intensive response during sleep which causes a long lasting change in the EEG, in the direction of higher activation (Halász, 1998).

However, ASDA consider arousal phenomena during sleep to be markers of sleep disruption, and as a harmful, detrimental and unnatural element of sleep. This limited conception of phasic arousal activity severely restricts a full understanding of these multifaceted, dynamic processes. Other schools of sleep research have, however, identified a range of microarousals, each classified according to its morphological features and reactive properties during sleep (Terzano, Mancina, Salati, Costani, Decembrino, & Parrino, 1985; Terzano, Parrino, & Spaggiari, 1988).

The Cyclic Alternating Pattern

The interrelationships and dynamic interplay between the various microarousal subtypes, as well as other phasic phenomena (i.e., sleep spindles), during sleep have been coherently conceptualised within the global framework of the *cyclic alternating pattern* (CAP; Terzano et al., 1985). The cyclic alternating pattern can basically be defined as “periodic EEG activity of *non-REM* sleep... characterized by sequences of transient electrocortical events that are distinct from background EEG activity and recur at up to 1 min intervals” (Terzano et al., 2001, emphasis added). These transient events, occurring within 60 seconds of one another, are referred to as *CAP cycles* (Figure 2). A CAP cycle is made up of two phases: Phase A (“activation”) and Phase B (“background”). A *CAP sequence* is comprised of at least two CAP cycles and can, theoretically, include a limitless number of cycles; however, the mean number of CAP cycles in a sequence is 5.6. A sequence lasts 2 minutes and 33 seconds on average (Smerieri, Parrino, Agosti, Ferri, & Terzano, 2007).⁵ Considering that there are on average around 44 CAP sequences per night of healthy human sleep, accounting for approximately 110 minutes (32%) of NREM sleep, CAP sequences make up a substantial portion of the sleep state (Smerieri et al., 2007). Moreover, the A phase of the CAP cycle can be divided into three different subtypes, each of which, it has recently been proposed, contribute to the dynamic organisation of sleep via a different mechanism (Halász & Bódizs, 2013).

⁵All 24 participants in Smerieri et al.’s (2007) study also displayed CAP sequences lasting 5 minutes and 30 seconds. It is also common for CAP sequences as short as < 1 minute to occur, and sequences as long as 15-18 minutes are said to be experienced by roughly 1 in 12 people per night.

Figure 1. The Sleep Hypnogram

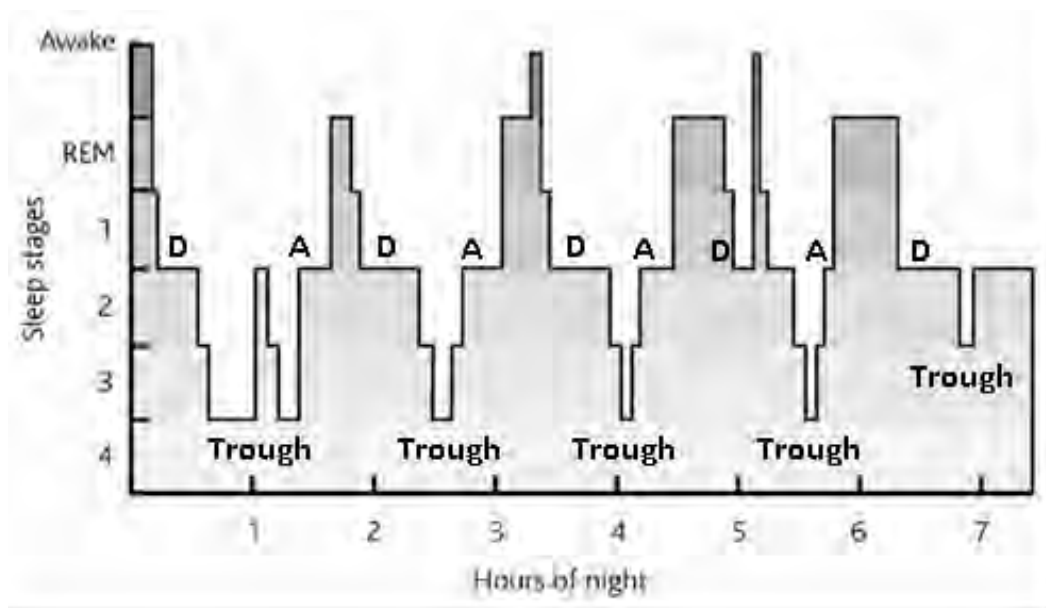


Figure 1. A graphic representation of the five macrostructural sleep stages from a single night of sleep. The five traditionally recognised sleep stages (the REM stage and the four NREM stages) are together referred to as the sleep *macrostructure*; through the visual scoring of the EEG record, the magnitude and distribution of these conventional sleep stages (i.e., the sleep hypnogram, as above) is constructed. Each night of sleep is comprised of between four and six sleep cycles; each cycle lasts approximately 100 minutes, and concludes with the end of the REM period. As the night progresses the troughs of the cycles get shallower and shorter (Rama, Cho, & Kushida, 2005). DS = Descending Slope; AS = Ascending Slope; REM = Rapid Eye Movement Sleep. Rechtschaffen, A., & Siegel, J. (2000). Sleep and dreaming. In E. R. Kandel, J. H. Schwartz & T. M. Jessell (Eds.), *Principles of neuroscience*, 4th Ed (pp. 936–947). USA: McGraw-Hill.

The CAP Subtypes

The A phase of the CAP cycle can be divided into three different subtypes—namely A₁, A₂ and A₃ (Terzano et al., 1985; Terzano et al., 1988). In terms of their morphology, the A₁ subtype is comprised of slower, synchronised EEG rhythms, while A₃ is comprised of faster, desynchronised rhythms. The A₂ subtype is a compromise between the A₁ and A₃ subtypes, with synchronised A₁ activity giving way to faster desynchronised A₃ activity the vast majority of the time (Terzano, & Parrino, 2000; Figure 3). Importantly, the A₂ and A₃ subtypes are akin to the widely recognised *microarousals*; 87% of traditionally scored microarousals were found to coincide with the A₂ and A₃ subtypes (Parrino, Smerieri, Rossi,

& Terzano, 2001). However, ASDA do not recognise the synchronised EEG events (Delta bursts and KCs) that comprise the CAP A₁ subtype as arousal events. Nevertheless, there is ample evidence for concomitant somatosensory and autonomic perturbations, characteristic of arousal activity, accompanying synchronisation type arousals that characterise the CAP A₁ subtype (Halász, 1998; Halász et al., 2004); while these autonomic activities are usually much less apparent than those seen in conjunction with the more traditional type of arousals, they may be equally effective (Parrino, Ferri, Zucconi, & Fanfulla, 2009). Furthermore, where microarousals are conventionally considered to be independent from one another and to occur spontaneously, CAP has shown that these same events have a sustained oscillatory nature (Terzano et al., 1988; Terzano & Parrino, 2000). Importantly, a single CAP sequence can contain mixed CAP arousal subtypes (Terzano et al., 2001; see Appendix A).

The arousal subtypes can further be considered as distinct events for several reasons. First, the synchronised and desynchronised types are generally prevalent during different portions of the sleep cycle. The A₁ (synchronised) subtype prevails during the descending slope and the trough of the sleep cycle, especially in the first few hours of the night when the homeostatic pressure is at its strongest, while the A₂ and A₃ (desynchronised) subtypes are more prevalent during the ascending slope and in anticipation of the REM state (Terzano & Parrino, 2000).⁶ Consequently, there is a ‘time of night’ effect on the distribution of the subtypes, with more A₁ arousals in the first half of the night. This purposeful distribution in accordance with the different portions of the sleep cycle and time of night is an indication that these phasic events may be crucially involved with the ultradian organisation of sleep (Terzano et al., 2005); this point will be reviewed in more detail later on, as it is fundamental to the relevance of considering dreaming in relation to the sleep microstructure.

⁶ A sleep cycle lasts 90-100 minutes on average, and is concluded with the end of a REM period (Figure 1). Each night of healthy adult sleep is comprised of four to six sleep cycles. The period of sleep immediately after Stage REM (except in the case of the first cycle) is the descending slope, the slowest or most synchronised middle portion of the cycle is the trough, and the shorter and steeper slope preceding REM is the ascending slope (Rama et al., 2005).

Figure 2. A Graphic Display of CAP and Non-CAP Sleep

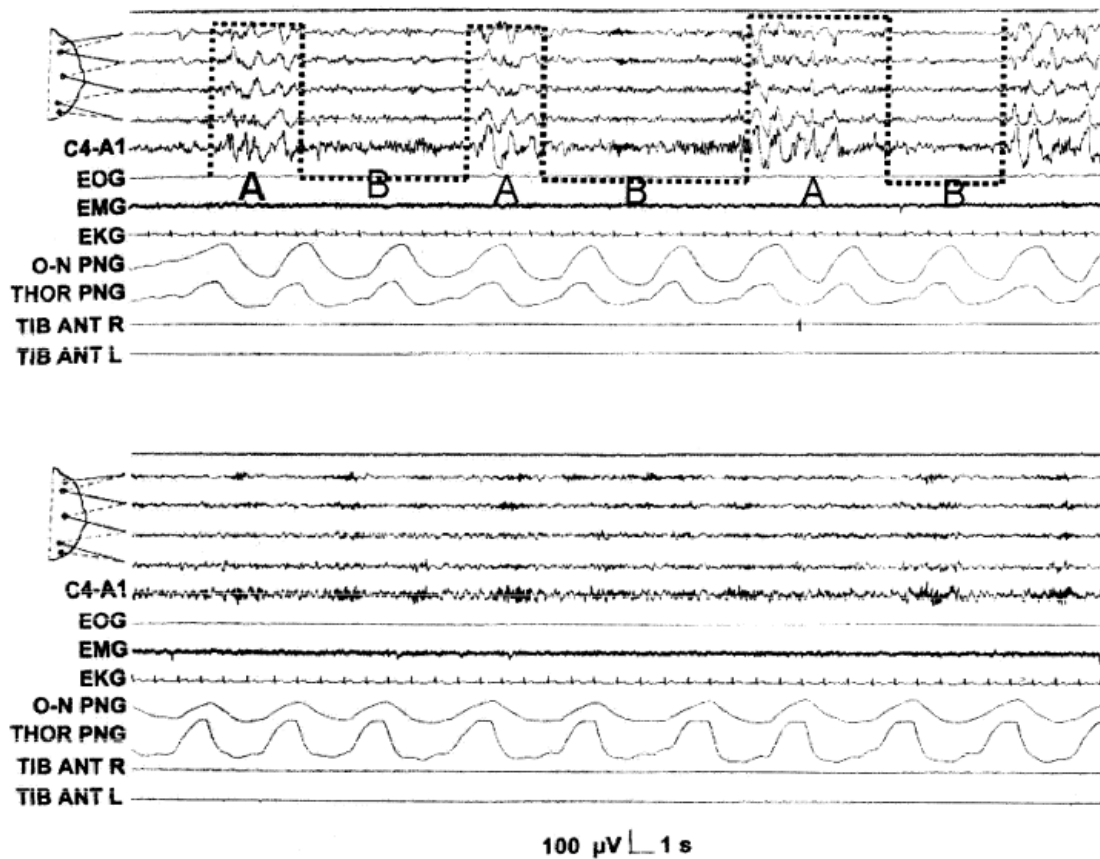


Figure 2. Example of a cyclic alternating pattern (CAP) sequence (top) and non-CAP (bottom) in S2 NREM sleep. Notice that CAP occurs as a spontaneous phenomenon in the absence of any respiratory or muscle abnormality. EOG = electrooculogram—eye movements; EMG = electromyogram—chin muscle; EKG = electrocardiogram—heart rate; O-N PNG, oro-nasal flow; THOR PNG, thoracic effort; TIB ANT R = right anterior tibialis muscle; TIB ANT L = left anterior tibialis muscle. Halász et al. (2004). *The Nature of Arousal in Sleep. Journal of Sleep Research*, 13(1), 1-23.

Figure 3. The CAP Arousal Subtypes

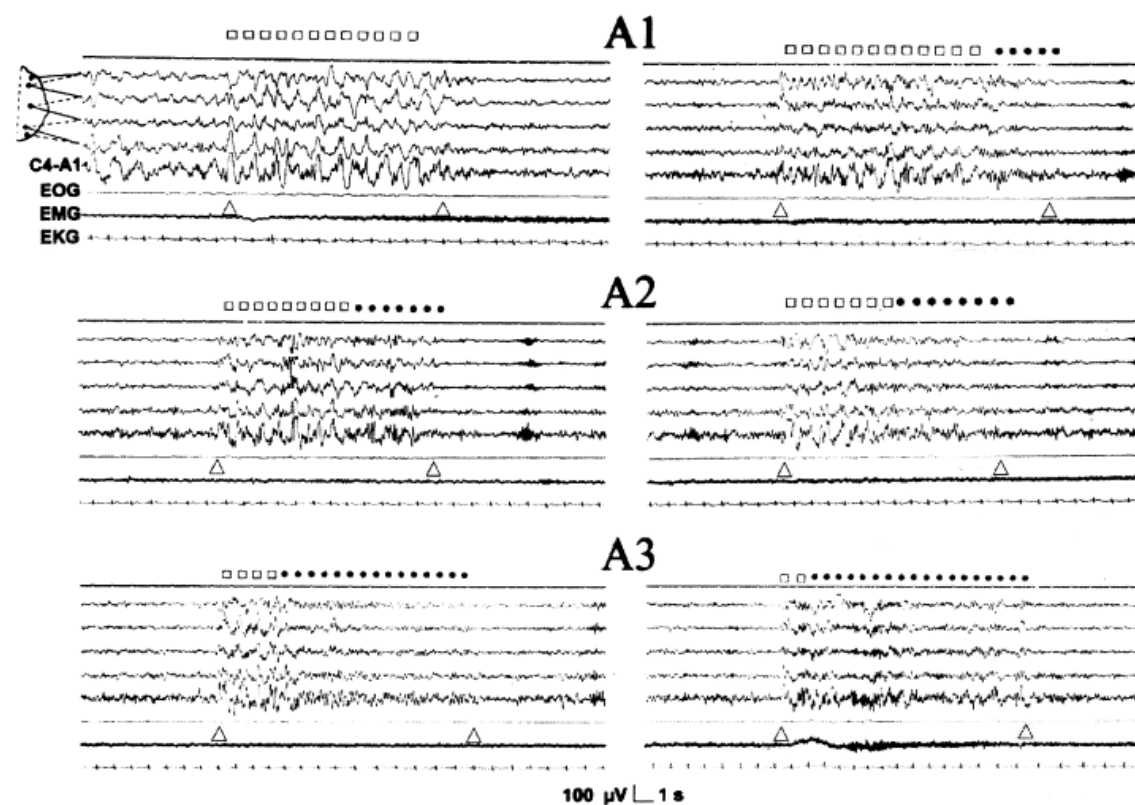


Figure 3. Specimens of phase A subtypes: A₁ (top), A₂ (middle), and A₃ (bottom). White boxes indicate electroencephalography (EEG) patterns in synchronization, black dots indicate EEG patterns in desynchronisation. Notice the progressive shift from dominant EEG synchrony to dominant EEG desynchrony from subtypes A₁ to subtypes A₂ and A₃. EOG = Electro-oculogram, EMG = Electromyogram, EKG = Electrocardiogram. Halász et al. (2004). *The Nature of Arousal in Sleep. Journal of Sleep Research*, 13(1), 1-23.

Second, the A subtypes have distinctly different cortical generators.⁷ Low-resolution brain electromagnetic tomography (LORETA) functional imaging⁸ based on the spectral

⁷ While the cortex is referred to here as the *generator* of the different EEG frequencies, it is important to note that this does not preclude the involvement of more subcortical brain regions in producing the different EEG frequencies. Rather, it implies only that the electrophysiological signals in the cortex are the measured end-point of these multiple sub-systems (Ferri et al., 2005).

⁸ LORETA makes use of 3D linear solutions to predict EEG spectral power (averaged EEG waveforms) within a three-shell spherical head model that includes the scalp, skull and brain compartments. Specifically, the brain

analysis of the CAP A subtypes has shown that A₁ (0.25 – 2.5 Hz) predominantly originates in the anterior frontal regions, while A₃ (9 – 12 Hz) is predominantly produced by more posterior parietal-occipital brain regions (Ferri, Bruni, Miano, & Terzano, 2005). In subtype A₂ there is a compromise between the slow and fast subtypes, and a resulting phasic arousal that begins in the frontal regions and moves toward the posterior parietal-temporal regions in an anteroposterior motion. As 90% of all CAP A₂, and even occasionally A₃, subtypes are preceded by an initial A₁ event, the topographic distribution almost always moves in an anteroposterior direction (Terzano, & Parrino, 2000).

CAP as a Marker of Sleep Instability

Periods of NREM sleep marked by CAP sequences exhibit a stereotyped reactivity, including a lack of habituation to external stimuli, and show a continuing variation in sleep depth (Terzano et al., 2002). In particular, an arousing stimulus applied during the B phase of a CAP sequence will immediately give rise to an A phase, whereas the reverse does not occur (Terzano & Parrino, 1991). Conversely, an arousing stimulus received during a period of NREM sleep not marked by any CAP sequences results in a generally brief, hypersynchronised EEG response that proceeds towards habituation. However, a robust or sustained stimulus during non-CAP sleep (below waking threshold), results in the immediate appearance of a CAP sequence that displays the same reactive character and morphology of spontaneous CAP sequences (Terzano, Parrino, Fioriti, Orofiamma, & Depoortere, 1990). The evoked CAP sequence may introduce a lightening of sleep depth or may continue as a damping oscillation before the recovery of NREM sleep without CAP (Parrino, Ferri, Bruni, & Terzano, 2012). Therefore, CAP can be considered a marker of sleep instability—“the appearance of CAP sequences represents an arousal control mechanism. They reflect all of the arousing influences and set into motion an oscillatory level-setting system...they provide a flexible adaptation for the system to defend against perturbations” (Halász & Bódizs, 2013, p. 24). Furthermore, CAP sequences reflect arousal instability in a higher duration range than individual microarousals (Halász, 1998).

In describing the potential relevance of the sleep microstructure (i.e., the cyclic alternating pattern) to dreaming, it is firstly important to understand how these phasic phenomena contribute to the dynamic organisation of sleep; this is best explained in light of the mechanisms largely responsible for regulating the sleep-wake and NREM-REM cycles.

compartment is only able to display EEG signals originating in the cortical grey matter and the hippocampus (Ferri et al., 2005).

Mutually Antagonistic Arousal and Sleep Promoting Mechanisms

The brain circuitry responsible for diffuse cortical arousal and waking was first identified by Moruzzi and Magoun (1949) as the *ascending reticular activating system* (ARAS). The ARAS was shown to begin in the rostral pons and to extend through the midbrain reticular formation. More recently, the basic neurocircuitry responsible for diffuse cortical arousal has been extended and refined (e.g., to include the posterior lateral hypothalamus), and it is now recognised that the ascending arousal system is comprised of two major branches (Saper, 1987; Saper, Chou, & Scammell, 2001). First, an ascending pathway originating in the upper brainstem, responsible for activating the thalamic relay neurons, is crucial for regulating thalamic activity. Specifically, a pair of acetylcholine-producing cell groups, the pedunculopontine and laterodorsal tegmental nuclei (PPT/LDT), provide a major source of input to the thalamic relay neurons and the reticular nucleus of the thalamus (Jones, 2003; Saper, Thomas, Scammell, & Lu, 2005); the input of the PPT/LDT to the reticular nucleus acts as a gating mechanism that can block or permit transmission between the thalamic-relay nuclei and the cerebral cortex, which is important for the mediation of wakefulness and sleep (Steriade, Nunez, & Amzica, 1993). The PPT/LDT fire most rapidly during wakefulness and REM sleep, and slow down during NREM sleep (Hobson & McCarley, 1975; Massaquoi & McCarley, 1992).

The second branch of the ascending arousal system bypasses the thalamus and instead activates neurons in the lateral hypothalamic area, basal forebrain, and the cerebral cortex (Jones, 2003; Saper et al., 2001). This pathway originates in the monoaminergic neurons of the upper brainstem and caudal hypothalamus, and includes the noradrenergic locus coeruleus, serotonergic dorsal raphe and median raphe nuclei, the dopaminergic ventral periaqueductal gray matter, and histaminergic tuberomammillary neurons (Saper, 1987). This arousal pathway activates the cerebral cortex, facilitating the processing of inputs from the thalamus (Saper, Fuller, Pedersen, Lu, & Scammell, 2010). During waking, the monoaminergic neurons fire most rapidly, slow down during NREM sleep, and cease during REM (Aston-Jones & Bloom, 1981; Fornal, Auerbach, & Jacobs, 1985; Lu, Jhou, & Saper, 2006; Steininger, Alam, Gong, Szymusiak, & McGinty, 1999). The cholinergic neurons of the basal forebrain, among others, remain active during both waking and REM sleep (Jones, 2003); therefore, REM sleep is characterised by activity in the cholinergic ascending arousal system and by relative silence in the monoaminergic ascending arousal system (Saper et al.,

2001).⁹ Damage to either of these ascending pathways results in a long-lasting impairment of arousal, inducing a state of chronic sleepiness or even coma (Ranson, 1939; Saper et al., 2010; Von Economo, 1930).

On the other hand, the ventrolateral preoptic (VLPO) nucleus, which is very active during sleep, sends outputs to all of the major cell groups in the hypothalamic and brainstem nuclei that are involved in promoting arousal (Sherin, Shiromani, McCarley, & Saper, 1996), and thereby prevents the firing of these arousal-promoting neurons via inhibitory gamma-aminobutyric acid (GABAergic) and galanergic outputs during sleep (Saper et al., 2001). Likewise, the VLPO is innervated by the major monoaminergic systems as well, allowing for its inhibition during waking (Chou et al., 2002). Consequently, the VLPO neurons and the monoaminergic ascending arousal system are *mutually inhibitory*: activity in the one system prevents the inhibitory inputs from the other system, while simultaneously promoting the disinhibition of its own firing (Saper et al., 2001). This self-reinforcing loop has earned these mutually inhibitory systems the descriptive label, the *flip-flop sleep switch*. According to Saper et al. (2001), a flip-flop circuit is an engineering term for a switch used to produce discrete states with sharp transitions; they argue that this is a particularly apt term for these mutually inhibitory sleep- and arousal-promoting systems, as sleep-wake transitions are relatively abrupt, with only 1 - 2% of each day spent in transitional states. Consequently, these two systems are considered to be bistable—an organism is either in one state or another (either awake or asleep)—as the self-reinforcing firing patterns produce a degree of resistance to switching when one side is firing briskly. This bistable property of the flip-flop model is posited to ensure the after switch stability of waking or sleep (or REM and NREM; Saper et al., 2001, 2010). Furthermore, large scale influences, such as the circadian and homeostatic drives, are thought to gradually shift the inhibition of one system to another (e.g., the

⁹ More recently it has been suggested that REM sleep is primarily controlled by two mutually inhibitory GABAergic systems in the mesopontine tegmentum (Lu, Sherman, Devor, & Saper, 2006). The previously proposed, and well-known, monoaminergic-cholinergic mechanism (Hobson & McCarley, 1975) would therefore only act to *modulate* these mutually inhibitory systems. Further evidence in support of this hypothesis comes from studies showing that lesions to the cholinergic or monoaminergic nuclei of the brainstem have only a limited effect on REM sleep (Jones, Harper, & Halaris, 1977; Mouret & Coindet, 1980; Shouse & Siegal, 1992), whereas lesions to either of the newly proposed REM-on (sublaterodorsal nucleus and periventricular white matter) or REM-off (ventrolateral periaqueductal grey matter and lateral pontine tegmentum) neurons cause significant changes to the amount of REM sleep (Sakai, Crochet, & Onoe, 2001; Sastre, Buda, Kitahama, & Jouvet, 1996).

homeostatic sleep drive allows for the inhibitory and self-promoting firing of the VLPO to begin, resulting in the transition from waking to sleep; Saper et al., 2005, 2010).

The Dynamic Organisation of NREM Sleep

Halász and Bódizs (2013) have recently developed the models of Saper et al. (2001, 2005) by acknowledging the asymmetrical features of the descending and ascending slopes of the sleep cycles, as well as emphasizing the critical role of sensory stimulation in relation to this asymmetry in the facilitation of sleep.

Arousal and Anti-Arousal: Natural Elements of Sleep

Halász et al. (2004) previously underscored the differences in the descending slope (DS) and ascending slope (AS) of the sleep cycles in relation to the development of sleep. Whereas the DS involves the gradual deepening of sleep and an orderly progression through the sleep stages, the AS is marked by rapid changes in the lightening of sleep depth that often results in stages being skipped (Halász, 1982). Due to these rapid transitions the AS slope is also 30-50% shorter than the DS, as well as steeper. Based on these asymmetries Halász and Bódizs (2013) propose that the working mode of the DS is “sleep promotion”, while that of the AS is “arousal/REM promotion”. In particular, during the DS the VLPO driven sleep system exerts inhibition over the arousal system, resulting in diminished excitatory input to the thalamus and the subsequent gradual hyperpolarization of the thalamic relay cells. This causes a burst firing pattern that leads at first to spindling and later to the development of delta activity (Steriade, 2006). Conversely, the opposite trend occurs during the AS, as the VLPO system comes to be inhibited by the arousal system (Halász & Bódizs, 2013; Halász et al., 2004). Therefore the DS and AS involve two different sleep processes, and consequently, stages of sleep traditionally considered to be homogenous (e.g., descending S2 and ascending S2), may differ in several important respects depending on the balance of underlying sleep-promoting to arousal-promoting influences.¹⁰

Importantly, the asymmetry of the DS and AS slopes is also reflected in the sleep microstructure. During the DS arousals are less frequent, especially in the first few cycles, and they are comprised of slower EEG activities associated with mild autonomic perturbations, i.e., the A₁ subtype (Halász et al., 2004). Alternatively, during the AS arousals are more frequent and their EEG morphology and concomitant autonomic perturbations are

¹⁰ This point will be revisited shortly. For now, it is important to keep in mind that essentially no dream studies to date have made this differentiation. The sometimes striking discrepancies in recall from the same NREM sleep stage across various studies may therefore be related to inconsistent sampling from the descending and ascending slopes of the sleep cycles.

stronger, fulfilling better conventional arousal expectations, i.e., A₂ and A₃ subtypes (Terzano, Parrino, Boselli, Smerieri, & Spaggiari, 2000; Terzano et al., 2005).¹¹ The differences in the morphology and frequency of arousal activity along the slopes is attributed to *state-specific responsivity*, in line with the view that “the reactivity of an organism to stimulation is determined by the given state in which the input arrives” (Halász et al., 2004, p. 9); as such, the descending and ascending portions of the sleep cycle represent different states of reactivity within NREM sleep, especially during the first portion of the night when the homeostatic sleep pressure allows for the disinhibition of the sleep-promoting (VLPO) system during the DS (Saper et al., 2001, 2010).¹²

However, phasic arousal activity may not only be influenced by these tonic sleep- and arousal-promoting processes, *but may simultaneously influence this asymmetry as well*. According to Halász and Bódizs (2013), the asymmetrical slopes of the sleep cycles are regulated in parallel by both tonic and phasic processes

...tonic regulation has an endogenous neuromodulatory, chemical origin, serving the biological clock and showing little flexibility to external influences. It may have a NREM-promoting (during the D slope) or REM-promoting (during the A slope) nature. On the contrary—according to our concept—phasic regulation is more related to those neural mechanisms *that serve the sensory connection with the environment*, characterised by faster synaptic procedures. It is more variable and more dependent on external factors.

(p. 37, emphasis added).

Therefore, phasic activity is thought to tailor the interactions between the sleep and arousal systems of the sleep switch model more immediately.

One particular way in which this phasic regulation is proposed to be achieved is through the effect of sensory stimulation (exogenous or endogenous) on arousal formation. For instance, an input received during the DS that results in a slow synchronisation type of

¹¹ These polysomnographic findings were further confirmed by computerised analysis that showed an increase in fast rhythms in NREM sleep preceding REM, and a rapid reduction post-REM at the beginning of the following cycle (Ferri et al., 2001).

¹² Importantly, the balance of the sleep- and arousal-promoting influences that underlie the DS and AS changes over the course of the night as well—with the synchronisation and deepness of the cycles decreasing, and the density and length of the REM stage increasing, from evening to morning. These across the night changes are due to long lasting homeostatic and circadian influences on the sleep switch (Saper et al., 2005).

arousal (A_1) is able to incur an *antiarousal* response (Hirshkowitz, 2002). In other words, when the sleep disturbing input is received during the burst-firing mode of the thalamocortical network (i.e., during the prevailing influence of the VLPO sleep-promoting system)—and it is not robust enough to disinhibit the arousal/wake-promoting system—it results instead in a consequently strengthened rebound response by the VLPO system (Steriade & Llinás, 1988). It is hypothesised that this antiarousal response not only prevents awakening, but that it *enhances* delta activity as well, by entraining sensory stimuli as “fuel” for the burst-firing thalamic machine of slow wave sleep (Halász & Bódizs, 2013).¹³

Alternatively, if the arousing stimulus is received during the AS or when the VLPO system is less active (when homeostatic pressure is lower), the arousal input is able to activate the cortex—resulting in more prominent A_2 or A_3 EEG arousal response—thereby driving sleep to become more superficial. The enhancement of the arousal-related networks during the AS is considered necessary for the successful transition into the REM state, and consequently, the phasic events along this slope facilitate the progression of sleep by stimulating the arousal/wake-promoting system (Halász et al., 2004; Terzano, Parrino, Rosa, Palomba, & Smerieri, 2002). These dynamics of the microstructural oscillations have led Halász et al. (2004) to conclude that “sensory stimuli may participate in the determination of the sleep profile and co-operate in shaping the course of the sleep cycles” (p. 10). As such, the same sensory stimulus may have a sleep-promoting effect if received on the DS, while it may induce an arousing effect if received during the AS.

Additionally, there is ample evidence to show that information processing, to a certain extent, continues during sleep (Atienza & Cantero, 2001; Atienza, Cantero, & Escera, 2001; Bastuji, García-Larrea, Franc, & Mauguière, 1995; Heiser et al., 2012). Both functional neuroimaging and evoked response potential (ERP) studies consistently show that participants are able to distinguish their own names from other names during sleep, as well as distinguish between meaningful and arbitrary stimuli (Perrin, García-Larrea, Mauguière, & Bastuji, 1999; Portas et al., 2000). As mentioned previously, the characteristic reactivity of unstable sleep¹⁴ (i.e., a lack of habituation to external stimuli, as well as increased vigilance), is an indication that phasic arousal activity also functions to facilitate the connection between

¹³ While the sleep-promoting effects of sensory stimulation (Bohlin, 1971; Oswald, 1960; Webb & Agnew, 1978), and the deepening of sleep on the descending slopes in relation to sensory stimulation (Hirshkowitz, 2002), have previously been reported in the literature, these processes have remained largely unexplained until now.

¹⁴ Unstable sleep refers to NREM sleep characterised by CAP sequences.

the sleeper and their external environment during sleep; this feature of sleep essentially allows for its reversibility and distinguishes it from coma (Parrino et al., 2012). In this way, NREM sleep is characterised by continuous oscillatory fluctuations in sleep depth that on the one hand allow adaptation in sleep due to external demands, while on the other hand permit phasic events to facilitate sleep state transitions by unbalancing the mutually antagonistic sleep- and arousal-promoting influences as needed (Halász & Bódizs, 2013).

This dynamic nature of the sleep microstructure has led Halász and Bódizs (2013) to further argue that phasic regulation provides *stability by allowing lability*; in other words, by allowing NREM sleep to be flexible, the range of phasic microstructural phenomena (as systematised within the CAP framework) accomplish two seemingly contradictory states—the maintenance of sleep separate from the environment and the simultaneous connection with the environment. As a result, the tonic, central regulation of sleep is posited to have a level-setting effect determining the overarching state, while phasic influences ensure fast “answers” to the demands of the external world, as well as simultaneously use this input to attempt to strengthen the state within which the external demand is made.¹⁵ Based on this principle of flexibility, Halász and Bódizs (2013) propose that between the deterministic macrostructural switches (sleep to wake, and NREM to REM), the NREM state does *not* appear to behave in the bistable manner proposed by Saper et al. (2001); instead, it is the ability of NREM sleep to maintain two contradictory states that ensures its durability and allows for the stereotypical sleep structure to unfold. Nonpathological phasic arousal activity during sleep should therefore be considered a natural and essential element of sleep.

Preliminary Evidence for an Association between Dreaming and CAP

While enhanced dream recall has been reported in relation to periods of intrasleep wakefulness in both questionnaire data (Cory, Ormiston, Simmel, & Dainoff, 1975; Schredl, Wittman, Ciric, & Götz, 2003) and through polysomnography (Takeuchi, Miyasita, Inugami, & Yamamoto, 2001), dreaming is yet to be studied in relation to sleep microstructure as systematised within the CAP framework, or in relation to any of the arousal and antiarousal

¹⁵ Additionally, the ability of the sleep microstructure to use sensory stimuli (generated either externally or internally via bodily sensations) to aid in the development of sleep is not merely opportunistic. Auditory stimuli presented during sleep will result in an increase in evoked arousals, and a consequent reduction in spontaneous arousals; however, the total number of arousals (evoked and spontaneous) will still sum to the baseline number of spontaneous arousals. In other words, it appears as though sleep contains a minimum number of phasic arousal phenomena each night, regardless of amount of external disruption, indicating once again that these events are an essential part of natural sleep (Terzano, Parrino, Fioriti, Orofiamma, & Depoortere, 1990).

features of sleep just discussed. Furthermore, based on the principle that phasic arousals during sleep are not awakenings—albeit they may be related to intrasleep wakefulness—there is also only so much that can be inferred from these studies in relation to the sleep microstructure. Nevertheless, these studies offer, albeit indirectly, valuable evidence of a potential relationship between dreaming and the sleep microstructure.

Enhanced Brain Reactivity and Dream Recall

An important link between dreaming and sleep related arousal processes has recently been established in a study comparing the sleep characteristics and brain activity of healthy subjects with habitually high and low dream recall frequencies (i.e., higher recallers and low recallers), using polysomnographic recordings and auditory event-related potentials (ERPs; Eichenlaub, Bertrand, Morlet, & Ruby, 2013). In particular, complex sounds (the participant's first name and an unfamiliar first name) were presented randomly and rarely amid repeated pure tones. Using this novelty odd-ball paradigm, during both sleep and waking, the authors were able to investigate the various steps in information processing: i) early auditory perception (the early N1 component); ii) attention orienting (the frontocentral novelty related P3 or P3a component related to unexpected sounds); and iii) late ERP components involved in higher cognitive processing (parietal components evoked by complex sounds). In line with the *arousal-retrieval* model¹⁶ of dreaming it was predicted that dream encoding and recall would be related to intrasleep awakenings, and furthermore, that intrasleep wakefulness might be a function of neurophysiological trait-related differences in high and low recallers.

It was found that high dream recallers had more intrasleep awakening time, as well as significantly longer awakenings, than low recallers; no other macrostructural differences were noted. Furthermore, high recallers were shown to have higher amplitude P3a¹⁷ and late latency responses¹⁸ to novel and unexpected stimuli, during both waking and sleep;¹⁹ a

¹⁶ According to the arousal-retrieval model of dreaming the brain is unable to code information into long term memory during sleep, and consequently, an effective transfer of a dream to long term memory is only possible if an awakening (or state of sleep proximal to waking) occurs during the short term memory trace of the dream (that would allow the dream to be encoded and subsequently recalled; Koulack & Goodenough, 1976).

¹⁷ Attention directed to sounds is a factor known to enhance the P3a component (Polich, 2007), and the larger the P3a the stronger the orientation of attention (Dominguez-Borras, Garcia-Garcia, & Escera, 2008; Escera, Yago, Corral, Cobera, & Nunez, 2003).

¹⁸ The enhanced late parietal component in high recallers—associated with complex cognitive processes, such as familiarity, episodic memory, and emotional processing (Eichenlaub, Ruby, & Morlet, 2012; Holeckova, Fischer, Giard, Delpuech, & Morlet, 2006; Kissler, Herbert, Winkler, & Junghofer, 2009)—is an indication that

significant correlation was also found for intrasleep wakefulness and the amplitude of the P3a during waking. These results suggest that attentional orientation to unexpected and novel stimuli is more pronounced in high recallers than low recallers. It was therefore concluded that “[high recallers] were more reactive to the external world than [low recallers]...during sleep...as during wakefulness” (Eichenlaub et al., 2013, p. 8). Based on these results, Eichenlaub et al. (2013) argued that the robust differences in brain reactivity between the two groups show that the cerebral organisation of high recallers is intrinsically different to low recallers, and that this difference may potentially facilitate either the production or encoding of the dream; in particular, the authors propose that high brain reactivity facilitates intrasleep wakefulness during sleep, which in turn facilitates the encoding of dreams into memory. As a result, they conclude that their findings “extend the arousal-retrieval model [by] proposing an explanation for the difference in intrasleep wakefulness between High- and Low-recallers through differences in brain reactivity” (p. 9).

The aforementioned results are important for the purposes of this study because they link arousal during sleep to dream recall. Although the study does not explore phasic arousal activity in particular, it could reasonably be presumed that the differences in reactivity found between high recallers and low recallers may be related to differences in sleep microstructure. Indeed, phasic arousal activity is involved in regulating the connection between the sleeper and external environment, and similarly, Eichenlaub et al. (2013) found that high recallers were more reactive to the external world during both waking and sleep. Furthermore, the induction of unstable sleep (phasic arousal activity) using auditory stimuli has previously been shown to result in more intrasleep wakefulness as well (Terzano et al., 1990). As such, an increase in intrasleep wakefulness in high recallers may have been accompanied by a concomitant increase in sleep instability. Assuming that there are microstructural differences between these two groups, studying sleep microstructure in relation to dreaming may extend these important findings by further explaining how enhanced intrasleep wakefulness and brain reactivity in high recallers is related to dream frequency; thereby also further characterising the proposed neurophysiological trait-related differences between these two groups.

cognitive processing during inattentive waking, as well as during REM sleep, may be more complex in high recallers than low recallers. Also, in the latest latencies (~1000 ms) novel sounds elicited a more positive response in high recallers than low recallers in *all* vigilance states.

¹⁹ The absence of between-group differences in the early N1 component either to standard tones or to first names suggests that the two groups did not differ in their primary auditory processing (Eichenlaub et al., 2013).

Arousal and Antiarousal Processes and Dreaming

In relation to more traditionally defined phasic arousal activity, a study by Takeuchi et al. (2001) found that dream recall in experimental awakenings from NREM sleep—that were made five minutes after the first appearance of sleep spindles or K-complexes (i.e., after the appearance of S2 sleep) in the late part of the night—was significantly ($p < .001$) positively correlated with the number of awakenings and microarousals in the portion of sleep prior to waking. However, the phasic arousal phenomena in this study were scored according to the more conventional ASDA guidelines that consider microarousals during sleep to be independent events—alternatively, CAP informs us that this is hardly ever the case. In most instances, arousals tend to occur within a stereotyped proximity of one another, essentially demarcating unstable NREM sleep (Terzano & Parrino, 2000). Therefore, while this study confirms a relationship between dreaming and microarousals during light NREM sleep, because the phasic arousal phenomena were not scored fully according to the CAP criteria, very little can be said regarding dreaming in relation to the sleep microstructure. For instance, was dreaming related to CAP sequences (i.e., unstable sleep) rather than individual microarousals not entrained within a sequence (i.e., stable sleep)? Furthermore, did the presence of the slower A₁ type of arousal influence recall in a similar way to the faster A₂/A₃ subtypes? The ways in which dreaming relates to the various arousal subtypes, as well as stable and unstable NREM sleep in general, requires further study before any definitive conclusions are reached.

Similarly, certain types of insomnia have been found to be related to changes in sleep microstructure as well as dream recall. Specifically, there is an increase in A₂ and A₃ arousals in both the AS and DS of the sleep cycle reported in insomnia (Parrino et al., 2004; Terzano & Parrino, 1992), as well as enhanced dream recall (Schredl, Schäfer, Weber, & Heuser, 1998). Schredl et al. (1998), in accordance with the arousal-retrieval model, explain the increase in dream recall as a function of increased nocturnal awakenings during sleep in these patients. However, no mention was made of the known changes in the arousal (A₂/A₃) and antiarousal (A₁) subtypes in insomnia in relation to dream recall. Additionally, other studies have found *less* dream recall in patients with insomnia (Pagel & Shocknesse, 2007), as well as less dream recall in other sleep disorders marked by excessive awakenings (i.e., sleep apnea; Schredl et al., 2006), and therefore, the mechanism for increased recall may be more complex than that indicated by the arousal-retrieval model.

Alternatively, the arousal-retrieval model suggests that factors such as *interference* may prevent dream recall; in particular, Koulack and Goodenough (1976) propose that

“experiences occurring during or shortly after awakening compete with the target material for space in the limited-capacity processing system, with the most salient of the set favoured in the competition” (p. 975). In relation to this, it may be valuable to investigate the extent to which antiarousal processes are related to negative dream recall. Delta activity has generally been shown to be inversely related to dream recall (Chellappa, Frey, Knoblauch, & Cajochen, 2011; Esposito, Nielsen, & Paquette, 2004; Wollman & Antrobus, 1987), and considering that roughly 60% of all CAP events are comprised of the A₁ (antiarousal) subtype (Terzano et al., 2002; Terzano & Parrino, 2000), it is reasonable to predict that these bursts in synchronised delta activity might contribute to a *lack of dream recall* through interference or some other, as yet unspecified, mechanism. This predicted inverse relationship between dream recall and the A₁ subtype is further supported by studies showing that more thoughtlike, and less hallucinatory, dreaming is experienced in the first half of the night from NREM awakenings, and more hallucinatory dreaming occurs in the second half of the night²⁰ (Antrobus, Kondo, & Reinsel, 1995; Cavallero, Cicogna, Natale, Occhionero, & Zito, 1992; Fosse, Stickgold, & Hobson, 2004; Rosenlicht, Maloney, & Feinberg, 1994; Takeuchi, Ogilvie, Murphy, & Ferrelli, 2003; Wollman & Antrobus, 1987). It remains uncertain, though, whether synchronisation type arousals (or antiarousals) could lead to thoughtlike activity due to the depredated recall of *ongoing* dream activity, or rather, if thoughtlike activity is characteristic of sleep containing these features. The arousal-retrieval model supports the former explanation, while the *general cortical activation* theory²¹ supports the latter; as there is no empirical work to date regarding dreaming and input-dependent antiarousal processes, it remains to be seen whether A₁ arousals share *any* particular relationship with dream recall.

Conclusion

Once thought to be an accessory to sleep, phasic arousal phenomena are now understood as essential to the dynamic construction of the sleep cycle. These advances in sleep science not only herald a new understanding of sleep processes, but potentially offer a

²⁰ To the extent that REM and NREM dreams could not be differentiated in terms of their hallucinatory or thoughtlike quality (Fosse et al., 2004).

²¹ Antrobus, Kondo, Reinsel, & Fein (1986) proposed that dreaming is a function of general cortical activation in conjunction with sensory gating thresholds. For example, during REM sleep the cortex is highly activated and the sensory thresholds for environmental stimuli are high, resulting in intense dream experiences. However, this perspective has been supported by a number of other researchers in one way or another over the last few decades (Antrobus, 1986; Dement, 1965; Hernandez-Peon, 1967; Zimmerman, 1970).

deeper understanding of dream processes as well. The asymmetry of the sleep cycles and the mutually antagonistic sleep- and arousal-promoting mechanisms that drive both the sleep macro- and microstructure can now be applied to the dream process. There is already evidence in the literature to suggest that dreaming may be related to these processes, and that the variable connection between the sleeper and the external world may be crucial to understanding dreaming during NREM sleep.

University of Cape Town

SPECIFIC AIMS AND HYPOTHESES

Non-REM sleep is far more nuanced and complex than previously acknowledged in the dream literature. Dream studies have yet to consider the potentially conflicting influences on dream recall of arousal and antiarousal processes, as well as the possible significance of unstable sleep on the dream process. The primary aim of this study, therefore, is to explore NREM sleep in new depths in relation to dreaming by drawing on the cyclic alternating pattern (CAP). While CAP does not ensure the complete predictability of the phasic arousal processes, it is a general marker of sleep instability that can be used as a tool for studying phasic activity in relation to dreaming.

With this in mind, this study aims *to examine sleep microstructure in relation to dreaming in a healthy population of young adults, and determine whether there is any relationship between dream recall and phasic arousal phenomena during NREM sleep*. Based on the findings in the literature thus far, it is predicted that dream recall will be: i) enhanced during unstable NREM sleep compared with stable NREM sleep; ii) enhanced by the presence of A₂ and A₃ arousal subtypes prior to waking; iii) reduced by the presence of the A₁ arousal (antiarousal) subtype prior to waking. The following hypotheses summarize these aims and predictions:

- H₁: Awakenings made within unstable NREM sleep will yield enhanced dream recall compared with awakenings from stable NREM sleep.
- H₂: Dream reports elicited after the CAP A₂ and A₃ subtypes during sleep will significantly approach REM dream reports both quantitatively and qualitatively.
- H₃: Dream reports elicited after the CAP A₁ subtype during sleep will result in significantly less dream recall than general NREM sleep (both stable and unstable).

METHODS

Design

A repeated measures quasi-experimental design was used to investigate the relationship between sleep mentation and sleep microstructure. Electroencephalography (EEG) was used to record sleep, and to allow for participants to be awoken in close proximity to EEG-defined phenomena of interest, so that mentation could be studied in relation to these phenomena. Each participant was required to spend three to four nights in the sleep laboratory. The first night functioned as an adaptation night, while the other nights were experimental.

Sleep stability was defined according to the presence or absence of oscillating phasic arousal phenomena that fulfilled the criteria for a CAP sequence (see Appendix A for scoring criteria). Specifically, NREM sleep periods characterised by CAP sequences were classified as *unstable*, while sleep periods characterised by an absence of CAP sequences were classified as *stable*. These two conditions were referred to as CAP and NCAP sleep, respectively. Additionally, NREM sleep was further categorised according to the classic sleep staging criteria (Rechtschaffen & Kales, 1968); light NREM sleep comprised Stage 2 sleep, while deep NREM sleep comprised Stage 3 and 4 sleep. Based on these aforementioned criteria, there were four preawakening conditions: i) unstable light NREM sleep; ii) unstable deep NREM sleep; iii) stable light NREM sleep; iv) stable deep NREM sleep. In accordance with the specific aims of this study, the main effects of sleep stability (CAP vs. NCAP) and sleep stage (light vs. deep NREM), as well as the CAP arousal subtypes (A_1 , A_2 , A_3), were investigated in relation dreaming.

The dependent variables in this study were all related to dream recall. Dreaming was operationally defined as any mentation during sleep that could be remembered and verbally described. In addition to more conventional dream experiences (i.e., sensory-perceptual imagery together with some dramatic progression), participants were also encouraged to describe any thoughts and sensory imagery, no matter how brief or fragmented (this brevity was taken into consideration in analysis). A complete lack of recall or mentation that could not be remembered was classified as *negative dream recall*.

The study adhered to the ethical guidelines for research with human subjects as specified by the Health Profession Council of South Africa (HPCSA), as well as the University of Cape Town (UCT) Codes for Research. Application for ethical approval was granted by the Psychology Department's Research Ethics Committee at UCT.

Participants

Twenty two²² healthy participants (14 female and 8 male) between the ages of 18 and 25 were recruited from the University of Cape Town's undergraduate student population; all participants were paid volunteers. In order to control for any confounding inter-individual differences in ability to verbally report the dream experience, all participants were required to be fluent English language speakers. To avoid any bias due to differences in habitual dream recall frequency, all participants were required to be moderate to frequent dream recallers (i.e., able to recall dreams on a biweekly basis).

Exclusion Criteria

- 1) *Psychiatric and sleep disorders.* Participants presenting with any chronic psychiatric disorders as assessed by the *Mini International Neuropsychiatric Interview (M.I.N.I.*; English version 5.0.0; Sheehan et al., 1998) or any sleep disorders as assessed by the *Pittsburgh Sleep Quality Index (PSQI*; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) were excluded from the study on the basis that these disorders may affect sleep architecture and dream recall (Benca, Obermeyer, Thisted, & Gillin, 1992; Nofzinger, 2005).
- 2) *Chronic medical conditions and medications.* Each participant was required to give a general medical history and was excluded if they had any chronic medical condition that could affect sleep (e.g. asthma or epilepsy; Bazil & Malow, 2005). The use of any chronic medications (excluding oral contraceptives), also warranted exclusion from the study.
- 3) *Substance use.* Any use of previous or current psychoactive drugs, or smoking (either socially or chronically), was considered a factor for exclusion from the study, as both nicotine and narcotics have been found to alter natural sleeping patterns (Domino & Yamamoto, 1965; Pagel, 2005).

Measurement and Materials

Dream Measures

The approach that this study took to quantifying and qualifying sleep mentation was multimodal: both subjective and objective ratings were used in combination so that the advantages of each method were benefitted from, while the disadvantages were compensated for. In particular, subjective ratings allowed for a more accurate representation of emotion,

²² A sample size of 20 is suggested as a minimum for dream studies as it ensures that main effects can be detected over individual differences (Antrobus, Fein, Jordan, Ellman, & Arkin, 1991).

perception, or depth of sleep (Schredl & Doll, 1998), whereas objective measures were useful for gauging the presence of certain elements within the dream experience (e.g., bizarreness).

The Multidimensional Dream Questionnaire (MDQ)

The MDQ is a self-report questionnaire that was constructed for this study. It consists in 28 questions, most of which were derived from the eight dream dimensions previously established by Hauri, Sawyer and Rechtschaffen (1967; see Appendix B). The goal of Hauri et al.'s (1967) original factor analysis was to produce a scale of a few dimensions able to cover the reported content of dreams. In total, the eight dimensions that emerged from the factor analysis accounted for 63% of the total variance and were found to be roughly orthogonal to each other. Good interrater reliabilities were achieved for the scales, with Pearson correlation coefficients mostly $> .8$ for trained raters (Foulkes, Larson, Swanson, & Rardin, 1969; Weisz & Foulkes, 1970). The eight dimensions are:²³

1. Vivid Fantasy: feelings of unreality (imagination, distortion) coupled with intensity of experience (dramatization, clarity, emotion); productivity (length of dream report) was also highly loaded on this dimension. (Q3, Q4, Q16, Q21, Q22)
2. Active Participation/ Active Control: the extent to which the dreamer is active and exerting control over the dream events. (Q14, Q15, Q19, Q20)
3. Pleasantness-Unpleasantness of the dream. (Q6)
4. Verbal Aggression in the dream, from the dreamer or other characters. (Q7)
5. Physical Aggression in the dream, from dreamer or other characters. (Q8.1, Q8.2)
6. Sexuality. (Q18)
7. Perceptuality/ Conceptuality: Is the dream more conceptual or more perceptual? (Q9, Q10, Q11, Q12, Q13)
8. Time: With what time in the dreamer's life is the manifest content mostly associated? (Q17)

In addition to the above questions based on the eight dimensions of dreaming, questions relating to the presence and intensity of each of the seven basic emotions, as defined by Panksepp (1998), were included (Q5.1 – Q5.8, see Appendix C for definitions). The basic emotions were selected due the established neurophysiological overlap between certain of these emotional systems and the neural correlates of dreaming (Solms, 1997, 2000).

²³ The questions in the MDQ based on each factor are included in parentheses (see Appendix B).

Furthermore, a self-rated global measure of dreaming was included (Q2, see Appendix B). Participants were required to rate *how much* they had been dreaming before waking, from 0 (*not at all*) to 4 (*greatly*); they were encouraged to rate the question based on their own subjective criteria.

Total Recall Count

Total Recall Count is one of the most established broad-based measures of dreaming and information processing during sleep, and includes a count of all of the words used by the participant to describe their mentation, excluding repetitions, redundancy, commentary on the dream material, or “ahs” and “ums” (Antrobus, 1983; Antrobus, Schnee, Lynn, Silverman, & Offer, 1976; Rosenblatt, Antrobus, & Zimler, 1992). Furthermore, total recall count is one of the most sensitive measures of the difference in REM and NREM mentation, with REM sleep reliably producing a substantially higher word count (Antrobus et al., 1995; Wamsley & Antrobus, 2008). Total recall count is also considered a reliable measure of tonic cortical activation, as consistent increases have been found in relation to the rising diurnal rhythm (Antrobus et al., 1995). In terms of interrater reliability, this measure has been found to have very good reliability, with Spearman Brown correlations between raters $> .9$ (Antrobus et al., 1991).

Relation to Everyday Experience

This scale, developed by Foulkes et al. (1969), requires raters is to determine how bizarre the dream is by indicating the extent to which the events that transpired are possible in everyday life. A rating of 5 indicated *no apparent relation to the subject's everyday life*, while a rating of 1 was given to a dream report that referred (hypothetically) *without distortion to some specific event in the subject's life* (Winglet & Cramer, 1979, p. 127, see Appendix D).²⁴ The scale has been found to have good interrater reliability ($r = .75$; Foulkes et al., 1969). In terms of past research, the scale has been used to study bizarreness and distortion in the dreams of institutionalised boys, who were found to have dreams that contained significantly *less* everyday content than controls (Foulkes, et al., 1969).

²⁴ In the original scale, 1 represented *no relation to everyday experience*, while 5 represented a dream *identical to everyday experience*; the directionality of the scoring was reversed in this study as this scale was used to rate bizarreness, and interpretation and explanation were more intuitive when higher ratings were representative of more bizarreness, and lower rating representative of less. For this reason as well, the scale is interchangeably referred to as the *bizarreness* scale.

Perceptual-Interaction Rating Scale

The Perceptual-Interaction Rating Scale was constructed for use in this study. It was intended as a broad-based measure of *overall* dream quantity and quality that could be applied to the verbal dream reports by external raters. The aim of this scale was to rate the amount of *interaction* between the dream characters and the dream environment—be the characters people, animals or even inanimate objects (see Appendix E). The more interaction described, the more perceptuality was inferred, as an assumption implicit to the scale was that the great majority of interactions that take place within a dream can only transpire within a suitable dream (pseudo-perceptual) environment. The scale ranged from 0 (*no recall*) to 9 (*extensive interaction within a visual scene*).

Awakenings

Verbal dream reports were elicited upon each awakening. The awakenings were made following a selected combination of physiological sleep events, as determined visually by the online EEG recording. Five to six awakenings were made on each experimental night—when sleep was particularly disrupted fewer awakenings were made, and in certain cases participants were asked to return for a third night to collect more dream reports. The aim was to collect four reports from light NREM sleep; four reports from deep NREM sleep; and three reports from REM sleep, for each participant. An effort was made to balance the number of awakenings for each participant between the early (± 10 pm to ± 2 am) and late (± 2 am to ± 6 am) portions of the night to control for the time of night effect on dream recall (Antrobus et al., 1995; Nielsen, 2000; Rosenlicht et al., 1994).

In REM, awakenings were made after 5 to 10 minutes of the simultaneous occurrence of rapid eye movements, reduced muscle tonus (EMG), and a desynchronised EEG. Awakenings followed one of four preawakening conditions in NREM sleep: i) unstable light NREM sleep; ii) unstable deep NREM sleep; iii) stable light NREM sleep; iv) stable deep NREM sleep. Stable sleep was defined as any NREM period characterised by at least 60 seconds of sleep free of any CAP sequences (individual arousal events that were not part of a CAP sequence were included; see Appendix A). Unstable sleep, on the other hand, was required to contain at least three CAP arousals within 60 seconds of one another, and the awakening had to occur less than 60 seconds from the last arousal.

At least 40 minutes of consolidated sleep was allowed to pass before an awakening was made. Additionally, awakenings were made at least 15 minutes after a REM episode to avoid any continued effects of REM sleep (Nielsen, 2000). Originally, the study aimed to collect awakenings from reports that contained either desynchronised arousals (A_2 and A_3) or

synchronised arousals (A_1); however, the sequences were largely mixed and the individual contributions of each type of microarousal could only be calculated with statistical manipulation (see Data Analysis). Furthermore, having to make the awakenings in relation to live CAP activity proved challenging since the unexpected termination of a sequence is always possible; therefore, no predetermined CAP sequence length was allocated. This was similarly the case with stable NREM sleep—it was uncertain when a CAP sequence would start, and as such, variable lengths of stable sleep were allowed before awakenings were made.

Likewise, due to this limited predictability of the occurrence and duration of CAP sequences it was not possible to fully randomise the experimental awakenings. Consequently, awakenings were not made after a predetermined duration of time, as is often done in dream studies as a control measure (Antrobus et al., 1995; Nielsen, 2000). Instead, all NREM awakenings were made according to the criteria mentioned previously for sleep stability and sleep stage, resulting in each dream report being attributed to one of the four preawakening conditions. Statistical methods were then used to control for the confounding effects of awakenings made at different points in the sleep cycle (i.e., the descending and ascending slope, and trough); different times of night; and after variable durations of stable and unstable sleep. It was not considered a disadvantage, however, that the duration of stable or unstable sleep prior to the experimental awakening was not controlled for, as this allowed for the additional investigation of both *CAP sequence length* and *duration of stable NREM sleep* to be studied in relation to dream recall. Awakenings following stable and unstable NREM sleep were counterbalanced, as far as possible, both within and between nights for each participant.

The preawakening criteria were reconfirmed post hoc. In certain cases, awakenings had to be moved from one group to another (e.g., from stable light NREM to unstable light NREM). The recategorization of awakenings led to an unequal number of reports for each of the four NREM conditions. Furthermore, due to very unstable sleep in specific cases (likely due to the experimental situation and the multiple sleep disruptions) certain participants had data missing for one of the four groups. The unequal samples sizes and missing data are further examined in the results section.

Assessing Homogeneity and the Inclusion/Exclusion Criteria

Wechsler Abbreviated Scale of Intelligence: Vocabulary subtest

The *Wechsler Abbreviated Scale of Intelligence* (WASI; The Psychological Corporation, 1999) Vocabulary and Similarities subtests were used to yield a *verbal IQ* (VIQ) score for each participant. These subtests take approximately 15 minutes to administer and

are widely used in research settings (Strauss, Sherman, & Spreen, 2006). Due to the participant age range (18-25 years) the raw scores for the VIQ were converted to age-corrected T scores, which represent the average performance for each age group on that particular subtest. Only those participants with an above average VIQ (> 100) were considered for participation in the study, so as to prevent any confounding of the verbal reporting of the dream experience due to difficulties with English language fluency.

Mini International Neuropsychiatric Interview

The *Mini International Neuropsychiatric Interview (M.I.N.I.)* (English version 5.0.0) is a structured interview intended to diagnose the major DSM-IV and ICD-10 psychiatric disorders. The interview can be administered in approximately 15 minutes by a clinician or a trained interviewer, and its brevity makes it particularly suitable as a screening tool for use in research. Furthermore, the M.I.N.I. has been found to have good psychometric properties and has shown good reliability and validity in eliciting the symptom criteria used in defining the DSM-IV and ICD-10 diagnoses (Sheehan et al., 1998). Each participant was administered the M.I.N.I. as a screen for any psychiatric conditions that could potentially affect sleep and dream recall.

The Pittsburgh Sleep Quality Index

The *Pittsburgh Sleep Quality Index (PSQI)* (Buysse et al., 1989) is a self-rated questionnaire designed to measure sleep quality in either clinical practice or research activities. The questionnaire assesses sleep quality and disturbances in the past 1-month time interval. The questionnaire is comprised of 19 separate items that are used to generate seven 'component' scores: the use of sleeping medication, sleep latency, sleep duration, subjective sleep quality, habitual sleep efficiency, sleep disturbances, and daytime dysfunction. The component scores add to produce one global score between 0-21 points. As an example of the validity of the measure, a global PSQI score > 5 was found to yield a diagnostic sensitivity of 89.6% in distinguishing good and poor sleepers (Buysse et al., 1989). As a global score of >5 on the PSQI indicates that the participant is a poor sleeper, only those participants scoring < 5 were included in this study.

Measuring physiological sleep variables

Recording techniques, equipment and scoring

A mixed bipolar longitudinal and referential montage was used, including the following bipolar derivations Fp1–F3, F3–C3, C3–P3, P3–O1 and Fp2–F4, F4–C4, C4–P4, P4–O2. Due to the fact that CAP is a global EEG phenomenon and is subsequently visible across all EEG leads in both hemispheres (with only minor differences in morphology and

amplitude across the various leads) a bipolar longitudinal montage has been found to guarantee a favourable detection of CAP during sleep (Terzano et al., 2001; Terzano & Parrino, 2000). Referential derivations were used (C3-A2, O1-A2, C4-A1 and O2-A1), in addition to longitudinal ones, as the increased distance between the electrodes enhances the amplitude of phasic arousal phenomena, making them more easily distinguishable where necessary (Terzano et al., 2001). Eye movements, muscle tonus and heart rate were recorded on two EOG (electrooculography), one EMG (electromyographic), and one ECG (electrocardiographic) channel, respectively; these channels are required to assist with the conventional sleep staging, as well as to score phasic arousal phenomena (Rechtschaffen & Kales, 1968; Terzano et al., 2001). Standardised filters for recording sleep were employed for the EEG and EOG (0.5 – 35Hz), EMG (10 – 70Hz) and ECG (1 – 70Hz) leads to ensure the integrity of the signal in each of the channels (Spriggs, 2009). The ground electrode was placed on middle of the forehead. Nihon Kohden pure silver disk electrodes (10mm) with a hole in the centre and a 150cm lead were used for the EEG channels, while disposable conductive adhesive electrodes were used for the EOG, EMG and ECG channels. The EEG leads were fixed in place with collodion adhesive and a conductive paste was put inside the electrodes to improve signal quality. A detachable head box allowed for mobility during the night.

Sleep was recorded using the NeuroFax EEG9000 32- channel polysomnograph (PSG) from Nihon Kohden, with Polysmith Online version 6 software. Thereafter, the sleep records were converted into EDF (European Data Format) files and Hypnolab v2.0 was used to score the sleep stages and CAP phenomena. Both sleep macrostructure and CAP were manually scored by a trained researcher according to the established scoring criteria for the classic sleep stages (using 30 second epochs) and CAP (Rechtschaffen & Kales, 1968; Terzano et al., 2001). However, only the CAP sequence immediately preceding each awakening was scored for further analysis; CAP parameters for the entire sleep record were not analysed. The scorer was blind to the identity of the participants, as well as the dream report related to each awakening.

Sleep Stage and CAP Parameters

Intrinsic to the aims of this study were multiple awakenings per participant, per night, and therefore disrupted sleep architecture was expected. As such, only some of the conventional sleep parameters were included for further analysis: *time in bed* (TIB): the total amount of time spent in bed; *sleep period time* (SPT): total sleep time including intrasleep waking; *total sleep time* (TST): total time spent in NREM and REM, excluding intrasleep

waking; *sleep onset latency* (SOL): the interval between lights-out and the first appearance of S1 sleep that subsequently progresses to S2; *sleep efficiency* (SE%): the percentage of time spent in bed asleep (TST/TIB); *waking after sleep onset* (WASO): the amount of time spent awake after sleep onset; the *total duration of all sleep stages* as percentage of the TST, including REM, S1, S2, S3, S4, and SWS (S3 + S4).

The CAP parameters analysed included: *sequence length* (SL): the total duration (in seconds) of the CAP sequence²⁵; *time since last A* (TSA): the duration of time (in seconds) from the last CAP arousal (A₁, A₂, or A₃) to the experimental awakening; *total A₁, A₂, and A₃*: The total number of A₁, A₂ or A₃ arousals prior to an awakening; *total A₁, A₂, and A₃ in the last three minutes*: The total number of A₁, A₂, and A₃ arousals in the three minutes immediately preceding an awakening; *total A₁, A₂, and A₃ in the last minute*: The total number of A₁, A₂, and A₃ arousals in the last minute immediately preceding an awakening.

Procedure

Screening

Two screening processes were used to ensure a sample was selected according to the inclusion and exclusion criteria. Potential participants were first required to fill in an online questionnaire; to progress to the second interview they were required to be free of any medical conditions or chronic illnesses that could affect sleep architecture (for instance, asthma). Smoking and previous or current drug use was also a factor for exclusion. The second screening session, which took place in a quiet room at the University of Cape Town psychology department, established whether potential participants had a VIQ > 100, a PSQI < 5, and whether they were free of any psychiatric conditions according to the M.I.N.I.; if all of these criteria were met the student was asked to participate in the sleep study. Consent was obtained at each stage of the screening process.

Sleep Study

The study took place at a hospital sleep laboratory that is in collaboration with the University of Cape Town. The sleep laboratory comprised two rooms. Sleep was monitored via the control room by a trained researcher, while the participant slept in a room adjacent to the control room that was partially soundproofed and had an en suite restroom. A one way mirror between the two rooms allowed the researcher to see into the participant's room.

All participants underwent an adaptation night to become familiar with the laboratory and the multidimensional dream questionnaire (see Measures). After the adaptation night, each participant's sleep was monitored using polysomnography for two inconsecutive nights

²⁵ Note that sequences longer than 15 minutes were scored as > 900 seconds.

in order for dream reports to be collected (the two nights were separated by 2 to 7 days). Each participant was thoroughly informed of the main purpose of the study and the procedures. In an attempt to curb any effort to please the experimenter, participants were encouraged to be completely honest in the reporting of their dreams; they were ensured that all variations of sleep mentation, and even no recall, were of interest. Consent was obtained before data collection began and participants were ensured that they were free to withdraw from the study at any time without consequence (see Appendix F).

Sleep night protocol

Prior to coming to the sleep laboratory all participants were instructed: i) to consume dinner not later than 19:00 that evening; ii) not to consume any alcohol on the day of the experiment or any caffeinated beverages after 3 p.m., to avoid changes in cortisol levels and subsequent effects on sleep (Landolt et al., 2004 ; Lovallo, Farag, Vincent, Thomas, & Wilson, 2006); iii) not to take any naps during the day of the experiment, as this may prolong their sleep onset in the laboratory (Landolt et al., 2004); iv) to go to bed at their usual time and get six to eight hours of sleep the night before.

Participants arrived at the sleep laboratory at 19:00, and between 19:00 and 21:45 each participant was prepared for sleep monitoring; electrodes were attached as per the selected recording montage. A total of ± 8 hours of sleep was recorded for each participant: lights off commenced at approximately 22:00 and participants were awakened at around 6:00 the following morning. Prior to lights out participants were reminded about the awakenings that would take place during the night; each participant was awakened five to six times throughout the night for a dream report (including the morning awakening). The electrodes were removed the following morning, and the participants were thanked and compensated in accordance with their participation agreement. This protocol was applied to every experimental sleep night.

Dream awakenings

When the required EEG awakening criteria were met, the researcher entered the room that the participant was in and called the participant by their name until they indicated with a verbal response that they were awake. Upon waking, participants were required to give a verbal description of the dream experience. In particular, once the participant was alert the following dream recall protocol, adapted from Foulkes, Spear, & Symonds (1966) and Antrobus et al. (1995), was carried out:

“Tell me everything that was going through your mind just before you were awoken.”

When the participant was finished the report, then he or she was asked:

“Is that everything that you can remember?”

If it was uncertain whether the dream was visual, the researcher followed up these questions with:

“Could you see what was going on? Was it visual?”

Immediately after the verbal report participants were required to rate their mentation according to a likert-type questionnaire (Appendix B). Because the researcher who elicited the dream reports was not blind to the preawakening conditions the above protocol was strictly adhered to, to prevent any potential experimenter bias. All procedures were carried out in English. After being awakened the participant was allowed to go back to sleep, and at least 40 minutes of consolidated sleep was allowed to pass before the next awakening. The entire report was audio-recorded and later transcribed by a trained researcher blind to the preawakening conditions and the aims of the study. The content of the verbal dream reports was then further rated by judges, blind to the preawakening conditions, according to numerous other criteria of interest.

Data Analysis

Balancing Type I and Type II error rates

By its very nature, the field of dream research tends to produce a multitude of variables, both dependent and independent. Any project that attempts to explore the relationships between dream variables and physiological variables (as is currently the case) needs to ensure that the risks of Type I and Type II errors are balanced. An approach that too stringently restricts one of these errors, while too leniently allowing for the other, runs the risk of generating spurious conclusions—be it accepting significance where it does not exist, or rejecting genuine effects that do exist. And then, as if the difficulty in balancing these errors were not enough, there is also the issue of measurement error that plays havoc with correlational relationships. Add to this the inevitability of unequal sample sizes, along with a multitude of measured and unmeasured individual differences, and the result is a very complicated analysis indeed.

Consequently, in a study such as this one—which is largely exploratory and relatively novel—no guarantee can be given as to which effects are genuine and which are not until the study has been replicated in a different sample. Therefore, it may be better to set a precedent

to be disproven, than to erroneously overlook a true and significant relationship in the data. To allow for genuine significant relationships to be revealed, and simultaneously cull those spurious relationships which add noise to the analysis, a significance level of $\alpha = .01$ was used across all inferential tests. This approach is relatively lenient in comparison to other popular methods of controlling the Type I error rate (for instance, Bonferroni correction); however, emphasis was placed on the congruence between significant results, current theories, and established findings, so as to limit the discussion and conclusions based on the present findings to those that offer true relevance in light of the aims of this research.

Dream Measures

The dream measures in this study were tested for statistical reliability using Cronbach's alpha and the intraclass correlation (ICC) coefficient. Two raters (blind to the preawakening conditions) were required to rate 70% of the dream reports according to the established criteria (see Measures). As interrater reliability was found to be satisfactory (Cronbach's $\alpha > .9$ for all objective measures; see Results) the scores given to each dream report by the first rater (R1, the author) were used for further analysis.

Furthermore, all measures that were developed for this study were assessed for reliability and validity. For the perceptual-interaction rating scale (PIRS), validity was established through comparisons with the established measure total recall count (TRC), as well as the self-rated amount of dreaming (Question 2, see Appendix B). Interrater reliability was obtained in the same way as for the established dream measures. With regards to the multidimensional dream questionnaire (MDQ), a principle components analysis was carried out to determine the simple structure, and to allow composite scores to be established from the 28 questions comprising the MDQ. These components were then compared with the original eight dream dimensions from which many of the questions were derived. In order to deal with the dependency in the data (i.e., multiple dream reports from each participant), the unstandardized residuals for each dream report were used in the principle components analysis rather than the raw scores (Figueredo, Ross, & Petrinovich, 1992).

The dream measures were also split into those that included negative recall (no recall and no content reports) and those that were based only on positive recall. The measures that included both negative and positive recall were the broad-based dream measures, intended to measure the *overall* amount of dreaming, and included total recall count, the perceptual-interaction rating scale, and amount of dreaming (Q2 of the MDQ). However, the measures of qualitative dream content variables (for instance, visual perception or everyday experience), were based *only* on positive recall. Although it has previously been suggested

that all dream measures include negative recall to avoid statistical power issues (Antrobus et al., 1995), this method was avoided in this study for the following reason: by including dream frequency in the qualitative dream content variables (i.e., including reports of no recall) the measures begin to represent overall recall rather than certain elements within the dream content. In other words, stages of sleep with a lower dream frequency may, nevertheless, have dreams that are equally visual compared to other stages of sleep with a higher dream frequency (when dreams do occur). However, if the negative recall is factored into the visual perception measure it will appear as though the former stage of sleep had less visual dreams than the latter, where in fact there was only less positive recall; for this reason, an effort has been made to differentiate between global measures of dreaming that include dream recall frequency and dream content variables that are based only on positive recall. It should be kept in mind then, that the global dream measures were always based on the full sample of dream reports in any condition, while the qualitative dream content measures were based only on the positive dream reports from that condition.

The dream measures (except for total recall count) were also all standardised to allow for easy interpretation; in most cases, the scoring units of any measure are arbitrary (except for measures like word count, for instance), and therefore, it was considered advantageous to work with standardised units as these could immediately communicate the value of any condition *as it relates to the rest of the sample* (as the standardisation was based on this sample of dream reports).

Sleep Stability in Relation to Dreaming

This analysis aimed to investigate the effects of sleep stage (light and deep NREM sleep) and sleep stability (CAP and NCAP awakenings) on dream recall. A factorial multivariate analysis of variance (MANOVA) was carried out. The MANOVA analyses were based on the unweighted participant means for each variable (the means were unweighted in an attempt to compensate for the different number of dream reports contributed by each participant). Although issues of dependency arise with this analysis because each participant contributed a mean score to each of the four conditions being compared (light CAP; light NCAP; deep Cap; deep NCAP), this approach was maintained because: i) a repeated measures analysis would only include reports for participants who had *positive recall* for all four of the conditions—this could potentially bias the sample because only frequent dream recallers would be included; and ii) by having all participants contribute to all the conditions, the main effects of sleep stage and sleep stability could still be detected above individual differences due to adequate sample size (see the section on Participants above).

CAP Arousal Subtypes and Dreaming

Due to the exploratory nature of this research, the CAP parameters of interest had to be thoroughly examined for the most relevant variables to be used for further inferential analysis. The aim was to reduce the number of CAP parameters to as few as possible to prevent issues of multicollinearity and reduced statistical power. The most relevant variables that emerged were included in a multilevel random coefficients (RC) regression model. A multilevel RC regression model was used over the Ordinary Least Squares (OLS) model, as this method is more equipped to handle dependency in the data (Cohen, Cohen, West, & Aiken, 2003). Due to the fact that each participant contributed multiple dream reports, there is a chance that dream reports from the same participant are correlated. The multilevel RC model allows for this dependency by making provision for clustering in the data. Therefore, not only will the RC model allow the extent of the dependency in the data to be assessed, but it can also be determined whether allowing for this clustering in the data is able to improve the overall model (more details appear in the Results section).

Furthermore, and as will be seen in the Results section, groups emerged from this analysis that had not originally been planned on; these groups, based on the CAP arousal subtypes, could only be attained through statistical manipulation. It is unlikely that sufficient awakenings could have been made to fulfil these criteria naturalistically, as the predictability of the CAP arousal subtypes is limited. For this reason, the groups that emerged were analysed using MANOVA analyses that took the *pooled averages* for the total dream reports in each group. Participant means could not be calculated because these groups emerged due to statistical manipulation *post hoc*. Despite the drawback of this approach, theoretically consistent results still emerged from these analyses.

RESULTS AND DISCUSSION

Participant Descriptives

The sample included 22 participants (8 males and 14 females) between ages 18 and 25 ($M = 19.71$, $SD = 1.59$). Each participant achieved a *WASI Verbal IQ* (VIQ) score above 100 ($M = 115.95$, $SD = 7.97$), as well as a *Pittsburgh Sleep Quality Index* (PSQI) score of five or less ($M = 3.38$, $SD = 1.53$). Using the *Mini International Neuropsychiatric Inventory* (M.I.N.I.) as a screening measure, all participants were free of any medical or psychiatric conditions able to cause atypical sleep architecture. All participants were aware of having dreamt at least once a week, and were able to recall their dreams at least once every two weeks; the group was therefore comprised of moderate to frequent dream recallers.

The participants spent a total of 70 nights in the sleep laboratory—three to four inconsecutive nights each, the first of which was an adaptation night, followed by two or three experimental nights. No data was collected for the first night, as this adaptation night did not include polysomnography. Furthermore, data from two participants was excluded from further analysis; in the first case the participant experienced exceptionally disrupted sleep. For the second case, the participant's dream report data was considered unreliable due to extreme sleep inertia subsequent to waking. Accordingly, data from 20 participants (6 males and 14 females; $M_{\text{Age}} = 19.65$, $SD = 1.60$), collected over 45 experimental nights, was used in the final analysis. Finally, of the 273 remaining dream reports collected six were eliminated due to poor EEG recording.

Sleep Macrostructure

While the macrostructural sleep parameters were not used for inferential analysis in this study, for descriptive purposes, the parameters have been reported in Table 1. As indicated by the mean *sleep period time* (SPT), participants were allowed to sleep for eight hours on average per night. Furthermore, the *sleep onset latency* (SOL) was in the normal range (Rama et al., 2005), and further indicated that the participants selected had good sleep quality. As participants' sleep was continuously disrupted throughout the night there was less deep sleep (S3 and S4) and an increase in the lighter NREM sleep stages (S1 and S2). Rapid eye movement sleep accounted for roughly one fifth (20%) of the total sleep time, which is close to the normative amount for this age range (Rama et al., 2005). An average sleep efficiency of 85% indicated relatively good sleep overall, despite the experimental disruptions. Figure 4 is a display of a macrostructural sleep hypnogram for a single participant, for a single night of sleep; this figure is an example of how the experimental

awakenings were made throughout the night and of the progression of the participant's sleep architecture despite the disruptions.

Table 1.
Macrostructural Sleep Parameters

Sleep Parameters	<i>M</i>	<i>SD</i>
TIB	507.19	28.50
SPT	481.96	27.84
TST	432.62	32.05
SOL	13.54	7.89
Sleep Efficiency %	85.38	5.56
WASO (min)	49.33	24.34
S1 (% TST)	10.08	5.76
S2 (% TST)	58.49	5.76
S3 (% TST)	8.43	2.83
S4 (% TST)	2.66	3.35
SWS (% TST)	11.09	4.25
REM (% TST)	20.34	5.02

Note. All measures excluding percentages are in minutes. Percentages have been calculated according to total sleep time. *M* = Mean; *SD* = Standard Deviation; TIB = time in bed; SPT = sleep period time; TST = total sleep time; SOL = sleep onset latency; SE = sleep efficiency; WASO = waking after sleep onset; S1 = stage 1; S2 = stage 2; S3 = stage 3; S4 = stage 4; SWS = slow wave sleep (S3 + S4); REM = rapid eye movement.

Figure 4. Sleep Hypnogram

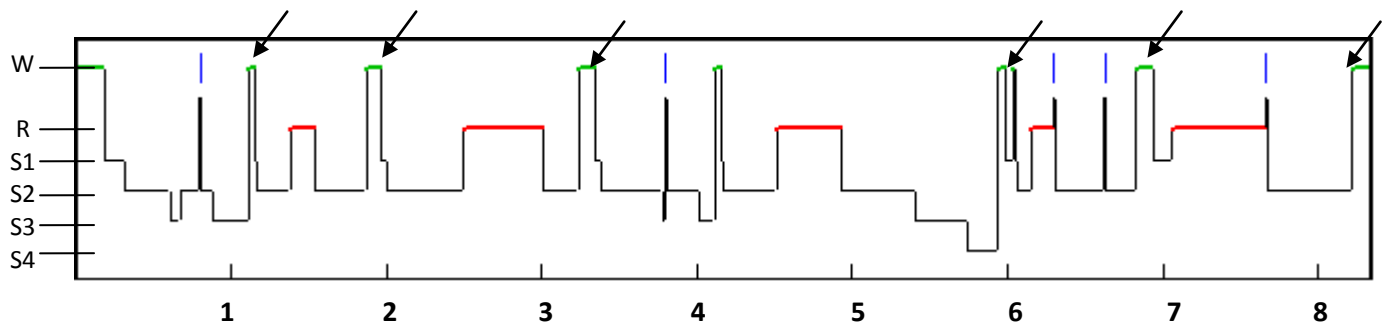


Figure 4. A sleep hypnogram for a single experimental night. Arrows indicate experimental awakenings. The numbers along the bottom of the figure indicate the number of hours asleep. The labels on the left of the hypnogram indicate stages of sleep. The green portions of the hypnogram represent periods of time when the participant was awake. The red portions of the hypnogram represent periods of REM sleep. W = waking; R = REM; S1 = Stage 1; S2 = Stage 2; S3 = Stage 3; S4 = Stage 4.

Dream Measures

A number of measures were developed for use in this study, alongside other established measures of dreaming. The newly developed measures were required to be valid and reliable to be used in further inferential analyses. It was imperative that the reported reliability for the established measures be confirmed as well. However, as the development of dream measures was not a central aim of the present study, only the essential descriptives and reliability and validity tests are reported in the main text. Where necessary, the reader is directed to the appropriate appendix for a fuller description of the measures.

The Multidimensional Dream Questionnaire

The MDQ is a self-report measure based on the eight dimensions of dreaming identified by Hauri et al. (1967), in conjunction with Panksepp's (1998) basic emotions, and a few other measures of interest (see Dream Measures). A principal component analysis (PCA) was conducted on 25 items from the MDQ to identify the simple structure of this questionnaire. Details of the full analysis appear in Appendix G. In summary, the PCA analysis resulted in a four component structure including 15 of the original items, explaining 62.97% of the variance (Table 2). The items that cluster on the same components suggest that

Component 1 represents *dream intensity*, Component 2 *pleasantness*, Component 3 *hostility*, and Component 4 *visual perceptuality*.

Reliability and Validity of the MDQ Subscales

Reliability analysis of the dream intensity, hostility, and visual perception components showed high internal consistency, with Cronbach's $\alpha > .75$ for each (Table 2). Analysis of the remaining pleasantness subscale revealed a less than optimum level of reliability, Cronbach's $\alpha = .68$. However, all item-to-total correlations were above the recommended limit of .3 (Field, 2009), and tests of *Cronbach's alpha if an item is deleted* also indicated that all items contributed to the overall reliability of this subscale. This subscale was subsequently retained for further analysis. A full reliability analysis of the MDQ subscales is reported in Appendix G.

Hauri et al.'s (1967) original factor analysis established eight independent dimensions of dreaming (see Measures); the four subscales from the MDQ were comparable to certain of these original dimensions, indicating the validity of these components in the present analysis. First, the MDQ's dream intensity component appears to be a hybrid of the vivid fantasy and active participation factors. Unlike the original analysis, bizarreness did not load significantly onto this component. Therefore, *dream intensity*, rather than vivid fantasy, was considered to be a more appropriate title for this subscale. Since active participation was intercorrelated with both the present dream intensity component, as well as Hauri et al.'s (1967) vivid fantasy dimension, it is reasonable to presume that this feature of dreaming contributes to the felt intensity of the dream as well. The next component, pleasantness, is directly comparable to the original pleasantness dimension. Furthermore, FEAR and PLAY were found in the present study to accompany pleasantness, thereby extending this dimension to include some of the appropriate basic emotions. Hostility was comprised of both verbal and physical aggression, in contrast to the original study, where each type of aggression was found to be an independent factor; the basic emotions found to accompany hostility were RAGE and DOMINANCE. Finally, visual perception is also directly comparable to the perceptuality dimension of the original study.

Table 2.

Multidimensional Dream Questionnaire – Rotated Components Matrix^a

Items ^b	Rotated Component Loadings			
	1 Dream Intensity	2 Pleasantness	3 Hostility	4 Visual
To Do	.768	.084	.053	.023
Active Thought	.744	.069	-.013	.003
Auditory	.698	-.287	.072	-.187
Emotionality	.676	.139	.177	.220
Role	.606	.051	-.071	.024
Storylikeness	.597	-.050	.096	.013
FEAR	.316	.835	-.188	.131
Pleasantness	-.106	.824	.092	-.141
PLAY	.259	-.595	-.246	.230
DOMINANCE	.004	-.104	.833	-.047
P. Aggression	-.044	-.061	.767	.070
RAGE	.136	.085	.680	.027
V. Aggression	.192	.287	.646	.128
Visual Clarity	-.056	-.005	.033	.914
Colour	-.010	-.113	.076	.891
Eigenvalues	3.49	2.08	2.73	2.19
% of variance	27.52	15.58	10.62	9.25
α	.79	.68	.76	.81

Note. Bolded numbers indicate component loadings above .4. Extraction Method: Principal Component Analysis. Rotation Method: Oblimin with Kaiser Normalization. α = Cronbach's alpha. P. Aggression = physical aggression; V. Aggression = verbal aggression.

^a Rotation converged in 10 iterations.

^b N = 159 dream reports from 20 participants.

Overall, consistency between the present analysis and previous research demonstrates the content and criterion validity of the four subscales. This analysis has additionally shown that the dimensions established by Hauri et al. (1976) remain valid for subjectively rated dream reports. The four subscales of the MDQ were subsequently used in further analyses.

Objective Dream Measures

Reliability Analysis

The three objective dream measures used in this analysis were: *total recall count* (TRC), PIRS, and the *everyday experience* (EE). Reliability coefficients for the three objective measures are reported in Table 3. Cronbach's α was $> .9$ for all objective measures,

indicating very good interrater reliability. Furthermore, the intraclass correlation (ICC), an analysis of the average rater-to-rater reliability, was also sufficiently high (all > .8). The interrater reliabilities for TRC and EE were comparable to the literature (Antrobus et al., 1995; Foulkes, Larson, & Swanson, 1969). Furthermore, the high interrater reliability for the PIRS—which was developed for this study—indicated that this measure was suitable for use in further analyses.

Table 3.
Inter-rater Reliability for the Objective Dream Measures^a

GLOBAL	Cronbach's α	Intraclass Correlation
Everyday Experience	.92***	.85***
PIRS	.92***	.85***
PSYCHOLINGUISTIC		
Total Recall Count	.98***	.96***

Note. The interrater reliabilities given are for two objective raters.

^a $N = 182$

*** $p < .0001$, 2-tailed.

Types of dream recall. Although the recollection of *any* mentation has previously been used as a criterion to dichotomise positive and negative dream recall (Foulkes, 1962), the value of differentiating between cognitive activity and dreaming has more recently been advocated (Nielsen, 2000). Consequently, each dream report was allocated to one of four categories according to its PIRS rating (Appendix E). The four groups were defined as: i) *no recall* (PIRS = 0); ii) *no content* (an entirely or almost entirely forgotten dream; PIRS = 1); iii) *cognitive activity* (static visual images, thinking, reflecting, bodily feelings, or vague or fragmentary impressions, PIRS = 2 – 4); and iv) *dreaming* (any visual-perceptual experience involving at least a single interaction between any dream character and another dream character, or any dream character and the dream environment, PIRS > 4). As these categories were found reliable (see Appendix H), they were used for further analysis as well.

Dream Composite: A Global Measure of Dreaming

Table 4 represents the Pearson correlation coefficients among nine dream variables of interest: the three objective measurements, the four MDQ subscales, as well as Questions 1

and 2 from the MDQ (see Appendix B); the relationships among the variables are with regards to sleep mentation in general, i.e., without any macro- or microstructural distinctions. A full discussion of the intercorrelations appears in Appendix I (along with the descriptive statistics for the basic emotions). The broad-based dream measures intended to represent *overall* dreaming all had moderately large and highly significant ($p < .0001$) positive relationships. In particular, *total recall count* (TRC), PIRS, and self-rated *amount of dreaming* (AD; Question 2 from the MDQ) were all highly intercorrelated. As TRC has been shown to be a reliable broad-based dream measure (Antrobus et al., 1995), the significant correlation of this measure with both AD and PIRS was an indication of the convergent validity of all three measures as broad-based dream measures. Also, and as would be expected from a broad-based dream measure, there were highly significant positive correlations with many of the qualitative dream content variables (e.g., visual perception). Based on the appropriate convergent nature of these measures and their consequent content validity, it was considered beneficial to form a composite measure of global dreaming called *dream composite* (DC).

Reliability of the Dream Composite Measure

A composite measure of these three variables (using the standardised Z scores for each) was found to have high internal consistency, Cronbach's $\alpha = .88$, ICC = .70. All *item-to-total* correlations were adequately high, with the lowest being $r = .54$. However, further exploration of *Cronbach's α if an item is deleted* indicated that if TRC was removed, the value of Cronbach's α would increase to .93. Therefore, despite the significant correlations between all items, PIRS and AD tended to be more reliably related when TRC was excluded, and consequently, only these two measures were used to create the DC measure. The resulting DC measure ranged from -1.43 to 3.13, and approximated a standard normal distribution. Total recall count was retained and analysed separately as an additional global dream measure.

Table 4.
Dream Measures: General Intercorrelations

	H	VP	P	DI	EE	DS	PIRS	TRC	AD
Hostility ^a	1	.19	-.14	.39***	.15	.21*	.29***	.32***	.16
Visual Perception ^a		1	.10	.34***	.15	.24*	.49***	.27***	.41***
Pleasantness ^a			1	-.001	-.004	.21*	.09	.05	.21*
Dream Intensity ^a				1	.14	.31***	.51***	.38***	.50***
Everyday Experience ^a					1	.16	.47***	.28***	.22*
Depth of Sleep ^b						1	.30***	.16	.35***
PIRS ^b							1	.69***	.87***
TRC ^b								1	.54***
Amount of Dreaming ^b									1

Note. Bolded values indicate statistical significance. H = hostility; VP = visual perception; P = pleasantness; EE = everyday experience; DS = depth of sleep; PIRS = perceptual-interaction rating scale; TRC = total recall count; AD = amount of dreaming.

^a Positive recall only, $n = 168$

^b All recall, including both positive and negative reports, $n = 267$

* $p < .01$, ** $p < .001$, *** $p < .0001$, 2-tailed.

The Sleep Cycle and Dreaming

The average percentage of NREM recall classified as dreaming (42.92%) was extremely consistent with two review studies ($45.9 \pm 15.8\%$; Foulkes, 1967) and ($43.0 \pm 20.8\%$; Nielsen, 2000) of NREM dream recall. Additionally, the percentage of recall classified as dreaming in REM sleep (87%) was also consistent with the figure ($81.9 \pm 9.0\%$) reported by Nielsen's (2000) review. When a more stringent definition of dreaming is employed (PIRS > 5), 74.1% of REM dreaming is still classified as dreaming, compared with only 24.88% of NREM recall. Consequently, REM dreaming was, on average, much more extensive than NREM recall in terms of ongoing activity within the dream environment. With regard to time of night effects, dreaming appeared to be enhanced in the late portion of the night, and cognitive activity drastically reduced; these findings are congruent with previous literature showing less thoughtlike activity in the later part of the night (Fosse et al., 2004; see Appendix J). Therefore, not only are the dream recall figures reported largely consistent with

past literature, but this consistency also corroborates the types of recall categories (based on the PIRS ratings) as valid and reliable.

REM sleep mentation also differed in both quality (i.e., visual perception, emotionality, dream intensity) and quantity (TRC and DC) compared with NREM mentation. These differences in dream content have been reported in previous studies (Nielsen, 2000; see Appendix J for a full analysis of the dream content variables as they relate to the sleep macrostructure). As such, the qualitative dream content measures in this study were consistent with previous findings, and were therefore considered valid and suitable for use in further analyses.

Finally, a series of analyses dividing the sleep macrostructure by the slopes of the sleep cycle (descending, trough, ascending) indicated no advantage over dividing it by sleep stage (light and deep NREM sleep; see Appendix J). Therefore, stage of sleep was used in subsequent analyses for the purposes of comparison with other dream studies. However, slope of the cycle did allow for a finer investigation of the arousal subtypes, and was subsequently retained in these analyses (i.e., in *The CAP Arousal Subtypes and Dreaming*).

Dreaming in Relation to NREM Sleep Stability/ Instability

The following analyses aimed to describe the quantity and quality of sleep mentation for awakenings following CAP and NCAP conditions, in all stages of sleep, as well as for the different parts of the sleep cycle.²⁶ It was predicted that there would be more dream recall, and more dreamlike recall, following unstable NREM sleep.

Types of Dream Recall

The types of dream recall categories were calculated using the PIRS. Each dream report was allocated one of the four types of dream recall categories. Descriptive statistics for the pooled dream report averages are reported in Table 5.

Light NREM sleep

The CAP and NCAP conditions were similar for the no recall and no content categories; in total, negative recall²⁷ was 5.40% lower for the CAP condition (28.30%) than the NCAP condition (31.20%). On the contrary, the NCAP condition had approximately twice as much cognitive activity compared with the CAP condition. With regards to dreaming, 63.30% of the recall from the CAP condition fell within this category—15.40%

²⁶ The scoring of the CAP parameters was found to be suitably reliable, and therefore, inferential statistics were carried out (see Appendix K).

²⁷ Negative recall is defined as all reports containing no recall, and is comprised of both the no recall and no content percentages.

more than the equivalent NCAP condition (47.90%). Awakenings made within 20 minutes of a REM period²⁸ yielded similar dreaming rates (47.70%) to awakenings within the NCAP condition; as such, these reports were included in the NCAP condition for all subsequent analyses.

Table 5.

Type of Dreaming for Pre-Awakening Conditions

Preawakening Condition	<i>n</i>	No Recall	White Dream	Cognitive Activity	Dreaming
Light NREM					
CAP	60	18.30	10.00	8.30	63.30
NCAP	48	20.80	10.40	20.80	47.90
< 15	19	15.80	26.30	10.50	47.40
Deep NREM					
CAP	59	44.10	16.90	11.90	27.10
NCAP	19	42.10	21.10	26.30	10.50

Note. All values, except for *n*, are percentages. Each row (i.e., preawakening condition) adds to 100%. The percentages are calculated according to the number of pooled dream reports from all participants, for each condition. Light NREM sleep refers to S2 sleep; Deep NREM sleep refers to S3 and S4 sleep.

CAP = cyclic alternating pattern (unstable sleep); NCAP = non-cyclic alternating pattern (stable sleep); < 15 = dream reports collected within 15 minutes of the end of a REM period. All < 15 minute reports were classified as NCAP.

Deep NREM sleep

CAP and NCAP conditions were comparable for the negative recall rates (61.0% vs. 63.2%, respectively). The NCAP awakenings once again yielded more than twice as much cognitive activity than the CAP awakenings. In contrast, CAP awakenings yielded more than twice as much recall in the dreaming category, compared with NCAP awakenings. When types of recall were examined as a function of stage of sleep, S3 had substantially less negative dream recall than S4 (51.9% vs. 71.4%, respectively). Also, the CAP-NCAP difference in mentation classified as dreaming was more substantial for S3 (35% vs. 8.3%,

²⁸ Even though an effort was made to make experimental awakenings outside of this time period, post hoc verifications of the scoring indicated that a small number of reports still fell within 15 minutes of the end of a REM period. As these reports were comparable to other NCAP reports, they were retained for further analysis.

respectively), than for S4 (10.5% vs. 14.3%, respectively).²⁹ Consequently, it may be the case that unstable sleep gives rise to more dream recall in S3 rather than S4 sleep. However, due to the uneven sample sizes and small samples (when split this way) inferential statistics could not be performed.

Overall, when stage of sleep was collapsed, differences in types of recall for the stable (NCAP) and unstable (CAP) conditions did not reach significance, $\chi^2(3) = 5.90, p = .06$. However, there was a trend towards more cognitive activity in stable (NCAP) conditions ($Z = 1.5, p = .12$).

Repeated Measures Analyses: Global Measures

Averages were calculated for each participant, for each of the four preawakening conditions. The number of reports for each participant for each awakening are reported in (Appendix L). The broad-based, or global, dream measures chosen for this analysis were: TRC, DC, remembrance,³⁰ and subjective depth of sleep. A series of paired samples *t* tests were run to investigate the differences in DC for CAP and NCAP conditions, in light and deep NREM sleep. Both *t* tests were run as one tailed as an effect in the direction of the CAP condition was predicted. Descriptive statistics are reported in Table 6. The results of the *t* tests indicated no difference in DC scores between CAP and NCAP conditions for light NREM sleep, $t(19) = 1.59, p = .06, r = .34$,³¹ however, differences between CAP and NCAP conditions were significant for deep NREM sleep, $t(13) = 2.60, p = .01, r = .58$. In general, light NREM mentation had significantly higher DC values than deep NREM mentation, $t(13) = 4.07, p = .0005, r = .75$.

Furtherore, a series of Wilcoxon signed-rank tests for nonparametric repeated measures data were run to test the differences for CAP and NCAP conditions, in light and deep NREM sleep. For TRC, there was no difference in light NREM sleep for the CAP and NCAP conditions, $z = -.14, p = .45, r = -.02$.³² Alternatively, TRC was significantly increased for CAP activity in deep NREM sleep, $z = -2.04, p = .020, r = -.39$. Furthermore, deep NREM sleep had a significantly lower TRC than light NREM sleep, $z = -2.355, p = .008, r = -.45$. Memory accuracy for light NREM sleep did not differ for CAP-NCAP

²⁹ S3: CAP, $n = 40$, NCAP, $n = 12$; S4: CAP, $n = 19$; NCAP, $n = 7$.

³⁰ Question 3 from the MDQ asked participant to rate how accurately they were able to remember their dreams. Negative reports were given values of 0 (see Appendix B.).

³¹ Field (2009) suggests converting the *t* statistic into a value of *r* to obtain the effect size: $r = \sqrt{\frac{t^2}{t^2 + df}}$

³² Field (2009) suggests converting the *z* statistic into a value of *r* to obtain the effect size: $r = \frac{z}{\sqrt{N}}$

conditions, $z = -.57$, $p = .29$, $r = -.09$, but did for deep NREM sleep, $z = -2.84$, $p = .001$, $r = .54$, with CAP having significantly better remembrance. Finally, subjective depth of sleep did not differ for NCAP and CAP conditions, for either light, $z = -1.11$, $p = .14$, $r = -.18$, or deep, $z = -1.03$, $p = .15$, $r = -.19$, NREM sleep.

The repeated measures results indicated more dreaming for unstable deep NREM compared with stable deep NREM sleep. Differences between light and deep NREM mentation in general, collapsing CAP-NCAP conditions, were substantial for the DC and TRC measures.

Multivariate Analysis: Dream Quality³³

Each of the four MDQ subscales³⁴ were entered together as multiple dependent variables in a factorial multivariate analysis of variance (MANOVA), that included the light-deep and CAP-NCAP conditions as independent variables. Overall, using Hotelling's trace statistic, no significant effects were found for any of the MDQ subscales for either light-deep NREM sleep, $T = .77$, $F(5, 50) = 1.77$, $p < .14$, or the CAP-NCAP condition, $T = .177$, $F(5, 50) = .53$, $p < .75$. Furthermore, there was no significant interaction between the light-deep and CAP-NCAP effects for the MDQ subscales overall, $T = .07$, $F(5, 50) = 0.70$, $p < .63$. Descriptive statistics are reported in Table 6.

The Basic Emotions

Seven of the basic emotions (excluding LUST) were entered together as multiple dependent variables in a factorial MANOVA, that included the light-deep and CAP-NCAP conditions as independent variables. Overall, using Hotelling's trace statistic, no significant main effects were found for any of the basic emotions for light-deep NREM sleep, $T = .19$, $F(6, 49) = 1.55$, $p < .18$, or the CAP-NCAP condition, $T = .14$, $F(6, 49) = 1.12$, $p < .37$. There was no significant interaction between the light-deep and CAP-NCAP effects for the MDQ subscales overall, $T = .14$, $F(6, 49) = 1.16$, $p < .34$ (see Table 6).

³³ These analyses assume that the groups being compared are independent; the data did not fulfil this requirement. However, the multivariate results were reported as these had the advantage of including all of the data, and not only data for participants who had positive recall for all conditions (see Methods).

³⁴ The subscales were all standardised, with a mean of approximately 0 and a standard deviation of approximately 1. The unweighted means for each participant for each condition were used and not the individual dream reports.

Table 6.
Quantitative and Qualitative Measures for Stable and Unstable NREM Sleep

A. Repeated Measures Analysis of Global Dream Measures				
Dream Measures	Light NREM Sleep ^a		Deep NREM Sleep ^b	
	CAP	NCAP	CAP	NCAP
TRC	23.23	25.91	17.99	10.21
Dream Composite	0.12	-0.17	-0.39	-0.65
Remembrance	1.30	1.40	1.44	0.71
Depth of Sleep	3.40	3.51	3.47	3.32
B. Multivariate Analysis of positive dream recall variables				
MDQ Subscales				
Visual Perception	-0.15	0.08	-0.30	-0.78
Pleasantness	-0.11	0.01	-0.13	0.02
Hostility	-0.06	0.00	-0.25	-0.20
Dream Intensity	-0.03	-0.11	-0.40	-0.41
Bizarreness	2.62	2.59	2.70	2.29
Basic Emotions				
CARE	0.24	0.41	0.24	0.43
PLAY	0.52	0.62	0.54	0.29
SEEKING	0.52	0.85	0.81	1.14
FEAR	0.48	0.45	0.48	0.00
GRIEF	0.11	0.03	0.04	0.14
RAGE	0.27	0.34	0.11	0.00
LUST	-	-	-	-
DOMINANCE	0.29	0.22	0.14	0.29

Note. The unweighted means for each participant were used (not individual dream reports) to calculate the averages. The DC measure is the average of the standardised PIRS and AD measures (see the Dream Measures section of this chapter). Remembrance was measured on a scale from 0 to 3, with 3 indicating the best remembrance. Depth of sleep was measured on a scale from 0 to 4, with 4 being the deepest sleep. The MDQ subscales are all standardised measures, with an approximate mean of 0 and *SD* of 1. Bizarreness was measured on a scale from 1 to 5, with 5 being completely bizarre. The raw scores for the basic basic emotions have been used (0 – 3). Light NREM sleep includes S2; Deep NREM sleep includes S3 and S4. TRC = total recall count; CAP = cyclic alternating pattern; NCAP = non-cyclic alternating pattern (stable sleep); MDQ = multidimensional dream questionnaire (see Appendix B);

^a *n* = 20 participants for repeated measures analyses for light NREM sleep.

^b *n* = 14 participants for repeated measures analyses for deep NREM sleep.

Sleep Parameters for CAP and NCAP Conditions

Previous literature has shown that the period of time spent in any sleep stage prior to waking is able to influence dream recall (Tracy & Tracy, 1974). Furthermore, longer sleep-to-wake periods have been shown to negatively correlate with dream recall (Shapiro, Goodenough, & Gryler, 1963). Therefore, both time-in-stage (TIS) and sleep-to-wake (STW) time were calculated for each of the CAP and NCAP conditions in light and deep NREM sleep, in order to control for any confounding influences. A series of one way ANOVAs were run on the data. Descriptive statistics for both light and deep NREM sleep are reported in Table 7.

Light NREM Sleep

STW times for CAP and NCAP conditions differed significantly, $F(1, 129) = 5.94, p = .016, \omega = .19$. Similarly, TIS was significantly longer for CAP conditions compared with NCAP conditions, $F(1, 129) = 10.05, p = .002, \omega = .25$. Pearson correlation coefficients were used to further determine whether TIS or STW times had any relationship with the global DC measure in light NREM sleep. No relationship between percentage dreaming and STW time was apparent for either stable or unstable light NREM sleep (see Table 7). Noteworthy, is that the difference in STW times did not reach significance at the adjusted $\alpha = .01$ level, and there was only a 1.45 second mean difference in waking times between the two conditions. This difference then, although significant, may not have practically had any impact on dream recall.

Deep NREM sleep

Differences for STW time, $F(1, 80) = 0.04, p = .83, \omega = .11$, and TIS, $F(1, 80) = 1.67, p = .20, \omega = .01$, did not reach significance for CAP and NCAP conditions in deep NREM sleep. Pearson correlation coefficients were again used to determine whether increased STW time or TIS were related to less dreaming in deep NREM sleep. No relationship between percentage dreaming and STW time, nor percentage dreaming and TIS, was apparent for either condition in deep NREM sleep (see Table 7). Sleep-to-wake times were significantly longer for deep NREM sleep ($M = 11.06, SE = 0.73$) than light NREM sleep ($M = 8.94, SE = 0.37$), $t(32) = 2.619, p < .007, r = .42$, after collapsing CAP and NCAP conditions; this is consistent with a lower overall dream recall (and generally more thoughtlike recall) for deep NREM sleep than light (Nielsen, 2000).

Table 7.
Pooled Averages - All NREM dream reports – Sleep Parameters

	Light NREM Sleep				Deep NREM Sleep			
	CAP ^a		NCAP ^b		CAP ^c		NCAP ^d	
	STW	TIS	STW	TIS	STW	TIS	STW	TIS
<i>M</i>	9.96	28.65	8.51	20.97	11.46	13.43	11.21	16.49
<i>SD</i>	3.05	14.69	3.71	12.99	4.53	8.09	4.50	11.88
	Correlations with Dream Composite							
<i>r</i>	-.09	-.08	-.05	.09	-.02	-.04	-.26	-.29
<i>p</i>	.46	.56	.69	.48	.88	.74	.28	.23

Notes. Pooled averages for all dream reports. Sleep to wake times are reported in seconds. Time in stage is reported in minutes. The Pearson correlations and associated significance values are an indication of the strength of the relationship of each variable with global dreaming, as measured with DC. STW = sleep-to-wake time; TIS = time in stage.

^a *n* = 64 dream reports

^b *n* = 67 dream reports

^c *n* = 63 dream reports

^d *n* = 19 dream reports

The CAP Arousal Subtypes and Dreaming

These analyses aimed to investigate whether the CAP arousal subtypes were related to dream recall in any particular way. It was predicted that the faster A₂ and A₃ arousal subtypes would be related to more dream recall, while the slower A₁ type would be related to less dream recall.

Analysis of the Arousal Subtypes in Unstable NREM Sleep

CAP Parameters of Interest

Arousal events occurring in three different time periods were considered: a) the number of each subtype in the entire CAP sequence preceding awakening; b) the number of each subtype for the last three minutes preceding the awakening; and c) the number of each subtype for the last minute preceding awakening; as well as d) the length of the entire CAP sequence preceding the awakening; and e) the temporal proximity of the last arousal to the awakening. Descriptive statistics are reported in Table 8. The average CAP sequence length preceding waking was approximately five minutes (*M* = 319, *SD* = 237 seconds). Sequences

in light NREM sleep also contained many more A_2 and A_3 arousals, with desynchronised arousal subtypes almost entirely absent for CAP sequences in deep NREM sleep.

General intercorrelations. Table 9 shows the Pearson correlation coefficients between all of the CAP variables of interest. As expected, many of these variables are highly intercorrelated, as most are partly comprised of the same events. For instance, the totals for each subtype inevitably include those arousals that occurred in the last minute and the last three minutes as well. Despite this overlap, all parameters were considered so that those best for further inferential analysis could be identified. The dependent variable chosen as a global measure of dreaming was DC. The CAP parameters that were significantly correlated with the DC measure were the A_1 and A_2 arousals in the *last minute* and *last three minutes* before waking. The A_3 subtype was not significantly correlated with the DC measure for any time period before waking; this lack of correlation may have been due to the relative minority of A_3 arousals prior to waking (see Table 8).

The A_1 - A_2 difference. The A_1 and A_2 CAP subtypes in the three minutes prior to waking were identified as being most significantly related to DC. However, these two variables were also very significantly negatively correlated with one another, and appeared to have opposite effects on dreaming: The slow A_1 subtype reduced dreaming, while the faster A_2 type enhanced it (Table 9). As different effects on dream recall were hypothesised for the A_1 and A_2 subtypes, a potential remedy to this situation was to create a single variable measuring *the number of A_1 arousals relative to A_2 arousals*. This new variable, the *A_1 - A_2 difference*, was created by subtracting the number of A_2 arousals from the number of A_1 arousals in the last three minutes before waking. As such, a positive number indicates more A_1 arousals in the last three minutes, a zero indicates an equal number of A_1 and A_2 arousals, and a negative number is an indication that there were more A_2 than A_1 arousals in the last three minutes.

Table 8.
Descriptives – CAP Measures for Light and Deep NREM Sleep

Light NREM (n = 54)		M	SD	Min	Max	%
CAP Seq. Length (sec)		215.76	171.05	37	758.00	-
Total						
	A ₁	4.24	4.34	0	24	56.07
	A ₂	2.57	2.99	0	15	35.52
	A ₃	0.59	1.42	0	8	8.51
3 Minutes						
	A ₁	2.70	1.88	0	7	55.28
	A ₂	1.68	1.68	0	7	33.32
	A ₃	0.47	1.01	0	4	11.40
1 Minute						
	A ₁	1.04	1.01	0	3	49.54
	A ₂	0.80	0.88	0	3	40.59
	A ₃	0.13	0.39	0	2	9.88
Deep NREM (n = 53)		M	SD	Min	Max	%
CAP Seq. Length (sec)		403.57	241.47	68.00	> 900.00	-
Total						
	A ₁	15.06	11.15	2	56	97.23
	A ₂	0.43	0.82	0	3	2.73
	A ₃	0.02	0.14	0	1	0.03
3 Minutes						
	A ₁	5.89	1.70	3	11	97.33
	A ₂	0.17	0.43	0	2	2.67
	A ₃	0.00	0.00	0	0	0.00
1 Minute						
	A ₁	2.15	0.91	1	4	96.86
	A ₂	0.08	0.27	0	1	3.14
	A ₃	0.00	0.00	0	0	0.00

Note. All CAP parameters measured in seconds. Descriptives for each of the subtypes are calculated for the total CAP sequence preceding the awakening (*Total*); for the last three minutes preceding the awakening (*3 Minutes*); and for the last minute preceding the awakening (*1 Minute*). The subtypes are calculated as a percentage of each group. CAP Seq. Length = The duration in seconds of the CAP sequence immediately preceding the experimental awakening.

Table 9.
CAP Measures: General Intercorrelations

	SL	TSA	Total			Last 3 min			Last 1 min			DC
			A ₁	A ₂	A ₃	A ₁	A ₂	A ₃	A ₁	A ₂	A ₃	
Seq. Length	1	-.18	.89**	.10	.09	.52**	-.22	.01	.30**	-.29**	.00	-.23
Time Since A		1	-.27**	.14	.05	-.29**	.13	.06	-.43**	.01	.01	-.10
Total A ₁			1	-.19	-.18	.72**	-.37**	-.24	.52**	-.36**	-.18	-.25
Total A ₂				1	.34**	-.42**	.73**	.28**	-.45**	.52**	.12	.25
Total A ₃					1	-.43**	.10	.95**	-.35**	.06	.66**	.13
A ₁ (3 min)						1	-.58**	-.47**	.77**	-.52**	-.39**	-.37**
A ₂ (3 min)							1	.10	-.58**	.79**	.01	.39**
A ₃ (3 min)								1	-.39**	.07	.64**	.13
A ₁ (1 min)									1	-.54**	-.27**	-.28**
A ₂ (1 min)										1	-.14	.32**
A ₃ (1 min)											1	.05
Dream Composite												1

Note. Bolded values indicate statistical significance. Correlations have been calculated for light and deep unstable NREM sleep ($n = 107$). Total = Total CAP sequence; Last 3 min = The last three minutes preceding the experimental awakening; Last 1 min = The last minute preceding the experimental awakening; SL = sequence length; Time Since A = The duration of time in seconds since the last arousal subtype; Total A₁, A₂, A₃ = The total number of each subtype in the CAP sequence preceding waking; A₁, A₂, A₃ (3 min) = The total number of each subtype in the last three minutes prior to waking; A₁, A₂, A₃ (1 min) = The total number of each subtype in the last minute before waking. DC = Dream Composite.

* $p < .05$, ** $p < .01$, 2-tailed.

The A₁-A₂ difference for the last three minutes before waking, *in light NREM sleep*,³⁵ was regressed on DC. The overall model was significant, $F(1, 52) = 9.15$, $p < .004$, indicating that the A₁-A₂ difference continued to share a significant amount of the variance in the DC

³⁵ The A₁-A₂ difference becomes redundant for deep NREM sleep as there were no cases with an equal number of A₁ and A₂ arousals, or more A₂ than A₁ arousals, in the three minutes preceding waking. Therefore, all analyses involving the A₁-A₂ difference were run on the light NREM CAP data ($n = 54$).

measure, $R = .39$, $R^2 = .15$. More importantly, though, the amount of variance accounted for by the A_1 - A_2 difference in the last three minutes before waking was exactly the amount of variance accounted for by the individual A_1 and A_2 predictors, $R = .39$, $F(2, 51) = 4.59$, $p < .015$, and therefore, the original relationship between the predictors and DC measure was retained. The negative β -value for the A_1 - A_2 difference was also highly significant, $\beta = -.39$, $t(52) = -3.03$, $p < .004$, and allowed for a less ambiguous interpretation of the results: As the A_1 - A_2 difference becomes more positive (more A_1 than A_2 arousals) so DC scores decrease, but as the A_1 - A_2 difference becomes negative (more A_2 than A_1 arousals), DC scores increase. It appears, then, that dream recall is influenced by the number of A_1 or A_2 arousals *relative* to one another, rather than an absolute amount of either prior to waking.

The only other CAP variables significantly related to DC were the A_1 and A_2 CAP subtypes in the last minute prior to waking. The A_1 - A_2 difference for the last minute before waking was calculated and entered as a second step in the OLS multiple regression analysis to see whether this variable counted for any variance in addition to the A_1 - A_2 difference in the last three minutes. This variable accounted for no additional variance, $\Delta R = .001$, $p = .79$. Therefore, the phasic arousal activity in the last three minutes appeared to be the best predictor of dream recall, and was subsequently included in a multilevel random coefficients (RC) regression model.

Multilevel Model: Light NREM Sleep

Predictor and outcome variables. The outcome variable in this multilevel analysis was the global dream measure, DC. The predictor variable was A_1 - A_2 difference. Descriptives are displayed in Table 10.

Table 10.

Descriptives for the Random Coefficients Regression Model

Variable	<i>n</i>	<i>M</i>	<i>SD</i>	Min	Max
Dream Composite	52	0.12	0.85	-1.43	1.52
A_1 - A_2 Differences	52	1.02	3.03	-6.00	7.00

Note. Dream composite is the standardised average of self-rated amount of dreaming (AD) and objectively rated dream recall and dreamlikeness (PIRS; see Results, Measures), and is a global dream measure that includes dream frequency as well as overall dream quality. The A_1 - A_2 difference is the number of A_1 arousals relative to A_2 arousals in the three minutes prior to waking, with a negative number indicating more A_2 arousals and a positive number indicating more A_1 arousals. Min = minimum; Max = maximum.

Dream reports. The number of reports contributed by each participant is shown in Appendix L. Four reports following an incomplete CAP sequence were excluded, as the relationship to dream recall for these conditions appeared to differ to those awakenings made after longer CAP sequences. Furthermore, awakenings made during a sleep stage shift—less than five minutes within a stage of sleep—were also excluded from the analysis. These criteria resulted in the exclusion of 10 dream reports. After these reductions, two participants were only able to contribute a single dream report to the analysis (P11 and P14); these participants' data were therefore excluded from the RC multilevel model, as power for the model is based on the number of reports *per person* (group) and a group contributing only one data point would drastically reduce the ability of the model to detect clustering in the data. Consequently, the final sample included 52 dream reports from 18 participants.

Calculation of the dependency in the data. The *intraclass correlation* (ICC) is a measure of the degree of dependence within a dataset as a result of nested data and repeated measures designs. The ICC ranges from 0 to 1, with 0 indicating complete independence of observations, and 1 indicating complete dependence.³⁶ For this analysis, using DC as the outcome variable, the ICC = 0.08.³⁷ Even though the dependency in the data was found to be relatively little, an OLS regression model that does not take account of clustering may still lead to an overestimation of the significance of predictors; as a result, a RC multilevel analysis was run on the data.

Testing fixed parts of the RC model. An OLS analysis of the fixed effects is identical to the multiple regression analysis previously run on the A₁-A₂ difference (above). As before, the A₁-A₂ difference had a negative correlation with DC scores (see Table 11). This indicated that the more A₁ arousals *relative* to A₂ arousals present in the three minutes before waking, the lower the DC scores. Specifically, for every unit change in the A₁-A₂ difference, there was change of 0.12 in the DC measure; because the A₁-A₂ difference is negatively correlated with DC, a unit increase in the A₁-A₂ difference resulted in a decrease of 0.12 in DC, while a unit decrease in the A₁-A₂ difference resulted in an increase of 0.12 in DC. The intercept (average DC value) when the A₁-A₂ difference was equal to zero (i.e., there were an equal number of A₁ and A₂ arousals before waking), was 0.25. The *predicted* and *actual*

³⁶ An assumption underlying the ordinary least squares (OLS) regression model is that the ICC = 0; i.e., that every case is independent and that there is no clustering or dependency in the data. Dependency in an OLS model can result in *alpha inflation*, or the overestimation of significance (Cohen et al., 2003).

³⁷ ICC = Variance of Intercepts/(Variance of Intercepts + Residual) = .058/ (.058 + 0.66) = 0.08; these values were derived from an unconditioned cell means analysis (random effects ANOVA; Cohen et al., 2003).

relationships between DC and the A_1 - A_2 difference are illustrated in Figure 3 and Figure 4, respectively.

Table 11.

A. OLS Regression for Fixed Effects: Prediction of DC from A_1 - A_2 Difference

	<i>B</i>	Std. Error	β	<i>Df</i>	<i>t</i>	<i>p</i>	95% Confidence Interval for <i>B</i>	
							Lower	Upper
Intercept	.247	.11	-	18.77	2.23	.057	-0.01	0.50
A_1 - A_2	-.123	.04	-.44	47.65	-3.51	.001	-0.19	-0.05

B. Random Coefficient Regression: Addition of random intercepts for participants

	Estimate	Std. Error	Wald Z	<i>p</i>
Level 1 residual	.53	.13	4.23	.0001
Variance intercepts	.05	.09	0.57	.57

Note. Model Parameters: -2 LL = 118.724, and Total Parameters = 4; $R = .39$, $R^2 = .15$. *B* = Beta coefficient; Std. Error = standard error; β = Standardised Beta coefficient; *Df* = degrees of freedom;

The relationship between the A_1 - A_2 difference and dreaming. The horizontal axis in Figures 3 and 4 represents a DC score of 0; because DC is standardised, this represents the *mean* value for all dream reports in the study (including REM). The bold numbers running parallel to the horizontal axis represent values for the A_1 - A_2 difference, with positive numbers indicating more A_1 arousals relative to A_2 arousals, and negative numbers representing the opposite; a value of zero indicates an equal number of A_1 and A_2 arousals in three minutes prior to waking. Moreover, the DC scores mean also conveniently roughly demarcates recall considered dreaming from recall considered to be more fragmented/ conceptual/ cognitive activity. On average, DC scores > 0.41 indicated more extensive dreaming (PIRS > 6). As the number of A_1 arousals relative to A_2 arousals increased, so DC scores decreased, and after a positive value of 2 for the A_1 - A_2 difference DC scores fall below the mean. Alternatively, as the number of A_2 arousals relative to A_1 arousals increased, so does dream recall (DC), and with A_1 - A_2 values < -3 the DC mean approximates and even exceeds that of the REM mean ($M = 0.79$). However, as shown in Figure 5—a distribution of A_1 - A_2 values—negative A_1 - A_2 difference values were less common than positive values. Also notable, is that A_1 - A_2 values < 2 are above the mean for light NREM sleep in *general*

(i.e., light NREM sleep after the CAP conditions have been collapsed). While the actual relationship between the A_1 - A_2 difference and DC (Figure 4) differed from the predicted values, the general trends were the same; negative A_1 - A_2 values were mostly above the mean, while positive values above one were generally below the mean for DC. Therefore, the predicted values provide a useful theoretical representation of the trends in the data.³⁸

Finally, the standard error for the A_1 - A_2 difference B -coefficient was small ($SE = 0.04$), and the 95% confidence interval did not cross zero (Table 11), providing further evidence that the relative number of A_1 to A_2 arousals in the three minutes prior to waking were significantly associated with changes in dream recall. According to the 95% confidence intervals for the B -coefficient every unit change in the A_1 - A_2 difference can cause an inverse change in the DC measure by ± 0.05 to ± 0.19 .

³⁸ The model was found to be relatively stable and none of the assumptions were grossly violated; therefore, this very basic model and its predictions may prove to be relatively generalizable (see Appendix M).

Figure 5. The Predicted Relationship between Dreaming and A_1 - A_2 Difference

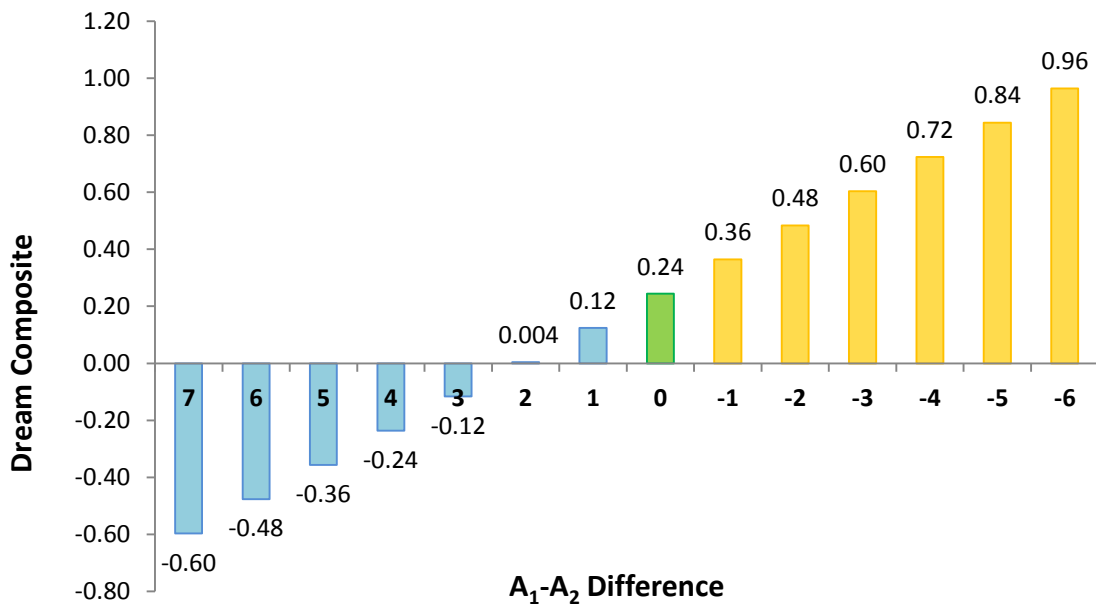


Figure 5. The predicted relationship between dreaming and the A_1 - A_2 difference in light NREM sleep. The dream composite variable is standardised; values below 0 on the y-axis are below the mean for all dream reports, while values above 0 are above the mean for all dream reports ($n = 267$). The A_1 - A_2 difference is inversely related to dreaming. The more A_1 arousals relative to A_2 arousals (positive values along the x-axis), the less dream recall. The more A_2 arousals relative to A_1 arousals (negative values along the x-axis), the more dream recall. The value of 0 on the horizontal axis indicates an equal number of A_1 and A_2 arousals.

Figure 6. The Present Relationship between Dreaming and A_1 - A_2 Difference

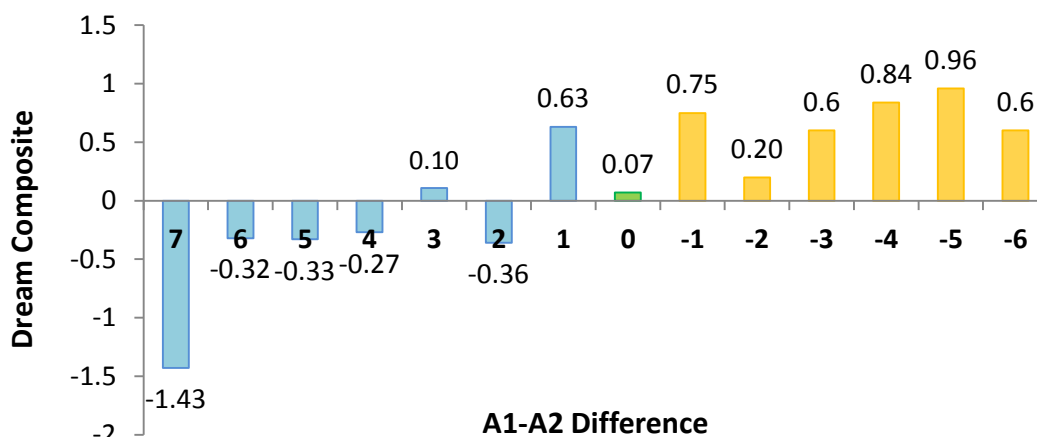


Figure 6. The empirical relationship between dreaming and the A_1 - A_2 difference ($n = 52$) in light NREM sleep. As above, the A_1 - A_2 difference is inversely related to dreaming (measured with the dream composite variable). More A_1 arousals relative to A_2 arousals have DC scores below the mean (below 0 on the y-axis). More A_2 relative to A_1 arousals result in recall above the mean.

Figure 7. Distribution of the A₁-A₂ Difference Values

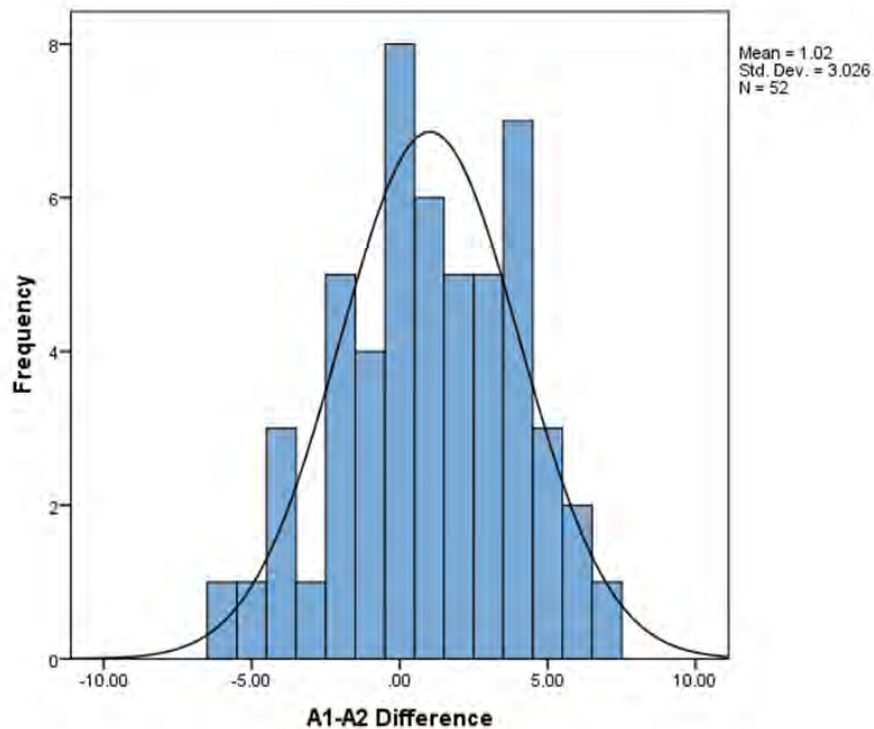


Figure 7. Histogram of the distribution of the values for the A₁-A₂ difference in light NREM sleep ($n = 52$). Values below 0 indicate more A₂ relative to A₁ arousals, whereas values above 0 indicate more A₁ relative to A₂ arousals. A value of zero indicates an equal number of A₁ and A₂ arousals in the three minutes prior to waking. The distribution is slightly negatively skewed, with more positive values than negative values.

Testing random parts of the RC model: random intercepts. The second part of the RC analysis made provisions for clustering in the data by allowing the intercepts in the model to vary. The results of this analysis are shown in Table 11. In order to determine whether allowing the intercepts to vary significantly improved the model, the change in the -2 LL statistic was examined. In the previous model, the -2 LL was 119.12, based on three parameters; in this model, the -2 LL was 118.72 based on four parameters. The $\chi^2_{\text{Change}} = 119.12 - 118.72 = 0.40$, $df_{\text{Change}} = 4 - 3 = 1$, was found to be nonsignificant,³⁹ indicating that the intercepts did not differ across groups, and therefore, that accounting for clustering in the

³⁹ The critical values for the chi-square statistic for one degree of freedom are 3.84 ($p < .05$) and 6.63 ($p < .01$).

data did not significantly improve the fit of the model. A further indication that there was no significant clustering in the data was the lack of change in the standard error and significance values for the outcome variable, the A_1 - A_2 difference. As a result, the RC multilevel model was essentially identical to the OLS regression model. This conclusion is somewhat counterintuitive though, as the DC scores for each participant for light NREM sleep CAP awakenings appeared, at face value, to vary significantly. A one way ANOVA verified that overall differences for DC between participants were nonsignificant, $F(18, 39) = 1.19, p < .32$. However, it might still be the case that real differences do exist, as the unequal and relatively small sample sizes (dream reports for each participant) likely resulted in some loss of power.

Sleep Parameters: A_1 - A_2 Difference

Sleep Cycle Distribution

Instead of using a continuous value for the A_1 - A_2 difference, this variable was split into three groups for the purpose of further descriptive and inferential statistics: i) values from -6 to -1, or A_2 majority; ii) 0 - 1, or approximately *equal A_1 and A_2* ; iii) values from 2 to 7, or A_1 majority. The distribution of these three groups according to the different portions of the sleep cycle was examined, along with differences in TIS and STW time. Descriptive statistics are reported in Table 12.

Most of the awakenings for the A_2 majority and equal groups fell on the ascending slope of the cycle, whereas the A_1 majority group was more evenly split along the different portions of the sleep cycle. This trend is consistent with the ultradian distribution of the CAP arousal subtypes (see Introduction). Additionally, the A_2 majority groupings were more common in the second half of the night; this was reiterated by the fact that the maximum number of A_2 arousals relative to A_1 arousals for the early half of the night was -4, compared to -6 for the late half of the night. However, the average DC scores were found to be *higher* for this group for the early half of the night ($M = 0.54, SD = 0.45$), than for the late half ($M = 0.44, SD = 0.76$). The A_1 majority group had only a slightly higher DC average for the late half of the night ($M = -0.23, SD = 0.89$), than the early half ($M = -0.29, SD = 0.75$). A one way ANOVA showed that there were no significant differences between the A_1 - A_2 groups for either TIS, $F(2, 51) = .702, p < .50$, or STW time, $F(2, 51) = .039, p < .96$. Therefore, significant differences between the A_1 - A_2 groups could not be accounted for by differences in these sleep parameters.

Table 12.
Parameters for the A_1 - A_2 Differences for Sleep Stage

	A ₂ Majority ^a	Equal ^b	A ₁ Majority ^b
S2 SLEEP (light NREM)			
Descending (n = 9)	1	3	5
Trough (n = 13)	2	1	10
Ascending (n = 32)	13	10	9
1 st Half (%)	37.50	42.90	54.20
2 nd Half (%)	62.50	57.10	45.80
Time in Stage (min)	33.08	33.52	29.05
Sleep-to-Wake (sec)	10.11	9.86	10.15
S3 & S4 SLEEP (deep NREM)			
Descending (n = 3)	-	-	3
Trough (n = 50)	-	-	50
Time in stage (min)	-	-	14.37
Sleep-to-wake (sec)	-	-	11.94

Note. A₂ Majority = Majority A₂ arousals prior to waking; Equal = An equal number of A₁ and A₂ arousals prior to waking; A₁ Majority = Majority A₁ arousals prior to waking.

^a Dream reports from 14 participants contributed to this group.

^b Dream reports from 12 participants contributed to this group.

^c Dream reports from 15 participants contributed to this group.

Dream Measures: Light NREM

A series of nonparametric tests were run to determine whether there were any significant differences in global dreaming, the basic emotions, or dream quality, for the A₁-A₂ groups (as described above). Results are reported in Table 13.

Global Dreaming

The broad-based, or global, dream measures chosen for this analysis were TRC, DC, and subjective depth of sleep. Each of the global dream measures were entered together as dependent variables in a Kruskal-Wallis test, that included the three A₁-A₂ groups as an independent variable. Overall, DC was found to be very significantly affected by the difference in A₁ and A₂ arousals prior to waking, $H(2) = 11.82, p < .001$, while the effect of the A₁-A₂ difference on TRC only trended towards significance, $H(2) = 5.58, p < .062$.

Subjective depth of sleep did not differ significantly between the three conditions, $H(2) = 1.51, p = .45$. A Jonckheere's test revealed a significant trend in the data for both TRC, $J = 327.5, t(2) = -2.34, p < .008, r = -.32$, and DC, $J = 373, t(2) = -3.22, p < .001, r = -.44$. Mann-Whitney U tests were used to follow up these findings. The A₂ majority group had significantly increased TRC compared with the A₁ majority group, $U = 112, p < .01, r = -.35$, but not with the equal group, $U = 98.50, p < .59, r = -.10$. With regards to DC, the A₂ majority group again had scores that were significantly higher than the A₁ majority group, $U = 78.50, p < .001, r = -.50$, but not the equal group, $U = 111.50, p < .99, r = .001$. Furthermore, the equal group also had significantly higher DC than the A₁ majority group, $U = 86, p < .006, r = -.41$. Therefore, as the number of A₁ arousals increased relative to the number of A₂ arousals, dream recall decreased.

MDQ Subscales and Bizarreness

Each of the four MDQ subscales⁴⁰ and bizarreness (everyday experience) were entered together as multiple dependent variables in a Kruskal-Wallis test, that included the three A₁-A₂ groups as an independent variable. Overall, none of the dream content measures were significantly affected by the number of A₁ arousals relative to A₂ arousals; however, Jonckheere's test did reveal a significant trend in the data for hostility, $J = 155.5, t(2) = -2.11, p < .02, r = -.44$. Mann-Whitney U tests were used to follow up this finding. The A₂ majority group had significantly more hostile dreams than the A₁ group, $U = 47, p < .001, r = -.44$. However, there was no difference in hostility for the A₂ majority and the equal group, $U = 60, p < .12, r = -.24$, nor for the equal and A₁ majority group, $U = 48.5, p < .27, r = -.10$. Therefore, despite the nonsignificant difference between the groups overall for hostility, there was a tendency for dream recall with more A₂ arousals relative to A₁ arousals to be more hostile.

The basic emotions. Seven of the basic emotions (excluding LUST), as well as total emotion, were entered together as multiple dependent variables in a Kruskal-Wallis test, that included the three A₁-A₂ difference groups as an independent variable. Overall, only total emotion was significantly affected by the number of A₁-A₂ arousals present prior to waking, $H(2) = 9.16, p < .007$. A Jonckheere's test revealed a significant trend in the data for total emotion, $J = 114.50, t(35) = -2.97, p < .001, r = -.50$. Mann-Whitney U tests were used to follow up this finding. The A₂ majority group had significantly more total emotion, $U = 123,$

⁴⁰ The subscales were all standardised, and the means and standard deviations for this sample ($n = 52$) were as follows: visual perception ($M = -.11, SD = 0.82$); hostility ($M = -0.02, SD = 0.69$); pleasantness ($M = -0.02, SD = 0.90$); and dream intensity ($M = -0.04, SD = 0.63$).

$p < .003$, $r = -.53$ than the the A_1 majority group. The group with a relatively equal number of A_1 and A_2 arousals had significantly more total emotion than the A_1 majority group as well, $U = 119.50$, $p < .01$, $r = -.49$. See Table 13 for descriptive statistics and results.

Table 13.

Dream Measures in Light NREM Sleep as a Function of the A_1 - A_2 Difference

Dream/ Sleep Measure	A_2 Majority ($n = 16$)	Equal ($n = 14$)	A_1 Majority ($n = 24$)	Significance
Global Dream Measures				
Dream Composite	0.49	0.31	-0.27	$A_2 > A_1^{***}$ $E > A_1^*$ -T^{***}
TRC	38.25	26.79	16.13	$A_2 > A_1^{**}$ -T^{**}
Subjective Sleep Depth	3.44	3.57	3.24	<u>ns</u>
MDQ				
Hostility	0.24	0.03	-0.33	$A_2 > A_1^{**}$ -T[*]
Visual Perception	-0.06	0.02	-0.27	<u>ns</u>
Pleasantness	-0.33	0.30	0.03	<u>ns</u>
Dream Intensity	-0.09	-0.01	-0.28	<u>ns</u>
Bizarreness	2.83	2.90	2.80	<u>ns</u>
Basic Emotions				
SEEKING	-0.65	-0.03	-0.06	<u>ns</u>
CARE	0.17	0.00	-0.38	<u>ns</u>
PLAY	0.08	0.42	-0.26	<u>ns</u>
FEAR	0.58	-0.30	-0.26	<u>ns</u>
GRIEF	0.44	-0.24	-0.24	<u>ns</u>
RAGE	0.26	0.18	-0.39	<u>ns</u>
DOMINANCE	0.45	0.01	-0.31	<u>ns</u>
Overall Emotion	0.16	-0.01	-0.25	$A_2 > A_1^{**}$ $E > A_1^{**}$ -T^{***}

Note. Pooled averages have used. Bolded values indicate statistically significant results at the adjusted $\alpha = .01$ level. Dream composite, The MDQ subscales, and the basic emotions have all been standardised. -T = A negative trend in the data—decreasing from the A_2 majority to the A_1 majority group; +T = A positive trend in the data—increasing from the A_2 majority to the A_1 majority group. TRC = total recall count; MDQ = multidimensional dream questionnaire; ns = nonsignificant; for scales of measurement refer to Table 6. or the Methods chapter.

* $p < .05$, ** $p < .01$, *** $p < .001$, 1-tailed.

Dreaming during CAP Relative to REM and NCAP Sleep

One of the predictions of this study was that dream recall from awakenings following unstable NREM sleep would be more dreamlike. Differences in dream recall as a function of the relative majority of either A₁ or A₂ arousals preceding the awakening, as well as stable NREM sleep, were therefore compared with REM dreaming.

Majority A₂, Light NCAP, and REM Awakenings

A series of nonparametric tests were run to determine whether there were any significant differences in global dreaming or dream quality for the A₂ majority group, light NCAP sleep, and REM sleep. Results are reported in Table 14.

Global dreaming. The broad-based, or global, dream measures chosen for this analysis were total recall count (TRC) and dream composite (DC). A series of Mann-Whitney U tests were run to determine whether any of the groups differed with regards to global dreaming. Compared with light NCAP sleep, REM mentation had significantly higher DC scores, $U = 643, p < .0001, r = -.51$, as well as a significantly higher TRC, $U = 808, p < .0001, r = -.42$. The difference in DC for the A₂ majority and NCAP conditions trended towards significance, $U = 2572.0, p = .023, r = -.22$; there was no difference for TRC between these groups, $U = 2726.0, p = .16, r = .11$. Furthermore, DC was significantly higher for REM than the A₂ majority, $U = 223.0, p = .001, r = .35$. With regards to TRC, the differences between REM and the A₂ majority group only trended towards significance, $U = 281.0, p = .016, r = .25$. Finally, Jonckheere's test revealed a significant trend in the data for both TRC, $J = 3454, t(2) = 4.51, p < .0001, r = .31$, and DC, $J = 3724, t(2) = 5.73, p < .0001, r = .37$; therefore, mentation from the A₂ majority group was more similar to REM mentation, in terms of overall dream recall and dream quality, than mentation from stable (NCAP) S2 sleep. A Kruskal-Wallis test indicated that subjective depth of sleep was not significantly associated with the preawakening conditions, $H(2) = 4.28, p = .11$.

Dream content variables. Each of the four MDQ subscales and bizarreness (everyday experience) were entered together as multiple dependent variables in a Kruskal-Wallis test, that included the three preawakening conditions (A₂ majority, NCAP and REM) as the independent variable. Overall, visual perception, $H(2) = 11.78, p < .002$, dream intensity, $H(2) = 12.62, p < .002$, and bizarreness, $H(2) = 8.42, p < .01$, differed significantly as a function of the three preawakening conditions. Jonckheere's test revealed a significant trend in the data for visual perception, $J = 1922, t(2) = -3.27, p < .001, r = -.33$, dream intensity, $J = 1863, t(2) = 2.83, p < .002, r = .29$, and bizarreness, $J = 1747, t(2) = 1.87, p < .01, r = .19$. Mann-Whitney U tests were used to follow up these findings. Visual perception, $U = 516, p <$

.001, $r = -.34$, dream intensity, $U = 471$, $p < .0001$, $r = -.38$, and bizarreness, $U = 661.0$, $p = .003$, $r = .29$, were significantly higher for REM mentation compared with stable (NCAP) light sleep. Only visual perception was significantly higher for REM recall compared with the A₂ majority group, $U = 208$, $p < .01$, $r = -.29$.

Majority A₁, Light NCAP, and REM Awakenings

Further analyses were carried out to assess the extent to which mentation from the A₁ majority condition differed from that of REM and light NCAP sleep. All analyses were nonparametric and carried out as above, with the same adjusted levels of significance. Results are reported in Table 14.

Global dreaming. REM mentation had significantly higher DC scores than the A₁ majority group, $U = 165$, $p < .0001$, $r = -.59$, as well as a significantly higher TRC, $U = 229.5$, $p < .0001$, $r = -.51$. There were no significant differences between the A₁ majority and NCAP conditions for either DC or TRC. Finally, a Kruskal-Wallis test indicated that, overall, subjective depth of sleep was associated with the three preawakening conditions, $H(2) = 7.70$, $p = .019$; a Jonckheere's test revealed a significant trend in the data for subjective depth of sleep, $J = 3144.50$, $t(2) = 2.58$, $p = .005$, $r = .23$. Follow up Mann-Whitney U tests showed that REM sleep was rated as subjectively deeper than the A₁ majority condition, $U = 468.0$, $p = .004$, $r = -.31$. Conversely, there was no difference in subjectively rated depth of sleep between stable light NREM sleep and the A₁ majority group, $U = 510.0$, $p = .04$, $r = -.20$.

Dream content variables. Each of the four MDQ subscales and bizarreness (everyday experience) were entered together as multiple dependent variables in a Kruskal-Wallis test, that included the three preawakening conditions (A₁ majority, NCAP and REM) as the independent variable (see Table 14.). Overall, visual perception, $H(2) = 13.91$, $p < .001$, dream intensity, $H(2) = 16.93$, $p < .0001$, and bizarreness, $H(2) = 8.78$, $p < .01$, differed significantly as a function of the three preawakening conditions. Furthermore, Jonckheere's test revealed significant trends in the data for visual perception, $J = 1866$, $t(2) = 3.72$, $p < .0001$, $r = .38$, and dream intensity, $J = 1919.5$, $t(2) = 4.04$, $p < .0001$, $r = .41$. Mann-Whitney U tests were used to follow up these findings. Those differences that reached significance at the $\alpha = .01$ level between REM sleep and the A₁ majority condition were visual perception, $U = 154.5$, $p < .0005$, $r = -.40$ and dream intensity, $U = 152$, $p < .001$, $r = -.40$. No significant differences were found, for any of the content variables, for the A₁ majority and NCAP conditions. An Additional Kruskal-Wallis test showed that overall emotion differed significantly as a function of the preawakening conditions, $H(2) = 11.39$, $p < .002$. Jonckheere's test further revealed a significant trend for overall emotion, $J = 1595$, $t(2) =$

3.31, $p < .0001$, $r = .35$. Follow up Mann-Whitney U tests showed that there was significantly less overall emotion for A₁ majority mentation compared to REM, $U = 128.5$, $p < .0005$, $r = -.41$, while the difference in overall emotion for the A₁ majority and NCAP conditions did not reach significance.

In summary, awakenings following a majority A₂ arousals in light NREM sleep yielded mentation that was more similar to REM mentation than either stable (NCAP) light NREM sleep, or unstable light NREM sleep characterised by a majority A₁ arousals.

Table 14.

Comparison of the A₁ and A₂ Majority Groups with Light NCAP and REM Sleep

Dream/ Sleep Measure	NCAP (n = 53)	REM (n = 54)	Significance		
			A ₂	A ₁	R vs. NCAP
Global Dream Measures					
Dream Composite	-0.08	0.79	A₂ > NC** R > A₂*** +T****	R > A₁**** +T****	R > NC****
TRC	25.08	65.85	R > A₂* +T****	R > A₁**** +T****	R > NC****
Subjective Sleep Depth	3.48	3.72	<u>ns</u>	R > A₁** +T**	<u>ns</u>
MDQ					
Hostility	-0.07	0.15	<u>ns</u>	A ₁ > R* +T*	<u>ns</u>
Visual Perception	-0.09	0.46	R > A₂** +T***	R > A₁*** +T****	R > NC***
Pleasantness	0.07	0.06	<u>ns</u>	<u>ns</u>	<u>ns</u>
Dream Intensity	-0.20	0.38	+T**	R > A₁*** +T****	R > NC****
Bizarreness	2.29	2.96	A ₂ > NC* +T**	<u>ns</u>	R > NC**
Overall Emotion	-0.06	0.13	A ₂ > NC*	R > A₁*** +T****	<u>ns</u>

Note. Pooled averages have used. Bolded values indicate statistically significant results at the adjusted $\alpha = .01$ level. Dream composite, the MDQ subscales, and overall emotion were standardised. -T = A negative trend in the data—decreasing from the CAP condition to REM; +T = A positive trend in the data—increasing from the CAP condition to REM. TRC = total recall count; MDQ = multidimensional dream questionnaire; ns = nonsignificant; for scales of measurement refer to Table 6. or the Methods chapter.

* $p < .05$, ** $p < .01$, *** $p < .001$, 1-tailed.

CAP parameters of interest: Deep NREM sleep

Pearson correlation coefficients for the CAP parameters of interest and the global dream measure, DC, for *unstable deep NREM sleep*, are presented in Table 15. Unlike during light NREM sleep, none of the CAP parameters for deep NREM sleep were significantly related to dreaming. Consequently, a RC multilevel regression model was considered inappropriate for the analysis of the data, and was not run. Furthermore, the number of A₁ arousals relative to A₂ arousals could not be calculated, as A₂ arousals were only a small minority that did not have any significant correlation with dreaming in this condition (Table 15). In conclusion, then, there were no apparent relationships between dreaming and the visually defined microstructural events for awakenings from unstable deep NREM sleep.

Table 15.
Correlations Deep NREM, CAP Parameters and Dreaming^a

	TSA	SL	TA ₁	A ₁ (3)	A ₂ (3)	DC
Time Since A	1	-.29	-.32	-.35**	.01	-.19
Sequence Length		1	.94**	.57**	.04	.04
Total A ₁			1	.67**	-.02	.06
A ₁ in last 3 min				1	-.08	-.05
A ₂ in last 3 min					1	.10
Dream Composite						1

Note. Bolded values indicate statistical significance. Time Since A = The amount of time in seconds since the last arousal; Total A₁ = The total number of A₁ arousals in the last CAP sequence preceding waking; TSA = time since A; SL = sequence length (seconds); TA₁ = total A₁; A₁(3) = A₁ in the last 3 minutes prior to waking; A₂(3) = A₂ in the last 3 minutes prior to waking; DC = dream composite.

^a $n = 53$

* $p < .05$, ** $p < .01$, 2-tailed.

Stable NREM sleep, the Sleep Microstructure, and Dreaming

By definition, a NCAP awakening was an awakening that was preceded by at least 60 seconds of sleep that did not contain a CAP sequence; i.e., at least 60 seconds of *stable* NREM sleep. However, certain microstructural events were still analysed in relation to dream recall from these conditions, based on the possibility that CAP parameters may still influence dreaming during stable NREM sleep.

General Intercorrelations

Table 16 (light NREM sleep)⁴¹ and Table 17 (deep NREM sleep) show the Pearson correlation coefficients between all of the CAP variables of interest and global dreaming (the DC measure). Based on the prediction that CAP parameters may still influence dreaming during stable NREM sleep, relationships between global dreaming and a) time since the last CAP sequence, b) time since the last isolated arousal, c) the number of isolated arousals preceding the awakening,⁴² d) the arousal subtypes (from a CAP sequence) in the last three minutes, and d) the isolated arousal subtypes in the last three minutes, were of interest. In addition, the hour of awakening was included to determine whether time of night had any influence on dream recall for the NCAP conditions. Surprisingly, none of the CAP parameters of interest were significantly correlated with DC for either light or deep NREM sleep. In light NREM sleep the A₁-A₂ difference for awakenings made within three minutes of a CAP sequence ($n = 16$), showed no relationship with dream recall, $r = .09$, $p = .73$. Noteworthy, though, is that trends in line with the analysis of unstable NREM sleep emerged when this group was split into groups—the A₂ group majority ($M = -.10$, $n = 5$), had higher scores than both the equal A₁ and A₂ ($M = -.27$, $n = 6$) and majority A₁ group ($M = -.36$, $n = 5$). Unfortunately, these groups were too small for inferential statistics to be run.

⁴¹ Two dream reports have been excluded from this analysis because the awakenings were made during a stage shift (i.e., < 5 minutes of a consolidated sleep stage).

⁴² To form part of a CAP sequence arousals have to occur within 60 seconds of one another; arousals that did not take place within this time frame were considered to be isolated events that do not have the same properties as arousals that oscillate within 60 seconds of each other (Terzano & Parrino, 2000). For instance, a CAP cycle within a sequence has an A and a B phase (see Appendix A), however, isolated arousals do not have a B phase.

Table 16.

CAP Parameters in Light NCAP Condition: General Intercorrelations^a

	TSS	TSI	TI	HA	A ₁	A ₂	iA ₁	iA ₂	DC
Time Since Seq.	1	.33**	.63**	.50**	-.04	-.36	-.23	-.02	.10
Time Since Iso. A		1	-.18	.27	.13	.11	-.50**	-.35**	.08
Total Iso. A			1	.22	-.10	-.26	.47**	.20	.14
Hour of Awakening				1	-.02	.11	-.38**	-.02	.09
No. A ₁ 3min ^b					1	-.27	.	.	-.07
No. A ₂ 3min ^b						1	.	.	-.19
No. iso A ₁ 3min ^c							1	-.11	-.21
No. iso A ₂ 3min ^c								1	.08
Dream Composite									1

Note. Bolded values indicate statistical significance. TSS = time since sequence; TSI = time since isolated arousal; TI = total isolated arousals; HA = hour of awakening; A₁ = total A₁ in the 3 minutes before waking; A₂ = total A₂ in the 3 minutes before waking; iA₁ = total isolated A₁ prior to waking; iA₂ = total isolated A₂ prior to waking; DC = dream composite.

^a $n = 65$

^b $n = 16$, awakenings that had a CAP sequence in the preceding three minutes

^c $n = 49$, awakenings that did not have a CAP sequence in the preceding three minutes

* $p < .05$, ** $p < .01$, 2-tailed.

In deep NREM sleep there were a number of small-to-medium correlations that may have reached significance had the sample been bigger (Table 17). The time of the awakening also did not seem to have any impact on dream recall, which is contrary to findings that show enhanced dream recall as a function of time of night (Fosse et al., 2004). Based on these findings, no further inferential analyses were carried out on the NCAP data.

Table 17.
CAP Parameters in Deep NCAP Condition: General Intercorrelations^a

	TSS	TSI	TI	HA	A ₁	iA ₁	DC
Time Since Seq.	1	-.08	.91**	.32	-.64**	-.73	-.28
Time Since Iso.		1	-.40	.36	.37	-.53	-.06
Total Iso. A			1	.16	-.66**	-.47	-.28
Hour of Awakening				1	-.06	-.66	-.02
No. A ₁ 3min					1	.	-.35
No. iso A ₁ 3min						1	.27
Dream Composite							1

Note. Bolded values indicate statistical significance. TSS = time since sequence; TSI = time since isolated arousal; TI = total isolated arousals; HA = hour of awakening; A₁ = total A₁ in the 3 minutes before waking; iA₁ = total isolated A₁ prior to waking; DC = dream composite.

^a $n = 19$

* $p < .05$, ** $p < .01$, 2-tailed.

GENERAL DISCUSSION

The aim of the present study was to explore sleep microstructure in relation to dreaming, to determine whether there is any significant relationship between dreaming and phasic arousal activity during NREM sleep. With this aim in mind, the first hypothesis predicted that *awakenings made during unstable NREM sleep would yield enhanced dream recall compared with awakenings from stable NREM sleep*. The results have only partially supported this hypothesis. Unstable deep NREM sleep was found to yield more dream recall than stable deep NREM sleep. In contrast, no significant differences were found between these two conditions for light NREM sleep. However, when unstable light NREM sleep was further delineated according to its composition by the *arousal subtypes* a different picture emerged; light NREM sleep predominantly characterised by the A₂ arousal subtype yielded more dream recall than light NREM sleep comprised predominantly of the A₁ subtype. These results support both the second and third hypotheses in this study; namely, that *the desynchronised arousal subtypes (A₂ and A₃) are related to enhanced dream recall, while the slower synchronised subtype (A₁) is related to less recall*. As a result, the first hypothesis does appear to be supported when only certain portions of unstable sleep are considered. To date the arousal-retrieval model has been primarily drawn upon to explain the occurrence of enhanced dream recall in relation to intrasleep wakefulness and arousal activity during sleep. This theory will be critically appraised in relation to the present results, and alternate explanations for the present results will be offered.

Furthermore, certain unexpected findings also require further explication. In particular, it was unexpected that the A₂, but not the A₃, arousal subtype was found to relate significantly to dream recall. Based on previous findings it was predicted that desynchronised phasic arousal activity would be related to enhanced dream recall in a linear fashion; however, this was not found to be the case. Possible explanations for this discrepancy are discussed. Furthermore, in certain circumstances the A₁ subtype appeared to be significantly related to enhanced dream recall (i.e., deep NREM sleep), but not in other cases (during light NREM sleep). These findings are discussed in relation to the relevant literature.

Synchronisation-type Microarousals and Dreaming

Synchronisation-type microarousals, although not widely accepted as arousal related activity, have been recognised for the past 30 years as part of the spectrum of phasic arousal phenomena (Halász, Kundra, Rajna, Pal, & Vargha, 1979). In fact, the term microarousal was first used to describe *either* desynchronised or synchronised phasic events associated with autonomic and/or motor arousal, though not with behavioural awakening (Halász et al., 1979). Today only desynchronised microarousal phenomena, accompanied by autonomic and motor activations, are widely recognised as phasic arousal activity in sleep (Atlas Task Force, 1992). It has been shown, however, that almost 90% of arousals from NREM sleep—even those that meet the criteria for ASDA defined microarousals—are preceded by *K-complexes* (KCs) or delta bursts (Halász, 1993). This initial burst of slow wave activity represents a readiness of the cerebral cortex, while sleep is simultaneously preserved; this slow activity may persist throughout the event (A_1), or open the way to more rapid activities with stronger cerebral, motor and autonomic activating effects (A_2 and A_3 ; Terzano et al., 2002). In this way, CAP provides a graded picture of the activating features of phasic arousal phenomena by including the range of slow and rapid EEG activities in their classification (Terzano & Parrino, 2000).

Homeostatic Sleep Processes and Dreaming

In order to try and understand the ways in which synchronised arousal activity may impact the dream process, or our memory thereof, the functions of the KC will be briefly discussed. The KC is a slow biphasic wave of high voltage; it is predominantly observed in S2 sleep, but is it also present in SWS. KCs can be evoked by external sensory stimulation and also appear spontaneously (Amzica & Steriade, 2002). The KC often forms part of a larger unit of reactive sleep events, and is frequently preceded by bursts of either alpha (8 – 12 Hz), delta (0.5 – 4 Hz), or sigma (12 – 15 Hz) activity (Halász, 2005). These units of reactive sleep events are recognised by the CAP system, and have been classified into the different arousal subtypes. The KC, despite being one of the most studied graphoelements of sleep, remains one of the most elusive in terms of its proposed functions. On the one hand, this phasic phenomenon is considered as an element of transient arousal during sleep (Ehrhart, Ehrhart, Muzet, Schreiber, & Naitoh, 1981), while on the other hand it has been argued that it is a sleep protective mechanism (Wauquier, Aloe, & Declerck, 1995). More recently, the KC has been shown to be a complex *multifunctional* phenomenon, the exact features of which rely on the nature of the eliciting stimulus and the background sleep activity within which it occurs (Halász, 2005; Jahnke et al., 2012).

In this way, the KC is comprised of at least two independent event-related potential (ERP)⁴³ components (Perrin, Bastuji, Mauguière, & Garcia-Larrea, 2000). Halász (2005) has hypothesised that the first component (N200-P300) is related to arousal processes and the processing of the external environment, while the later component (N550-P800) is a sleep protecting mechanism, reflecting antiarousal processes. This hypothesis is supported by a number of findings. For instance, in relation to the later component, functional magnetic resonance imaging (*fMRI*) has shown predominantly negative BOLD signal changes in both sensory and nonsensory cortices in response to an auditory stimulus during NREM sleep (Czisch et al., 2004; Czisch, Wetter, Kaufmann, Pollmächer, & Holsboer, 2002). Furthermore, these BOLD signal decreases were positively associated with the number of KCs and delta waves that occurred in response to the auditory stimulus (Czisch et al., 2002, 2004). Thus the authors concluded that the “BOLD signal is caused by a general response to sensory stimulation reflecting a deepening of sleep that could counteract potential arousal effects” (Czisch et al., 2002, p. 256). This is precisely the mechanism described by Halász and Bódizs (2013) for the antiarousal effect of the A₁ subtype.

In this study dream recall was significantly reduced following periods of light NREM sleep comprised predominantly of phasic A₁ activity in the minutes before waking. As A₁ arousals are mainly comprised of bursts of slow delta activity and KCs, this finding is consistent with studies of the electrophysiological correlates of dreaming that show an inverse relationship between frontal delta activity (0.5 – 4 Hz) and dream recall (Chellappa et al., 2011; Esposito et al., 2004; Wollman et al., 1987). Moreover, the sleep negativities related to the second, sleep promoting, component of the KC (N350 and N550) have been found to be related to sleep inertia—“the ubiquitous phenomenon of cognitive performance impairment, grogginess and tendency to return to sleep immediately after awakening” (Bastuji, Perrin, & Garcia-Larrea, 2003, p. 198). According to the arousal-retrieval model of dreaming, then, dream recall may be reduced in relation to A₁ arousals because this activity fundamentally interferes with the ability to recall any prior dream experiences. This is consistent with Bastuji et al.’s (2003) conclusion that KCs are arousal driven on the one hand,

⁴³ Event-related brain potentials are defined as “voltage deflections recorded from the scalp arising from summated postsynaptic potentials of large neuronal populations” (Atienza et al., 2001, p. 2032). The amplitude and latencies of the successive ERP wave forms are used to determine the time course of neuronal information processing, while the topographic distribution of the voltages is used to estimate the neuroanatomical loci of these processes (Atienza et al., 2001).

but memory “erasers” on the other, preventing the encoding and retrieval of memory during sleep.

How is it, then, that unstable S3 sleep yielded the most dream recall, while the presence of A₁ arousals in light NREM sleep led to less dream recall? While at first glance these findings appear to be contradictory, it should be pointed out that mentation from both light and deep NREM sleep characterised predominantly by A₁ arousals was comparable. In particular, both were similar with regards to total recall count, global dreaming, visual perception, and dream intensity. Additionally, both light and deep NREM sleep comprised predominantly of the A₁ CAP activity appeared to, on average, yield reports of fragmented imagery and thoughtlike activity.

In this regard, Czisch et al. (2002) note in their imaging study that the “...more pronounced deactivation during light NREM sleep compared to SWS would underline a sleep-enhancing mechanism as SWS cannot be deepened to a similar extent as light NREM sleep” (p. 257). Accordingly, A₁ arousals in light NREM sleep represent prominent deactivations occurring against a relatively active tonic background, whereas during deep NREM sleep they provide the only source of arousal activity against a background of synchronisation. The sleep promoting mechanisms associated with the A₁ subtype thus appear to reduce dream recall during light NREM sleep, while, despite these sleep promoting mechanisms, the arousal component associated with the A₁ subtype appears to enhance recall in deep NREM sleep; this is an important point that will be elaborated upon shortly.

Additionally, unstable deep NREM sleep yielded the highest self-rated remembrance values for NREM sleep, indicating *less* sleep inertia from unstable than stable deep NREM sleep. If not due to differences in the capacity to remember a dream, though, why is mentation from unstable deep NREM sleep still generally thoughtlike and fragmented? One explanation is that slow wave activity is *fundamentally incompatible* with the dream process, as dreaming might require *consciousness* during sleep—not only to be remembered, but to be generated (Nir & Tononi, 2010). Consciousness, as defined by Tononi (2004), refers to the subjective experience that arises from the integration of information, the integrity of which is reliant on effectively connected specialised regions in the thalamocortical system. Neuronal groups are effectively connected if the firing of one set is able to causally affect the firing of another set (Tononi and Sporns, 2003).⁴⁴ Importantly, consciousness should not be confused

⁴⁴ The term *consciousness* can refer to a number of different states, and has been given numerous definitions by various authors. Theories of affective, subcortical, consciousness have recently been proposed as well

with *connectivity* or *responsiveness*; connectivity refers to the connection of consciousness to the external world and the experiencing of external stimuli, whereas responsiveness is the ability of an organism to spontaneously respond to the environment (Sanders, Tononi, Laureys, & Sleight, 2012). During waking we are conscious and both connected and responsive to our environments, though these three concepts can be dissociated. For instance, in rare cases anaesthesia has left patients both conscious and connected to their external environments, while simultaneously spontaneously unresponsive (Moerman, Bonke, & Oosting, 1993). Likewise, certain portions of sleep are consciousness, and to lack both connectivity and responsiveness (Sanders et al., 2012).

However, not all sleep is conscious. Slow wave activity in sleep is positivity correlated with a decrease in the effective connectivity between cortical areas, and subsequently reduced brain reactivity, which has been related to both thalamic and cortical gating mechanisms (Esser, Hill, & Tononi, 2009). In particular, *thalamic gating* is able to cause a functional dissociation between the cortex and the thalamus during sleep (Steriade, 2000).⁴⁵ As waking shifts into sleep, a *1 Hz slow oscillation* generated in the neocortex gradually entrains large neuronal populations and shapes neuronal activity during NREM sleep (Steriade et al., 1993); specifically, the 1 Hz oscillation causes thalamocortical activity to alternate between a depolarising phase associated with neuronal firing (ON state), and a hyperpolarising phase associated with neuronal silence (OFF state; Massimini, Huber, Ferrarelli, Hill, & Tononi, 2004). The 1 Hz oscillation is reflected in EEG recordings as high amplitude, low frequency activity (i.e., delta waves), as it represents widespread synchronous changes in large neuronal populations (Steriade et al., 1993). When this bistable state—depolarisation-hyperpolarisation; ON-OFF—is consolidated, it results in drastically reduced transmission of incoming sensory information from the thalamus to the cortex, as the

(Panksepp & Solms, 2012). In the current context, as defined above, consciousness refers to a subjective state arising from effectively connected cortical and subcortical regions (Tononi, 2004). A discussion of the *type* of consciousness (or awareness) that belongs to dreaming is beyond the scope of this chapter; for now, it is acknowledged that the dream is a subjective experience that, according to lesion and neuroimaging studies (Solms, 1997; Braun et al., 1998), requires at least certain limbic regions and posterior association cortex to be effectively connected to transpire. Throughout this discussion, the term consciousness will be used with these considerations in mind.

⁴⁵ Essentially this means that “...effective communication from subcortical structures to the cortex via the thalamocortical system is diminished or absent and therefore no sensory information reaches the cortex” (Mena-Segovia & Bolam, 2011, p. 89). This state is induced by decreased firing of the ascending projection neurons in the brainstem (Saper et al., 2005).

frequently occurring OFF states cause signal transmission to be disrupted (Llinás & Steriade, 2006). Compared with REM and waking, then, neuronal firing is essentially different during NREM sleep, where neurons show a burst-pause pattern of firing instead of a tonic one (Vyazovskiy et al., 2009).

Congruent with this interpretation, it has also been shown that activity evoked by transcranial magnetic stimulation (TMS), while able to propagate through functionally connected networks in the cortex during waking, remains localised during SWS (Massimini et al., 2005). As TMS bypasses the thalamus, it is argued that the thalamic gate cannot be responsible for this reduction in intracortical transmission, and the likely mechanism proposed is a *cortical gate* governed by an increase of GABA released per spike burst during SWS compared to other stages of sleep (Esser et al., 2009). Regardless of the precise mechanisms involved, reduced effective connectivity during SWS is argued to result in a reduced capacity for consciousness (Esser et al., 2009).

Along these lines, Nir and Tononi (2010) have suggested that dreaming may require long ON states during sleep, as is the case in REM sleep and NREM later in the night (Vyazovskiy et al., 2009). Conversely, during early sleep, when homeostatic sleep pressure⁴⁶ is high and slow waves predominate, short ON periods alternate with longer OFF periods; later in the sleep period, when slow wave activity is diminished, ON periods are longer and are only occasionally interrupted by shorter OFF periods (Vyazovskiy et al., 2009). Subsequently, and in line with Nir & Tononi's (2010) predictions, we would expect less effective connectivity and less dreaming during states of sleep predominated by homeostatic pressure (and slow wave activity). Consistent with this interpretation, in the present study awakenings from S4 sleep tended to yield exceptionally low dream recall regardless of the presence of phasic CAP activity. Indeed, S4 sleep is the most synchronised stage of sleep (Achermann, Dijk, Brunner, & Borbély, 1993); it is comprised of consolidated thalamocortical bursting and cortical gating (Esser et al., 2009), indicating a significantly reduced capacity to support effective connectivity, and therein consciousness (Massimini et al., 2005). This hypothesis is further supported by a recent set of studies that found that, using TMS, visual dream imagery could be enhanced by simultaneously inhibiting certain frontal regions and stimulating the right posterior parietal cortex during S2 sleep, but not during

⁴⁶ Longer periods of wakefulness lead to enhanced slow wave activity, while slow waves progressively decrease with time spent asleep. This pressure for sleep is referred to as *homeostatic sleep pressure*, of which delta and slow wave activity (0.25 – 4 Hz) is considered a marker (Borbély & Achermann, 1999; Borbély, Achermann, Trachsel, & Tobler, 1989).

SWS (Jakobson, Fitzgerald, & Conduit, 2012a, 2012b). The authors concluded that this discrepancy was likely due to differences in cortical connectivity between the two NREM states.

Homeostatic Features of Light NREM Sleep

While circadian and ultradian influences on dreaming have been acknowledged in the literature (Nielsen, 2004), and many models of dreaming have been based on the gradual increase in cortical activation throughout the night (Antrobus, 1986; Nielsen, 2004; Wamsley, Hirota, Tucker, Smith, & Antrobus, 2007; Zimmerman, 1970), the influence of homeostatic pressure, especially as it relates to phasic arousal activity during sleep, has not previously been recognised. In this study, sleep comprised predominantly of A₁ antiarousal processes, whether in the first or second half of the night, was associated with reduced dream recall and increased cognitive activity. This is the first time that these changes have been associated with A₁ activity in light NREM sleep. Indeed, regardless of the slope of the sleep cycle, relatively increased A₁ arousals led to decreased dream recall (and more fragmented and thoughtlike recall). As a result, future studies need to acknowledge the vastly different phasic activity in light NREM sleep, especially in the first half of the night, and the impact it may have on dream recall.

Phasic Arousal Activity, Reward Processing, and Dreaming

How are we to reconcile this view with the fact that, not only in this study but in several others, dreaming has been reported from sleep predominantly characterised by delta and slow wave activity (Cavallero et al., 1992; Pivik, 1970; Tracey & Tracey, 1974), and in some cases to the same extent as light NREM sleep (Cavallero et al., 1992)? A possible explanation may be that *dreaming is critically related to phasic arousal processes during SWS*. As indicated by the results in this study, most mentation during the deeper stages of sleep (especially S3) occurred during unstable periods characterised by CAP activity. As the A₁ subtype is proposed to have both activating and sleep promoting aspects (Halász, 2005; Halász & Bódizs, 2013; Jahnke et al., 2012; Terzano et al., 2002), it may be that the arousal-driven portion of this activity facilitates dreaming via several mechanisms.

The Dopaminergic Hypothesis of Dreaming

Central to the impending discussion is the hypothesis that dreaming is primarily generated by those neurophysiological systems involved in motivational, exploratory, and reward related behaviours (Solms, 1997, 2000), of which the mesolimbic dopamine (ML-DA) system is the primary driver (Panksepp, 1998). The ML-DA system is defined as the “system [that] is formed by dopamine neurons located in the ventral tegmental area...which project to

the nucleus accumbens, prefrontal cortex, septum, amygdala, and hippocampus” (Dahan et al., 2006, p. 1232). This system has been termed the SEEKING system by Panksepp (1998) and it is thought to “...drive and energize many mental complexities that humans experience as persistent feelings of interest, curiosity, sensation seeking” (p. 145). It is also involved in reward processing, which refers to “...an instinctual affective and exploratory drive to seek biologically-important stimuli in the external or internal (‘intrapsychic’) environment” (Perogamvros & Schwartz, 2012, p. 1936).

The ML-DA system has been critically implicated in the generation of dreaming due to several converging lines of evidence.⁴⁷ First, human lesion studies have consistently found dream loss to be related to lesions in the parieto-temporo-occipital (PTO) junction and the bilateral white matter of the ventromesial quadrant of the frontal lobes (Solms, 1997). Specifically, with regard to frontal lesions, injury to the head of the caudate nucleus and the ventral striatum, the most ventromesial regions of the frontal lobe, were shown to be particularly involved in dream loss (Yu, 2001a). This region is also central to the large ML-DA fibre pathway transmitting dopamine from the VTA to the higher parts of the PFC (Panksepp, 1998). In addition to dream loss, a dramatic reduction in motivated behaviour was found for patients with bilateral vmPFC lesions, further implicating the ML-DA system in dream loss (Jus et al., 1973; Németh, Hegedüs, & Molnár, 1988; Paus, 2001; Solms, 1997).

Moreover, several neuroimaging studies have confirmed that these regions are highly activated during REM sleep, when dreaming is most prevalent (Braun et al., 1997, 1998; Dang-Vu et al., 2005; Maquet et al., 1996; Nofzinger et al., 1997). It has also been reported that there is an increase in dopamine release in humans during REM sleep, within the ML-DA system (Gottesmann, 2004). More recent studies based on the internal measurement of dopamine (through microdialysis and single cell recordings) during the sleep-wake cycle in rats, have also found a substantial increase in dopamine cell activity and terminal release during REM sleep (Dahan et al., 2007; Lena et al., 2005). Similarly, when the dopaminergic pathway that runs through the ML-DA system is transacted, in the surgical procedure known as *modified prefrontal leucotomy*, cessation of dreaming occurs (Jus et al., 1973). Finally, L-Dopa—a drug that specifically stimulates dopamine in this region—has been found to intensify the bizarreness, emotionality and vivacity of dreaming, while the REM cycle remains unaltered (Hartmann, Russ, Oldfield, Falke, & Skoff, 1980). Similarly, a cessation of

⁴⁷ An exhaustive review of this literature is beyond the scope of this discussion. The interested reader is referred to either Perogamvros and Schwartz (2012), or Solms (2000).

dream recall has been associated with Parkinson's disease, which is usually the result of an insidious depletion of dopamine in various forebrain regions (Sandyk, 1997). In all, then, there is sufficient evidence to crucially implicate the SEEKING system in dream generation.

The basic emotions in dreaming. This is the first study to examine the basic emotions in dreaming, as well as to ask specifically about feelings of anticipation, motivation, and seeking in dreams (see Appendix C). Congruent with the above evidence is that of all the self-rated basic emotions in dream content, SEEKING was the most commonly rated *throughout all stages of sleep*. In addition to SEEKING, PLAY and CARE were the next most rated basic emotions in dreaming. Of the more aversive emotions, FEAR was rated the most. However, FEAR may still be related to ML-DA system activity, as the act of seeking out a place to hide or trying to get away from something also involves motivated behaviours (Panksepp, 1998). Furthermore, the nucleus accumbens and the VTA have been found to be activated in response to both reward and punisher anticipations (Carter, MacInnes, Huettel, & Adcock, 2009; Perogamvros & Schwartz, 2012).

Overall, positive emotions predominated during dreaming. While objective rating techniques have previously emphasised that aversive emotions predominate in dreaming (Hall & van der Castle, 1966), this appears to be linked to a bias towards verbally reporting negative dream content in a way that overshadows positive content (Schredl & Doll, 1998). This study is therefore consistent with other findings of predominantly positive self-rated dream emotions (Schredl & Doll, 1998). These findings are congruent with theories placing the ML-DA at the centre of dreaming (Perogamvros & Schwartz, 2012; Rotenberg, 1993; Solms, 2000; Solms & Turnbull, 2002). Nonetheless, the validity of these findings awaits replication.

The reward activation model. In addition to the involvement of reward processing in dreaming, the *reward activation model* (RAM) has recently proposed that reward processing also contributes to the consolidation of memories with a high motivational/ emotional relevance during sleep, as well as aids in the modulation of REM sleep through projections to REM generating structures (Perogamvros & Schwartz, 2012). Due to the strong mutual interconnections between the hippocampus and the VTA, Perogamvros and Schwartz (2012) propose that activation of the hippocampus during sleep may stimulate the VTA and lead to reward activation during sleep; in turn, VTA activity can lead to the reactivation of certain memories in the hippocampus. It is predicted that during SWS in particular, the reactivation of the ventral striatum and the hippocampal complex allows for the consolidation of "memory-reward associations" (Lansink, Goltstein, Lankelma, McNaughton, & Pennartz,

2009). Therefore—and as originally proposed by Freud (1900/2006), and more recently by Solms (1997, 2000)—“in dreams abstract thoughts and memories are converted into concrete perceptions” (Solms, 2001, p. 7). Put another way, these memories and abstract thoughts may provide the *nucleating point* or impetus for dreaming during NREM sleep.⁴⁸

Sleep Instability as a Gateway to Dreaming During SWS

A₁ arousals have recently been hypothesised to be associated with improvements in homeostasis and memory consolidation. As hippocampus-dependent declarative memories can be enhanced through the artificial boosting of slow wave activity with TMS (Massimini, Tononi, & Huber, 2009), and local increases in slow wave power density are related to improvements in task performance (Huber, Ghilardi, Massimini, & Tononi, 2004), it has been proposed that CAP may “use” external stimuli during sleep to produce increased slow wave activity when homeostatic pressure is high (Halász & Bódizs, 2013, see Introduction). In particular, the A₁ subtypes deliver bursts of delta activity in response to stimulation during sleep, providing additional homeostasis (i.e., slow waves) to frontal regions (Halász & Bódizs, 2013). In support of this hypothesis, it has been shown that A₁ arousal activity was increased the night after participants completed an implicit learning paradigm (Ferri et al., 2008). Additionally, an improvement in certain neuropsychological tasks has been correlated positively with A₁ activity night before (Aricò et al., 2010). Thus, it may be that memory consolidation processes related to A₁ activity during sleep may occasionally stimulate the VTA and the ML-DA system, leading to increased dream recall during these stages.

Furthermore, the ON phase of the 1 Hz slow oscillation—specifically in relation to high amplitude slow waves (> 140 μ v)—is dynamically equivalent to “fragments” of wakefulness during sleep (Destexhe, Hughes, Rudolph, & Crunelli, 2007). It has been proposed that these micro-wake-like bursts of neuronal firing “...might provide brief epochs of network dynamics that aid the transfer of memory traces between short-term storage sites in the hippocampus and long-term memory space in the neocortex” (Destexhe et al., 2007, p. 338). In other words, these periods might reinstate the waking resting-state network for the purposes of the replay, and possible consolidation, of previous experiences (Dang-Vu et al.,

⁴⁸ This point is further supported by the fact that damage to the highest levels of the perceptual systems (PTO junction) results in complete dream loss, whereas damage to the primary visual cortex does not (Solms, 1997). Unlike waking perception then, that is fed forwards from the lowest levels of the perceptual system, dreaming is thought to reverse the normal sequence of perceptual events and to involve the backwards projection of memory systems—activated by an arousing stimulus in the ML-DA system—in the form of concrete images onto the perceptual systems (Braun et al., 1998; Solms, 2000, 2001).

2008; Destexhe et al., 2007). As the excitatory phase of the 1 Hz slow oscillation (the ON phase) is effective in grouping KCs and delta waves, it is argued that “slow waves rarely appear as isolated features, but in most cases converge into collectives resulting in the phase A₁ subtypes” (Parrino et al., 2012, p. 32). Consequently, the A phase of the A₁ subtype always coincides with the ON phase of the 1 Hz oscillation. In this way, active microstates are transiently activated in conjunction with the A₁ arousals during sleep (Halász & Bódizs, 2013). In line with Perogamvros and Schwartz’s (2012) hypothesis, the replaying of experiences during the wake-like ON phase of the 1 Hz oscillation—and subsequently during phasic A₁ activity—may result in activation of the VTA if a particularly motivational experience is replayed. Also consistent with this proposal is that SEEKING was still rated as the most common emotion during deep NREM sleep, even for the most fragmented and thoughtlike recall; is the persistence of this emotion further evidence of the motivational salience of certain memories during consolidation, even in those cases where a dream proper does not evolve?

A potential caveat to this line of argumentation is that the ON phase of the 1 Hz oscillation continues during stable SWS. Why then would dream recall be more likely during unstable sleep? There are two potential answers to this question. First, despite that memory consolidation continues throughout deep NREM sleep, dreaming may be more likely to occur before the UP states of the 1 Hz oscillation reach their shortest turnover and duration—as is the case in consolidated, stable deep sleep (Parrino et al., 2012; Vyazovskiy et al., 2009). As mentioned, Nir and Tononi (2010) maintain that dreaming requires longer UP states to occur. Therefore, the more *stable* deep NREM sleep becomes, the more likely that consciousness (and hence dreaming) cannot be maintained. Unstable deep NREM sleep may therefore allow for the opportunity for effective connectivity (i.e., consciousness) to be reinstated, even if only briefly. That dreaming in the present study was found significantly less during periods of stable SWS provides support for this interpretation. Similarly, dream recall may have been reduced during S4 sleep, regardless of CAP activity, because this stage is more consolidated than S3. In this way, reward processing related to memory consolidation probably continues throughout SWS, but the chances of these memory consolidation processes leading to a dream are drastically reduced.

Local Arousal Activity during Sleep

The ways in which consciousness may be reinstated in SWS require further exposition. In certain instances stimulation of the VTA, and the subsequent transient restoration of the dream process, may *not* successfully be disrupted by the cortical gate, (or

the OFF state of the 1 Hz oscillation), resulting in localised arousal and dreaming. There are numerous lines of evidence that indirectly support this hypothesis. First, recent intracortical recordings have shown that aspects of NREM sleep and wakefulness can occur simultaneously in different parts of the cortex in both mammals and humans, despite global behavioural shutdown (Rattenborg, Lima, & Lesku, 2012). For instance, Nobili et al. (2011) found frequent wake-like activations in the motor cortex of epileptic patients during SWS lasting from 5 to more than 60 seconds, accompanied by the simultaneous deepening of sleep (i.e., increased slow wave activity) in the dorsolateral prefrontal cortex. A similar finding of local wake-like aspects during NREM sleep in the rat suggests that these findings may be a normal feature of mammalian sleep (Vyazovskiy et al., 2011). Therefore, it may be the case that mentation during SWS occurs due to the localised arousal (and effective connectivity) of certain neurophysiological systems that are needed to sustain dreaming.

Additional support for this notion comes from the study of certain parasomnias. It has been proposed that the ML-DA system may be responsible for episodes of nocturnal eating, as dysfunctions of this system, and especially the accentuation of dopamine production, have been related to eating disorders (Johnson & Kenny, 2010; Perogamvros et al., 2012; Shinohara et al., 2004; Wang, Volkow, & Fowler, 2002). Also, reward sensitivity, a dopamine related trait, has been shown to be associated with overeating (Davis Strachan, & Berkson, 2004). Likewise, Perogamvros et al. (2012) describe two cases of nocturnal eating with a history of childhood somnambulism and increased novelty seeking and reward sensitivity traits in wakefulness; importantly, these psychometrically measured traits have been shown to be related to increased activity in ML-DA system (Beaver et al., 2006; Hutchison, Wood, & Swift, 1999; Krebs, Schott, & Düzel, 2009; Netter, Henning, & Roed, 1996; Schweinhardt, Seminowicz, Jaeger, Duncan, & Bushnell, 2009). Together with evidence that the ML-DA system remains active during sleep, Perogamvros et al. (2012) propose that "...the activation of the reward system during sleep may offer a permissive condition for episodes of nocturnal overeating to occur, especially in patients with elevated reward sensitivity and novelty seeking" (p. 5). Similarly, they suggest that behaviours in other parasomnias, such as locomotion in sleepwalking, aggression in REM sleep behaviour disorder, and sexual behaviours in confused awakenings, may be related to exploratory and instinctual behaviours instigated by the ML-DA during sleep.

Importantly, for the purposes of this discussion, Perogamvros et al. (2012) further point out that there appears to be a "facilitatory influence" of arousals on episodes of nocturnal eating, as polysomnography revealed an increased arousal index for both patients

mentioned above. Noteworthy, in this regard, is the extensive literature on abnormalities in CAP and sleep instability in relation to parasomnias. For instance, not only has increased arousal instability been noted in insomnia (Terzano & Parrino, 1992), depression (Farina et al., 2003), eating disorders (Dalla Marca et al., 2004), sleep apnea syndrome (Terzano, Parrino, Boselli, Spaggiari, & Di Giovanni, 1996), periodic limb movements (Parrino et al., 1996), nocturnal frontal lobe epilepsy (Nobili et al., 2006; Terzaghi et al., 2008), and primary generalised epilepsy (Terzano, Parrino, Anelli, Halász, & Portera-Sánchez, 1989), but in turn, CAP has been found to modulate the occurrence and distribution of some of these events during sleep (Parrino et al., 2012). In particular, the A phase of the CAP cycle is thought to trigger and pace the events in sleepwalking (Guilleminault, 2006; Zucconi, Oldani, Ferini-Strambi, & Smirne, 1995), and bruxism (Kato, Rompre, Montplaisir, Sessle, & Lavigne, 2001; Macaluso et al., 1998), among others (for review, see Parrino et al., 2012). Also, childhood disorders of sleepwalking and sleep terrors show a typical pattern of *hypersynchronous delta activity* (Guilleminault, 2006). These delta bursts have been related to an increase in the percentage and rate of CAP subtype A₁ in SWS, and a decrease in the B duration of the CAP cycle, indicating an abnormally fast oscillatory pattern (Guilleminault et al., 2005; Guilleminault, Kirisoglu, Rosa, Lopes, & Chan, 2006; Zucconi, Oldani, Ferini-Strambi, & Smirne, 1995). Therefore, increased instability and fragmentation in SWS likely contributes to parasomnias (Guilleminault et al., 2006).

Considering the present findings of increased mentation in relation to unstable SWS in a healthy population, as well as Perogamvros et al.'s (2012) recent proposal that reward related activity may permit certain parasomnias during sleep, it is plausible that *arousal instability and the ML-DA system during sleep may be intimately related*. This suggestion is further supported by the fact that both of Perogamvros et al.'s cases of nocturnal eating had a childhood history of sleepwalking, which has been linked to both exploratory behaviours (Perogamvros & Schwartz, 2012), and hypersynchronous A₁ arousal activity (Guilleminault et al., 2006). An interesting endeavour would be to investigate CAP activity in patients with nocturnal eating disorders; based on the conclusions being drawn in this study, abnormal CAP activity is predicted in the SWS of these patients. It is uncertain at this stage whether arousal instability may accentuate ML-DA activity or act as a gateway for its release in sleep (i.e., through localised arousal), or perhaps both. This novel avenue of inquiry no doubt requires further confirmation and investigation.

Activation of the Brainstem during SWS

Linked to the latter ideas is the second proposed mechanism for arousal instability leading to enhanced dream recall during SWS. Specifically, the pedunculopontine tegmentum (PPT) constitutes the main components of the reticular activating system (Mena-Segovia & Bolam, 2011), and is one of the primary arousal-promoting mechanisms during sleep and waking (Saper et al., 2005, see Introduction). Furthermore, the PPT sends direct excitatory inputs to the VTA (Floresco, West, Ash, Moore, & Grace, 2003). As the PPT has been shown to be activated by *salient stimuli* (Pan & Hyland, 2005), Perogamvros and Schwartz (2012) have suggested that activation of this neuronal group may indirectly stimulate the ML-DA system during sleep. In relation to this, the desynchronised arousal subtypes (A_2 and A_3) have been linked to REM-on mechanisms in the brainstem (i.e., the PPT; Terzano et al., 2005); this will be discussed in more detail shortly. For now, however, I would like to suggest that the A_1 subtype may also have the potential to activate the PPT in certain circumstances.

The first component of the KC (N200-P300)—the graphoelement most commonly situated at the start of phasic arousals—has been shown to be related to low level information processing during sleep (Atienza et al., 2001; Halász, 2005; Jahnke et al., 2012). This has been interpreted as “a window” for monitoring the external environment during sleep (Halász et al., 2004). As many KCs and phasic arousal phenomena during sleep are *reactive* (i.e., related to input, either endogenous or exogenous), it may be the case that during unstable SWS the PPT, and hence the VTA, is stimulated *if stimuli are appraised as significant*. Put another way, A_1 arousals allow the opportunity for low level information processing; if an individual perceives a stimulus during sleep as being significant during this window of information processing, especially during unstable sleep, the PPT may be briefly activated. This possibility is further supported by the fact that the PPT is *already* activated during every ON phase of the 1 Hz oscillation (Dang-Vu et al., 2008; Mena-Segovia & Bolam, 2011), which, as mentioned, also temporally binds the A phases of the CAP cycles (Parrino et al., 2012). Therefore, depending on individual expectations and appraisals of the external environment (related to A_1 arousal activity), the transient activations of the PPT that exist throughout SWS may be further enhanced by salient stimuli (or perceptions thereof), leading to enough activation of the VTA for dreaming to be instigated. It may even be the case that these windows of activation in conjunction with motivationally salient memory activation of the VTA are needed for dreaming to occur in SWS.

A trait-like propensity for dreaming in SWS. In this study, as in others, not all participants were able to recall mentation from deep NREM sleep. Specifically, 25% of this

sample was unable to recall any mentation after both experimental nights. This is comparable to the 33% reported in the literature that were only able to produce one dream report in five nights, while 17% of samples generally were unable to produce any mentation after several nights from deep NREM sleep (Nielsen, 2000). These figures indicate that *sleep instability alone is not sufficient for dreaming to occur in the deeper stages of NREM sleep*, but that there are individualised differences, that perhaps in conjunction with sleep instability, result in a propensity for dreaming. A likely candidate for these differences is a trait-like predisposition for heightened reactivity to the external environment during sleep (Eichenlaub et al., 2013), and lowered auditory arousal thresholds⁴⁹ (AATs)—which are perhaps also another way of measuring heightened reactivity. Indeed, both heightened reactivity and lowered AATs have been shown to be related to increased dream recall (Eichenlaub et al., 2013; Zimmerman, 1970). This trait-like propensity for a higher level of attentional processing during sleep may mean that the sentient function hypothesised to be associated with KCs and A₁ arousals (as well as A₂ and A₃) is enhanced in these individuals. Therefore, high dream recallers may, especially during periods of unstable sleep, be more salient than low recallers, which may provide additional stimulation to the PPT.

Final Remarks on A₁ Activity and Sleep Mentation

Finally, it was found that mentation recalled following majority A₁ arousals was mostly classified as cognitive activity, or thoughtlike dreaming; this finding warrants attention. As argued thus far, arousal instability during SWS may provide a gateway to dreaming. In line with the ideas proposed by Nir and Tononi (2010), more often than not dreams are not realised in deep NREM sleep due to proposed physiological constraints on consciousness. When dreaming does occur it is likely in the form of localised arousal activity instigated by stimulation of the VTA, either directly through motivationally salient memories, or indirectly through the PPT (Perogamvros & Schwartz, 2012). As such, fragmented imagery and thoughtlike mentation may arise from periods of sleep comprised of A₁ arousal activity because the arousal portion of the phasic event is able to produce mentation, but shortly after this mentation may be prone to degradation and fragmentation by the sleep promoting effects of the A₁. Similarly, any ongoing mentation may be disrupted by the sleep promoting effects of the A₁ subtype, whether it starts within the CAP sequence or not. Considering that A₁ activity continues throughout the night, albeit not to the same extent as

⁴⁹ Auditory arousal thresholds are established by presenting a sleeping participant with gradually increasing auditory tones until they awaken; low AATs indicate a propensity to awaken after softer tones (Watson & Rechtschaffen, 1969; Zimmerman, 1970).

during the early hours (Terzano & Parrino, 2000), there is always the possibility of this disruption.

Desynchronised Microarousals and Dreaming

The arousal-retrieval model dictates that dream recall is only possible if an effective transfer of the dream experience into long term memory occurs during the short term memory trace of the dream; this effective transfer is hypothesised to be reliant on wake-related arousal processes occurring during the dream process (Koulack & Goodenough, 1976). Based on this hypothesis, it has recently been proposed that heightened brain reactivity may result in more waking during sleep, which would offer more opportunity for the transfer of the dream experience into long term memory and subsequently more dream recall (Eichenlaub et al., 2013). Sleep microstructure was not considered in Eichenlaub et al.'s (2013) study; however, as periods of NREM sleep demarcated by phasic arousal activity are considered to be unstable and to consequently allow for more reactivity to the external environment, it was proposed that phasic arousal activity may be related to more dream recall as well. Consistent with this observation, although not in relation to high and low recallers, unstable NREM sleep was found in the present study to yield more frequent dream recall than stable NREM sleep. This was particularly the case for unstable light NREM sleep characterised by A₂ arousals.

Interpreting these results within the framework of the arousal-retrieval model, it is unsurprising that the A₂ arousal subtype was related to enhanced dream recall. This subtype is associated with heightened arousal (higher EEG-related frequencies and autonomic perturbations) and reactivity in NREM sleep (Terzano et al., 2002), and accordingly, it may contribute towards the necessary level of arousal needed to encode and retrieve the memory trace of the dream experience (Koulack & Goodenough, 1976). Conversely, the *absence* of a relationship between the A₃ subtype and dream recall is counter-intuitive to a central assumption of the arousal-retrieval model; namely, that increased arousal results in increased dream recall. To reiterate, the A₃ subtype is representative of the most intense phasic arousal activity possible during sleep without behavioural waking (Atlas Task Force, 1992; Terzano et al., 2002; Parrino et al., 2012), and based on previous findings of enhanced recall in relation to intrasleep wakefulness and microarousals (Terzano et al., 1990), it was predicted that this arousal subtype would be significantly related to greater dream recall.

In trying to explain this apparently contradictory finding an examination of phasic arousal phenomena during REM sleep may be valuable. It has recently been shown that using an ongoing auditory stimulus to produce arousal activity in REM, subsequently suppressing

eye movements, results in *less* dream recall; indeed, 65% of the awakenings following such eye movement suppression in REM resulted in no imagery reports (Stuart & Conduit, 2009). Based on these results, it was concluded that "...the low-intensity repeated stimulus presented during the current study *engaged both attentional and arousal mechanisms, leading to a shift from internal cognitive processing to the outward processing of the external stimulus*" (p. 13, emphasis added). In other words, pervasive phasic arousal activity is able to draw some attention towards the outside world, and potentially away from an ongoing dream process, without resulting in behavioural waking.

This phenomenon has likewise been observed under more naturalistic conditions. In particular, Mullin, Kleitman, and Cooperman (1937) were able to show that depth of sleep varied as a function of temporal proximity to transient body movements during sleep. They tested this by administering a gradually increasing auditory tone several times throughout the night to a sleeper until they awakened enough to press a signal key that eliminated the tone. Following any major body movement, an initial increase and then decrease in the response threshold to the stimulus was reported. Moreover, body movements were shown to increase as the night progressed, with lower response thresholds maintained when the interval between movements was shorter. The authors concluded that depth of sleep throughout the night is best described by numerous curves of responsiveness (rather than a single curve), and that these curves share a direct relationship with body movements (Mullin et al., 1937). Furthermore, studies of gross body movements during REM sleep have revealed that awakenings in close proximity to these movements result in *less* dream recall compared with awakenings following longer periods of REM sleep devoid of such activity. Dream recall from REM sleep, immediately following a gross body movement, was comprised of 30% dreaming, 30% no content reports, and 40% fragmented and thoughtlike dreaming (Dement & Wolpert, 1958). Furthermore, 82% of REM dreams that were made after 10-20 minutes, but that were as short as REM dreams made after 4 minutes, were preceded by a body movement several minutes before. Based on these findings the authors concluded that "...long continuous dreams are more likely to be recalled in the absence of gross body movements while fragmented dreams are more likely to be recalled if the periods contain body activity" (p. 545).

Presently, gross body movements are part of the scoring criteria for ASDA defined microarousals (Atlas Task Force, 1992), as well as the A₂ and A₃ arousal subtypes within the

CAP framework (Terzano et al., 2001).⁵⁰ In fact, the accompaniment of either autonomic or muscle activity is mandatory to the scoring of microarousals and PAT (*phases d'activation transitoire*).⁵¹ PAT arousals have also been shown to be most prevalent during REM sleep (Schieber et al., 1971). It is reasonable to assume then, that the body movements referred to by Dement and Wolpert (1958) are phasic arousal activities occurring during REM sleep. Similarly, the A₂ and A₃ CAP arousals also follow the same time course described by Mullin et al. (1937)—they increase as the night progresses (Smerieri et al., 2007)—and therefore, the body movements described are most probably related to microarousals (or A₃ type arousals) occurring during the course of the sleep cycle. Indeed, even before the discovery of the sleep microstructure it was acknowledged that both spontaneous and provoked movements indicated transient elevations in the depth of sleep (Kamiya, 1961). Therefore, the conclusion reached by Conduit et al. (2009)—that arousals during sleep are able to engage attentional mechanisms and lead to a shift outward towards processing the external environment—appears valid for naturally occurring microarousals accompanied by autonomic and motor concomitants *throughout* sleep.

If microarousals during REM sleep are associated with fragmented dreaming, then what effect might they have on NREM dreaming? One explanation is that arousals, and associated body movements, may cause externalised attention (i.e., too much connection to the external environment) that may hinder the retrieval of the memory trace for the preceding dream, leading to what Koulack and Goodenough (1976) termed *interference*—when memories compete during sleep for space in the limited-capacity processing system, with the most salient of the set favoured in the competition. Originally it was proposed that interference occurred only during or shortly after awakening; however, the great majority of the time microarousals occur within an ongoing oscillatory pattern (i.e., a sequence), and it is therefore possible that interference may begin even before the awakening is made. Indeed, Halász et al. (2004) have described arousals and information processing as being “two sides of the same coin” (p. 18), as phasic arousal activity, according to these authors, offers windows for information processing during sleep.

In this way, the A₃ arousal subtype appears to either disrupt or enhance dreaming (as the present results that show *no* relationship with A₃ type arousals, not an inverse one); the A₂

⁵⁰ Occasionally, milder autonomic perturbations, including body movements, accompany the A₁ arousal subtypes as well (Terzano & Parrino, 2000).

⁵¹ While PAT, microarousals and the desynchronised CAP subtypes (A₂ and A₃) have slightly different scoring criteria, it is important to note that it is mostly the *same* phasic events that are being referred to.

subtype appears to be more consistently related to dream recall, while the A₁ subtype may have a degrading effect.⁵² As a result, dream recall during NREM sleep, and especially during unstable NREM sleep, appears to be prey to a number of factors related to either too much connectivity with the external environment, or an interference with consciousness. If we consider that NREM sleep is almost continuously oscillating between these factors, and that these oscillations are affected by individual and circumstantial differences, the discrepancies between REM and NREM dreaming are perhaps less surprising.

Furthermore, Sanders et al. (2009) argue that connectivity to the environment is controlled by the action of neuromodulators, and hypothesise that “unperturbed norepinephrinergic neurotransmission is important in maintaining connectedness, due to its central role in controlling attention to external stimuli” (p. 8). In particular, norepinephrine is involved with orienting attention to environmental stimuli (Coull, Büchel, Friston, & Frith, 1999), and is therefore argued to be well placed to control connectedness during sleep (Sanders et al., 2009). Moreover, inadequate suppression of norepinephrine has been shown to result in maintained connectedness under anaesthesia (Schrouff et al., 2011). While norepinephrine is reduced in NREM sleep, it still continues to modulate this state (Gottesmann, 2004). Consequently, the aminergic neuromodulation of NREM sleep may make this state more vulnerable to connectivity than REM, which is cholinergically modulated (Hobson et al., 2000; Hobson & McCarley, 1975), as it is proposed that cholinergic modulation subserves disconnected consciousness (Sanders et al., 2009). Considering that NREM dreaming is characteristically more fragmented and brief than REM dreaming (Antrobus et al., 1995; Foulkes, 1962; Monroe et al., 1965; Nielsen, 2000; Pivik, 1970), it is surprising that the fragmentary effects of phasic arousal activity on sleep mentation have hitherto not been considered, especially in view of the ubiquity of these phenomena in NREM sleep (Terzano & Parrino, 2000). Also interesting in this regard is that dream recall has been found to be enhanced following a noradrenergic beta-receptor blocker (Thompson & Pierce, 1999); whether dream recall was enhanced because of a decreased capacity for attentional shifts during sleep remains to be seen.

Compared with the instability and fragmentation of NREM sleep, then, REM sleep is stable and activated; it is a state that is neurophysiologically geared towards consciousness and a disconnection from the environment (Llinás & Paré, 1991). Conversely, NREM sleep is

⁵² Although, as discussed, this subtype may also paradoxically act as a gateway to dreaming; especially during deep NREM sleep.

variable in terms of its connectivity and consciousness. However, as the sleep period progresses and homeostatic pressure declines those neurophysiological factors subserving consciousness are progressively more sustained in NREM sleep (Nir & Tononi, 2010; Vyazovskiy et al., 2009). Corroborating this fact is that 74% of spontaneous morning awakenings have been found to arise from S2 sleep, and only 36% from REM; moreover, these awakenings yielded 95% dream recall from REM, and 91% from S2, and were indistinguishable in terms of their content (Cicogna, Natale, Occhionero, & Bosinelli, 1998). As such, late morning S2 mentation appears to no longer differ from REM mentation. However, connectivity to the environment is enhanced as the night progresses as well (as AATs become increasingly lower; Watson & Rechtschaffen, 1969), which brings the discussion to its next crucial point: *Dreaming appears to be related to a capacity for increased connectivity during sleep.*

The Paradoxical Disconnection of Dreaming

Nir and Tononi (2010) have recently pointed out that “[one’s] disconnection from the external environment when dreaming poses a central unsolved paradox, the answer to which might be instrumental for understanding dreams” (p. 97). In this respect, both the present findings, as well as previous research (Watson & Rechtschaffen, 1969), have shown that *subjectively rated depth of sleep is positively and significantly related to enhanced dream recall.* Furthermore, Watson and Rechtschaffen (1969) found that subjectively rated depth of sleep was related to actual AATs during sleep, making this subjective measurement a reliable indication of sleep depth. In this study REM sleep was subjectively rated as the deepest stage of sleep. All stable NREM sleep, as well as preawakening conditions predominantly comprised of desynchronised (A₂ and A₃) arousals, *did not* differ significantly from REM sleep in terms of depth of sleep; the only NREM sleep condition that did differ significantly from REM sleep was the A₁ majority group. This finding is paradoxical if we consider that the A₂ arousal subtype is related to *heightened* awareness during sleep compared with the A₁ subtype (Halász, 1998). However, if we consider that more dreaming was reported for the majority A₂ group than the A₁ group, it may be that *the dream process itself is able to facilitate the disconnection between the sleeper and their environment during sleep.*

This interpretation directly contradicts various theories of dreaming that propose that a prerequisite for the dream process is a *lack of connectivity* with the external environment (Antrobus, 1986; Hobson et al., 2000). Similarly, Llinás and Paré (1991) propose that wakefulness and REM sleep are equivalent brain states, albeit waking is governed by external sensory inputs, while REM sleep “*can be considered as a modified attentive state in which*

attention is turned away from sensory input, towards memories” (p. 522). More recently, the hypothesis proposed by Llinás and Paré (1991), that REM sleep is essentially a closed thalamocortical loop with intrinsically generated consciousness, has found support in studies of *phasic* REM sleep (i.e., REM sleep characterised by eye movement activity). Functional magnetic resonance imaging (fMRI) has shown that within human REM sleep widespread thalamocortical synchronised activity—reducing the processing of external stimuli (i.e., a thalamic gate)—is selectively enhanced during phasic REM, compared with tonic REM (Wehrle et al., 2007). Similarly, the highest auditory arousal thresholds were found for phasic REM and SWS (S3 and S4), while tonic REM and S2 were comparably lower (Ermis, Krakow, & Voss, 2010). Reduced higher order processing of external acoustic stimuli during phasic compared with tonic REM sleep has also been shown by ERP studies (Sallinen, Kaartinen, & Lyytinen, 1996). Together these findings suggest that only phasic REM can be considered a closed loop during sleep; the rest of S2 NREM sleep and tonic REM is comparatively more open to processing external stimuli.

Consequently, only during phasic REM sleep is the sleeper robustly disconnected from the environment through thalamic gating mechanisms. While some studies have found enhanced dream recall following awakenings from phasic REM sleep compared to tonic REM sleep (Molinari & Foulkes, 1969), both conditions yield high percentages of dream recall (Pivik, 1991; Rechtschaffen, 1973), and in certain cases neither quantitative nor qualitative differences between the two conditions have been observed (Pivik, 1970). Furthermore, dreaming outside of phasic REM sleep is well documented (Cicogna et al., 1998; Oudiette et al., 2012; Pivik, 1970), and especially during sleep onset when AATs are lower than phasic REM as well (Carskadon & Dement, 2011; Foulkes & Vogel, 1965), and consequently, it cannot be argued that a complete disconnection from the environment is a *prerequisite* for the dream process to occur. On the contrary, with the exception of phasic REM sleep, it appears as though *periods of sleep that are more vulnerable to connecting with the external environment are regularly related to dreaming in both REM and NREM sleep*. This conclusion corroborates Eichenlaub et al.’s (2013) finding that high dream recallers are more reactive to their external environments than low dream recallers. The enhanced P3a response in high dream recallers is associated with *higher attentional orientation* during sleep, meaning that they are susceptible to increased connectivity with the environment (Eichenlaub et al., 2013). Therefore, paradoxically, it may be that these participants have higher dream recall not only because they wake up more, but because they are more at risk for waking up.

Are Dreams the Guardians of Sleep?

Dreaming during sleep has been described as a state of disconnected consciousness (Nir & Tononi, 2010). Based on both the present findings and those in the literature, it is hypothesised that the dream process is *fundamentally involved in facilitating this disconnection*, rather than being just a product of it (as proposed by other models of dreaming). The novelty of this proposal makes it difficult at this stage to specify any exact mechanisms whereby this may be achieved. However, some additional findings in support of the idea are presented.

Sleep State Misperception: A Consequence of Failed Dreaming?

In this study dreaming was most associated with A₂ arousal activity, which occurs when an A₁ antiarousal response gives way to desynchronised arousal activity—i.e., when sleep-promoting mechanisms fail to suppress arousal activity during sleep (Terzano et al., 2002). The A₂ subtype thus represents more pronounced activation of arousal and attentional processes than the A₁, resulting in increased connectivity with the external environment and information processing (Halász et al., 2004). However, the A₂ subtype is less pronounced, in terms of its autonomic and motor associations, than the A₃ subtype (Terzano et al., 2001). At least some of the time, though, increased dream recall was associated with A₃ arousals as well. Congruent with the present results, it has been shown experimentally that the induction of phasic arousal activity without waking during light NREM sleep (using an auditory stimulus), is able to produce enhanced visual dream imagery (Conduit, Bruck, & Coleman, 1997).

Interestingly, it has also been shown that *sleep state misperception* in paradoxical insomnia—the subjective feeling of being awake despite being objectively asleep as defined by polysomnography—is associated with an increase in: i) the total CAP rate in S1 and S2, ii) the arousal index, and, iii) the percentage of A₂ activity (Parrino, Milioli, De Paolis, Grassi, & Terzano, 2009). Furthermore, the severity of insomnia has recently been shown to be negatively correlated with novelty seeking and reward dependence (Park, An, Jang, & Chung, 2012), psychometrically measured personality traits which correspond with ML-DA activity (Dreher, Meyer-Lindenberg, Kohn, & Berman, 2008). Based on the theoretical assumption that ML-DA is fundamental to dream genesis, it is therefore plausible that underactivity of the ML-DA system may be implicated in sleep state misperception. In other words, when dreaming does not occur in relation to desynchronised arousal activity (especially A₂), a functional disconnection is not fully achieved.

Consistent with this conclusion is the description of a case of dream loss following a temporo-occipital haematoma

After the brain injury the patient started complaining about his bad quality of sleep. He spent many hours in bed, seemingly asleep, but used to wake up with the feeling of not sleeping much...After a while the patient was able to explain that his problem was a lack of dreaming and not a difficulty to sleep.

(Poza & Marti Massó, 2006, p. 2)

While sleep state misperception is not specifically referred to in this case the patient's description meets the criteria for it, especially considering an intact sleep macrostructure was verified via polysomnography after his brain injury (Poza & Marti Massó, 2006). Whether this feeling of being awake persisted in REM or NREM sleep is uncertain; based on the present findings, though, it would be informative to investigate subjective depth of sleep, sleep state misperception, and sleep fragmentation in brain injured patients experiencing dream loss.

Age-Related Changes in Sleep Fragmentation, Dopamine, and Dreaming

The fragmentation of sleep and increased intrasleep wakefulness associated with aging is well documented (Dijk, Duffy, & Czeisler, 2001; Haimov & Lavie, 1997; Parrino, Boselli, Spaggiari, Smerieri, & Terzano, 1998). In particular, healthy elderly adults show decreased sleep consolidation and increased awakenings, especially in the second half of the night (Dijk et al., 2001), as well as an increased percentage of A₂ and A₃ arousals, and a decreased percentage of A₁ arousals (Parrino et al., 1998). Congruent with the decrease in A₁ arousals is less homeostatic sleep pressure (measured according to slow wave density), and less S4 sleep (Guazzelli et al., 1986; Wei, Riel, Czeisler, & Dijk, 1999). Moreover, the sleep of older individuals is generally more susceptible to disruption by auditory stimuli due to lower AATs (Dijk et al., 2001). While the decrease in slow wave propensity (and A₁ arousals), and spindles is hypothesised to be related to increased spontaneous awakenings in S3 sleep (Salzarulo et al., 1999), the inability for consolidated S2 has led to conclusions that "...age related changes in sleep consolidation are primarily related to a diminution of the protective role of nonREM sleep, including stage 2 sleep" (Dijk et al., 2001, p. 576).

Furthermore, age related decreases in dopamine activity in the elderly have been documented (Kaasinen et al., 2000; Volkow et al., 2000), and decreased basal dopamine levels in the midbrain in relation to reduced striatal activity in the anticipation of reward have

been found as well (Dreher et al., 2008; Mell et al., 2009). Finally, dreaming in the elderly is reported to be significantly less visual in both REM and NREM sleep (Fein et al., 1985), and report length has been found to drop substantially in the *late portion of the night*, in contrast to younger subjects whose report length increased linearly with time of night (Nielsen, 2010). Moreover, percentage dreaming from REM and NREM sleep is reduced in elderly adults compared with young adults (Chellappa, Münch, Blatter, Knoblauch, & Cajochen, 2009).

In light of the discussion thus far, it is tempting to propose that age-related decreases in dopamine may be related to reduced dream recall, and subsequent sleep fragmentation in the elderly. Furthermore, while intrasleep wakefulness is associated with increased dream recall in younger subjects (Eichenlaub et al., 2013), it is conversely associated with *decreased* dreaming in the elderly. Moreover, less dreaming occurs precisely when sleep fragmentation is at its worst later on in the night, when the protective mechanism provided by slow wave activity has declined. In line with the conclusions being drawn in this discussion, the following questions warrant attention: Are ontogenic reductions in dopamine related to decreased dream recall in the elderly? Is reduced dreaming related to sleep fragmentation and lowered AATs in the later portion of the night? If both of these questions prove positive, one might ask whether dreaming provides a protective mechanism whereby a functional disconnection can be maintained during certain stages of sleep. Considering that there are a number of other factors leading to the decline in sleep quality, such as reduced spindles and slow waves (which provide thalamocortical gating), and changes in cholinergic modulation (Bartus, Dean, Beer, & Lippa, 1982), the exact role that the ML-DA system may play in sleep maintenance (via dreaming) awaits further investigation.

Dreaming as a 'Gateway' to REM Sleep

As discussed in the introduction, desynchronised microarousals facilitate the transitioning to REM by stimulating arousal-promoting mechanisms (Halász & Bódizs, 2013; Halász et al., 2004). In the present study enhanced dream recall was found specifically in relation to an equal, or increased, number of A₂ arousals relative to A₁ arousals. Furthermore, 77% of awakenings following either an equal number of A₂ and A₁ arousals, or majority A₂ arousals, occurred on the *ascending slope* (AS) of the sleep cycle. The preference for A₂ activity on the AS could be taken as an indication that, had the awakening not been made, that this relative increase in A₂ activity was in preparation for an ensuing REM period. This finding offers potential support for the second hypothesis of RAM, that "...the activation of emotionally-relevant information such as reward can potentially influence the generation of REM sleep" (Perogamvros & Schwartz, 2012, p. 1940). The authors suggest that the VTA is

able to modulate activity in the sublaterodorsal nucleus of the pons, a key structure in generating REM sleep (Boissard et al., 2002); in particular the VTA provides efferent projections to the sublaterodorsal nucleus “thus potentially gating the onset of REM sleep” (p. 1940). Accordingly, dreaming was found in this study to accompany portions of sleep anticipating the REM period. Furthermore, these arousal subtypes have been linked to REM on mechanisms that involve activation of the PPT (Terzano et al., 2005).

Nevertheless, while the tonically regulated slopes of the sleep cycle largely determine the type of phasic activity (Halász et al., 2004) dreaming appeared equally likely to accompany the remaining 23% of predominant A₂ arousal activity occurring *outside* of the AS of the sleep cycle. Therefore, dreaming appears to be fairly predictably and consistently related with this arousal activity, regardless of when in the sleep cycle it occurs. Could it subsequently be argued that dreaming is merely an epiphenomenon of this activity? An alternative argument, based on the lines of reasoning in this discussion so far, is that dreaming essentially facilitates A₂ activity by preventing these arousals from resulting in behavioural awakening and pathologically fragmented sleep.

In support of this argument is the fact that dreaming and A₂ activity appear to be dissociated in paradoxical insomnia, where, as mentioned, a pathological increase in these types of arousals are accompanied by sleep state misperception rather than dreaming (Parrino et al., 2009). Thus, dreaming is not *unconditionally* related to this activity, and when it does not accompany it, pathological sleep disruption ensues. Furthermore, Halász & Bódizs (2013) propose that, by allowing sleep to remain flexible, microstructural phenomena are able to accomplish two seemingly contradictory states: *the maintenance of sleep separate to the environment and the simultaneous connection with the environment*. This standpoint is perhaps best elaborated upon by the following statement

Every biologic system tries to assure autonomy to achieve independence from the surrounding, and at the same time relies on the interrelationship between the organism and the surrounding world, which is essential for adaptation and survival of the system. Therefore an organism should avoid external stimuli and try to regain the original prestimulus state, but paradoxically, it will use the stimulus for building up its autonomic state

(Halász et al., 2004, p. 18).

According to this perspective dreaming may naturally accompany phasic arousal phenomena that subserve a connection with the environment to allow for the simultaneous, and paradoxical, autonomy of the sleeping state from it. In this way, dreaming may provide stability to the fluctuating depths of sleep and arousal activity especially occurring prior to REM sleep, and later on in the night. It may well be predicted, then, that dream loss due to brain injury will lead to increased sleep fragmentation prior to REM sleep and subsequently delayed REM latencies. However, based on this hypothesis, dream loss may affect any portion of NREM sleep that is less protected by homeostatically induced antiarousal processes, potentially resulting in a sleep profile that is similarly disrupted to that of normal aging (above).

Permeability of the Functional Disconnection

Many authors consider the relative lack of incorporation of various stimuli into dream content to be an indication that the dream process requires disconnection from the environment, or at least is an indication of it (Horne, 2012; Nir & Tononi, 2010). However, little attention has been paid to instances of successful incorporation. Often when a stimulus is incorporated it is either novel, threatening, or meaningful; for instance concern-related verbal stimuli were incorporated significantly more compared with neutral stimuli (Hoelscher, Klinger, & Barta, 1981), and more physically invasive stimuli such as water spray (42%; Dement & Wolpert, 1958), electrical stimuli (56%; Koulack, 1969), and a pressure cuff on leg (87%; Nielsen, 1993) are more often incorporated than less invasive stimuli (e.g., neutral auditory stimulus). Recently it has been shown that olfactory stimuli are able to change the emotional tone of dreams depending on the hedonic characteristics of the stimulus used (i.e., rotten eggs versus smell of roses; Schredl et al., 2009). Therefore, the disconnection apparent in dreams appears to be more permeable when external stimuli require further appraisal and higher levels of information processing. As a result, dreaming may allow a certain degree of information processing during sleep without full waking—a kind of flexible appraisal of the environment when necessary. As it has been proposed that CAP allows *stability through lability* during NREM sleep, this notion of a flexible disconnection permitted by the dream process is congruent with that proposed for CAP and unstable sleep.

Dreaming: Interest Turned Inwards?

Freud was the first to propose that dreams “serve the purpose of prolonging sleep instead of waking up. Dreams are the guardians of sleep, and not its disturbers” (Freud, 1900/2006, p. 223). He argued that dreams are part of a process of wish-fulfilment that

enables the sleeper to continue sleeping because “the internal demand which was striving to occupy him has been replaced by an external experience, whose demand has been disposed of” (p. 223). More recently, a number of very important parallels have begun to be drawn between Freud’s drive theory and concept of libido, and the ML-DA system (Panksepp & Solms, 2012; Perogamvros & Schwartz, 2012; Solms, 1997, 2000, 2012; Solms & Turnbull, 2002; Yu, 2001a). The ML-DA has also been placed at the centre of the dream process (Perogamvros & Schwartz, 2012; Solms, 2000), and is proposed to be essential to the functions of REM sleep as well (Horne, 2012; Perogamvros & Schwartz, 2012). Might the dream then serve to gratify the internal demand of the ML-DA system during sleep? Regardless of whether the dream is initiated via psychological (memory and thoughts) or sensory (arousal) sources?

Horne (2009) has proposed that dreaming may act as a distraction from wakefulness, thereby allowing the continuity of sleep. Similarly, Nir and Tononi (2010) have alluded to the notion that dreaming may alter attentional mechanisms during sleep, consequently assisting in a disconnection. A proposed neural mechanism whereby dreaming is able to protect sleep is unfortunately beyond the scope of this discussion, and considering the novelty of many of the predictions put forth, is perhaps also premature. However, assuming for the moment that the dream process is able to facilitate a functional disconnection during portions of the sleep cycle that are more at risk for disruption, I propose that the mechanism whereby this may be achieved is along the lines of those mentioned above. Considering the substantial amount of evidence linking the ML-DA system to dreaming, the novelty of the dream environment may be able to draw attention inwards when it is threatened to be drawn outwards; a kind of *internalisation of interest* that would otherwise be spent on the environment. The ability of the dream process to occasionally incorporate more meaningful stimuli is also an indication that this interest can be more forcefully drawn outwards when needed, or that the dream process may incorporate stimuli that are of moderate concern for better appraisal. This hypothesis is not meant to imply, however, that this is the only function of the ML-DA system during sleep.

NREM Dreaming: Supplementary ML-DA Activation?

Like the reactive deactivations (antiarousals) that protect sleep, dreaming may protect activated sleep by facilitating arousal activity through a functional disconnection. It may also act as a gateway for REM sleep (Perogamvros & Schwartz, 2012). However, is it possible that the occurrence of dreaming in relation to A₂ arousals (and also A₃ arousals at times) not only provides a protective mechanism for sleep, but that phasic arousal activity also provides

a mechanism whereby the ML-DA can be stimulated by external and endogenous stimuli? Halász and Bódizs (2013) argue that during the descending slope and trough of a sleep cycle and when homeostasis is high, phasic antiarousal activity (A_1) not only protects sleep, but also provides additional homeostasis (slow wave activity) to frontal regions. In this way, external stimuli are used as “fuel” for homeostasis, providing “delta injections” during sleep (p. 42). Could the dream system be stimulated in the same way? Are A_2 and A_3 arousal subtypes, in addition to stimulating arousal-promoting mechanisms during sleep in relation to ultradian and circadian factors, likewise able to provide dopamine injections to the ML system?

The ML-DA has been crucially implicated in a number of processes during REM sleep, including: memory consolidation and emotional regulation (Perogamvros & Schwartz, 2012), energy homeostasis (Horne, 2012), neurodevelopmental processes (Nieoullon, 2002), and the satisfaction of motivational urges and therein psychological tensions (Solms, 1997, 2000). Furthermore, the portion of sleep that arguably provides the most intense stimulation to the ML-DA system—phasic REM—also leaves an animal exceptionally vulnerable to its environment due to the reduced responsiveness of this state (Ermis et al., 2010). This is perhaps the reason why phasic REM only occupies 20-30% of the REM episode (Aserinsky, 1969), and why prior fear conditioning has been found to significantly reduce REM-related PGO activity (Sanford, Silvestri, Ross, & Morrison, 2001). *When an animal or human is unable to sustain REM, can phasic arousal processes partially replace the functions of phasic REM sleep?*

These questions are especially intriguing considering that PGO activity is proposed to aid brain development during early gestation by providing “...a substitute for a lack of external stimulation, especially within the confines of the uterus” (Horne, 2012, p. 3). Similarly, PGO activity is generally considered “an internally generated, sustained-‘pseudosensory bombardment’ of the cortex, to which the animal is constantly reorienting its attention, internally” (Horne, 2012, p. 3). Evoked PGO responses also do not habituate during REM sleep, which suggests that each external stimulus is treated as novel (Johnson & Lubin, 1967). While PGO activity is not a determinant of dreaming, evoked arousals (and inferred PGO activity) have been shown to result in enhanced dreaming in S2 sleep (Conduit et al., 1997). This study has also shown that dreaming is enhanced during unstable sleep characterised by CAP activity; and likewise, CAP activity (or unstable sleep) has also been shown *not* to habituate to external stimuli, indicating that during unstable sleep each stimulus received is appraised as novel (Halász et al., 2004). It could reasonably be argued, based on

these parallels, that external stimuli and the perception of external stimuli (i.e., PGO activity), influence dreaming and ML-DA system similarly. Finally, in relation to this, it has been proposed that PGO activity is involved with producing the disconnection apparent during phasic REM sleep (Ernis et al., 2010). The ways in which a functional disconnection is achieved in NREM sleep in relation to external stimuli might therefore be similar to the disconnection achieved during phasic REM in response to intensive pseudostimuli (i.e., PGO activity), albeit less robust.

Additional Observations

Dreaming during Stable Sleep

It is important to point out that dreaming did not only occur with unstable sleep in this study. Substantial dreaming still took place during stable NREM sleep. This is further evidence that there are several underlying factors involved with dream genesis during sleep, only some of which have attempted to be elucidated here. For instance, access to memory stores, or memory consolidation processes, may at any time provide a psychical source of dreaming. The more interesting question, then, may be why certain individuals are more prone to dreaming as a result of psychical sources? As has also been discussed in some detail, sleep instability alone is not sufficient for dreaming to occur. Individual differences in brain reactivity, as well as perhaps the motivational and emotional salience of memories from the previous day, may be crucial factors determining the frequency of dream recall on an interindividual basis.

Furthermore, stable NREM sleep had substantially more cognitive activity than unstable sleep. It has been suggested in this study that the fragmentary and thoughtlike quality of NREM dreaming can arise from the impact of certain phasic arousal activity on the dream process during NREM sleep. The reasons for cognitive activity in stable NREM sleep are less clear. It may be that this type of mentation persists when there is insufficient impetus for more involved dreaming, as may be the case for much of stable NREM sleep. Alternatively, the awakening technique in this study may have caused hypnopompic imagery, especially during stable sleep (this is discussed further in the upcoming section).

Time of Night Effects

The most pronounced time of night effects in this study appeared to be related to the prevalence of A_2 activity in the second half of the night, rather than later sleep in general. This was evidenced by the fact that dream recall was reliably related to unstable sleep characterised by A_2 activity, the conditions for which were ideal in the second half of the night. This was not the case for stable NREM sleep, where no time of night effects were

present. This may have been an idiosyncrasy of the present study or it may be the case that dreaming is more prevalent in the second half of the night only during unstable NREM sleep; this effect is likely related to the predominance of desynchronised arousal activity and the relative decrease in synchronised arousal activity during the second half of the night. This proposal no doubt requires further investigation; if it is confirmed, it may be the case that the time of night effect so commonly associated with dreaming is largely a product of changes in phasic activity. It should be kept in mind, though, that phasic and tonic processes interact during sleep, and that changes in phasic activity are underlain by tonic, chemical changes as well.

Limitations of the Present Study and Future Research

Several points concerning the current methodology should be made. First, the method of awakening during sleep was not ideal. Future studies should use a buzzer or stimulus that is able to produce waking more instantaneously. As the awakening method in this study involved the experimenter walking into the room and calling the participant's name, the awakening period was most likely protracted. While the same method of awakening was used throughout, protracted awakenings have previously been shown to result in thoughtlike mentation (Shapiro et al., 1963). It may especially be the case that those periods of sleep that contained substantial cognitive activity (i.e., stable sleep), might contain less thoughtlike recall, or perhaps even less overall recall, were more abrupt awakenings made. Alternatively, prolonged waking during periods of unstable sleep may have led to more dreaming. The reasons why prolonged waking would lead to opposing types of mentation from stable and unstable NREM sleep might itself be a potentially interesting investigation.

Second, the adaptation night in this study did not include EEG monitoring; in future, it would be better to ensure via polysomnography that all participants' are free of sleep disorders. In this study, only subjective measures of sleep quality were used to screen for sleep disorders. Furthermore, this would allow for the microstructural baselines to be established; this may contribute additionally valuable data, as it has been proposed in this study that microstructural differences exist between high and low dream recallers.

Third, although the sleep lab in this study was partially soundproofed, being in a hospital setting there was still a considerable amount of noise. While an effort was made not to awaken participants after any loud auditory stimuli, the possibility that some of the preawakening phasic arousal activity in this study occurred in relation auditory stimuli cannot be excluded. In the case of the A₃ arousals specifically, the lack of relationship with dreaming may be partially due to certain of these phenomena being evoked by novel and unexpected

stimuli. This would not change the conclusions reached in this study regarding the impact that varying degrees of arousal may have on dream recall; nonetheless, the issue of spontaneous versus evoked CAP activity in relation to dream recall no doubt requires further investigation.

Fourth, it was not considered in this study how earlier disruptions may affect the sleep microstructure later in the evening. The number of awakenings per night (five to six) likely resulted in artificially induced homeostatic pressure in the later portion of the night. While this was helpful in making conclusions regarding the effect of A_1 arousal activity on dream recall, fewer awakenings per night may allow for less disruption and more naturalistic microstructural activity. Furthermore, as many of the groups were conceptualised post hoc, and through statistical manipulation, in certain cases the samples sizes are small (i.e., the A_2 majority group). Future methodologies should aim to remedy this. Furthermore, due to the novelty of this project, and the unpredictability of CAP, the awakening schedule was only very roughly predetermined. Based on these findings, though, future studies will be able to set a more precise awakening schedule.

Finally, participants all followed the same sleep schedule, with lights off between 22:00 and 23:00. While none of the participants' habitual bed times differed greatly from this time, no effort was made to schedule sleep onset according to their natural sleep times. Likewise, participants' sleep schedules were not objectively confirmed prior to their participation. Taking objective measures to ensure that participants are not sleep deprived in any way, as well as tailoring the experimental sleep schedule to their individual sleep time, could only ensure more accuracy.

Concluding Remarks

In summary, the findings of this study suggest that components of the sleep microstructure may independently, and variably, impact upon the dream process and therein influence the quality and occurrence of NREM dreaming. Furthermore, arguments have been put forth that dreaming is not only receptive to these physiological sleep processes, but that it may provide a protective function by accompanying periods of active sleep that are on the threshold of disruption, *essentially facilitating a functional disconnection between the sleeper and their environment*. Several lines of converging evidence suggest that the ML-DA system is critically related to dream genesis; assuming that this is the case, the affective quality of the ML-DA system—i.e., highly motivated and novelty driven—manifested in the form of a dream, may provide a subjective environment whereby attentional processes can be altered during sleep: Interest can be drawn inwards instead of outwards, essentially providing a

functional disconnection during certain stages of sleep. Therefore, paradoxically, I propose that a functional disconnection is partly achieved by *using* external disruptions and stimuli, to a certain extent, to stimulate the ML-DA system. Similarly, it is proposed that during phasic REM sleep the endogenous production of pseudosensory information is able to drive the ML-DA in a manner akin to actual external stimuli; the crucial difference being that outside of phasic REM sleep this process is more prone to disruption, and adaptively so.

Additionally, during unstable sleep habituation to external stimuli does not occur, indicating heightened information processing during these periods. Dreaming may therefore allow for the simultaneous low level appraisal of the environment and the continuation of sleep. The incorporation of a small percentage of more meaningful stimuli into the dream environment may be taken as evidence of the adaptive flexibility of the functional disconnection allowed by the dream. Furthermore, the inconsistency in dream recall between individuals may partly be attributed to individual variations in the vulnerability of sleep as a function of trait-like differences in reactivity to the external environment, which it has already been shown are related to dream frequency. Finally, it is hypothesised that, where necessary, NREM dreaming may allow for the partial substitution of the functions subserved by phasic REM sleep. Inherent to this hypothesis is the further prediction that the ML-DA system is critically involved with the functions of phasic REM sleep as well.

In conclusion, the primary aim of this study was to explore the relationship between sleep microstructure and mentation, in the hopes of trying to better understand the psychophysiology of NREM dreaming. The present results have tentatively confirmed that the ways in which dream recall relates to various aspects of the sleep microstructure may have the potential to further our understanding of the dream process. Indeed, decades of research on the macrostructural sleep stages have not managed to elucidate the essential functions and mechanisms of dreaming; subtler aspects of sleep are needed to provide the complexity necessary for a fuller understanding of the dream process. In this regard, the disconnection of the sleeper from the environment is an unsolved paradox that may be instrumental in understanding the dream process; by drawing on the sleep microstructure this study has attempted to explain this paradox, the central hypotheses of which await confirmation.

REFERENCES

- Achermann, P., Dijk, D., Brunner, D. P., & Borbély, A. A. (1993). A model of human sleep homeostasis based on EEG slow-wave activity: Quantitative comparison of data and simulations. *Brain Research Bulletin*, *31*(1), 97-113.
- Amzica, F., & Steriade, M. (2002). The functional significance of K-complexes. *Sleep Medicine Reviews*, *6*(2), 139-149.
- Antrobus, J. (1983). REM and NREM sleep reports: Comparison of word frequencies by cognitive classes. *Psychophysiology*, *20*(5), 562-568.
- Antrobus, J. S. (1986). Dreaming: Cortical activation and perceptual thresholds. *Journal of Mind and Behavior*,
- Antrobus, J. S., Fein, G., Jordan, L., Ellman, S. J., & Arkin, A. M. (1991). Measurement and design in research on sleep reports.
- Antrobus, J., Kondo, T., Reinsel, R., & Fein, G. (1995). Dreaming in the late morning: Summation of REM and diurnal cortical activation. *Consciousness and Cognition*, *4*(3), 275-299.
- Antrobus, J., Schnee, R., Lynn, A., Silverman, S., & Offer, V. (1976). A psycholinguistic coding manual for reports of sleep experience. *Educational Testing Service Collection*, 8737
- Aricò, D., Drago, V., Foster, P. S., Heilman, K. M., Williamson, J., & Ferri, R. (2010). Effects of NREM sleep instability on cognitive processing. *Sleep Medicine*, *11*(8), 791-798.
- Aserinsky, E. (1969). The maximal capacity for sleep: Rapid eye movement density as an index of sleep satiety. *Biological Psychiatry*,
- Aserinsky, E., & Kleitman, N. (1953). Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. *Science*, *118*(3062), 273-274.
- Aserinsky, E., & Kleitman, N. (1955). Two types of ocular motility occurring in sleep. *Journal of Applied Physiology*, *8*(1), 1-10.
- Aserinsky, E. (1967). Physiological activity associated with segments of the rapid eye movement period. *Research Publications - Association for Research in Nervous and Mental Disease*, *45*, 338-350.
- Aston-Jones, G., & Bloom, F. (1981). Activity of norepinephrine-containing locus coeruleus neurons in behaving rats anticipates fluctuations in the sleep-waking cycle. *The Journal of Neuroscience*, *1*(8), 876-886.

- Atienza, M., & Cantero, J. L. (2001). Complex sound processing during human REM sleep by recovering information from long-term memory as revealed by the mismatch negativity (MMN). *Brain Research, 901*(1), 151-160.
- Atienza, M., Cantero, J. L., & Escera, C. (2001). Auditory information processing during human sleep as revealed by event-related brain potentials. *Clinical Neurophysiology, 112*(11), 2031-2045.
- Atlas Task Force of the American Sleep Disorders Association. (1992). EEG arousals: Scoring rules and examples. *Sleep, 15*(2), 174-184.
- Bartus, R. T., Dean, R. L., Beer, B., & Lippa, A. S. (1982). The cholinergic hypothesis of geriatric memory dysfunction. *Science, 217*(4558), 408-414.
- Bastuji, H., García-Larrea, L., Franc, C., & Mauguière, F. (1995). Brain processing of stimulus deviance during slow-wave and paradoxical sleep: A study of human auditory evoked responses using the oddball paradigm. *Journal of Clinical Neurophysiology, 12*(2), 155-167.
- Bastuji, H., Perrin, F., & Garcia-Larrea, L. (2003). Event-related potentials during forced awakening: A tool for the study of acute sleep inertia. *Journal of Sleep Research, 12*(3), 189-206.
- Bazil, C. W., & Malow, B. (2005). Sleep and epilepsy. *Handbook of Clinical Neurophysiology, 6*, 281-291.
- Beaver, J. D., Lawrence, A. D., van Ditzhuijzen, J., Davis, M. H., Woods, A., & Calder, A. J. (2006). Individual differences in reward drive predict neural responses to images of food. *The Journal of Neuroscience, 26*(19), 5160-5166.
- Benca, R. M., Obermeyer, W. H., Thisted, R. A., & Gillin, J. C. (1992). Sleep and psychiatric disorders: A meta-analysis. *Archives of General Psychiatry, 49*(8), 651.
- Benson, D. F., & Greenberg, J. P. (1969). Visual form agnosia: A specific defect in visual discrimination. *Archives of Neurology, 20*(1), 82.
- Bischof, M., & Bassetti, C. L. (2004). Total dream loss: A distinct neuropsychological dysfunction after bilateral PCA stroke. *Annals of Neurology, 56*(4), 583-586.
- Bohlin, G. (1971). Monotonous stimulation, sleep onset and habituation of the orienting reaction. *Electroencephalography and Clinical Neurophysiology, 31*(6), 593-601.
- Boissard, R., Gervasoni, D., Schmidt, M. H., Barbagli, B., Fort, P., & Luppi, P. (2002). The rat ponto-medullary network responsible for paradoxical sleep onset and maintenance: A combined microinjection and functional neuroanatomical study. *European Journal of Neuroscience, 16*(10), 1959-1973.

- Borbély, A. A., & Achermann, P. (1999). Sleep homeostasis and models of sleep regulation. *Journal of Biological Rhythms*, *14*(6), 559-570.
- Borbély, A. A., Achermann, P., Trachsel, L., & Tobler, I. (1989). Sleep initiation and initial sleep intensity: Interactions of homeostatic and circadian mechanisms. *Journal of Biological Rhythms*, *4*(2), 37-48.
- Bosinelli, M., Cicogna, P., & Molinari, S. (1974). The tonic-phasic model and the feeling of self-participation in different stages of sleep.
- Braun, A. R., Balkin, T. J., Wesensten, N. J., Gwadry, F., Carson, R. E., Varga, M., . . . Herscovitch, P. (1998). Dissociated pattern of activity in visual cortices and their projections during human rapid eye movement sleep. *Science*, *279*(5347), 91-95.
- Braun, A., Balkin, T., Wesenten, N., Carson, R., Varga, M., Baldwin, P., . . . Herscovitch, P. (1997). Regional cerebral blood flow throughout the sleep-wake cycle. an H2 (15) O PET study. *Brain*, *120*(7), 1173-1197.
- Brown, J. W. (1972). *Aphasia, apraxia, and agnosia: Clinical and theoretical aspects*. Springfield: CC Thomas.
- Buysse, D. J., Reynolds III, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193-213.
- Carskadon, M. A., & Dement, W. C. (1994). Normal human sleep: An overview. *Principles and Practice of Sleep Medicine*, *4*, 13-23.
- Carter, R. M., MacInnes, J. J., Huettel, S. A., & Adcock, R. A. (2009). Activation in the VTA and nucleus accumbens increases in anticipation of both gains and losses. *Frontiers in Behavioral Neuroscience*, *3*
- Cavallero, C., Cicogna, P., Natale, V., & Occhionero, M. (1992). Slow wave sleep dreaming. *Sleep: Journal of Sleep Research & Sleep Medicine*,
- Chellappa, S. L., Frey, S., Knoblauch, V., & Cajochen, C. (2011). Cortical activation patterns herald successful dream recall after NREM and REM sleep. *Biological Psychology*, *87*(2), 251-256.
- Chellappa, S. L., Münch, M., Blatter, K., Knoblauch, V., & Cajochen, C. (2009). Does the circadian modulation of dream recall modify with age? *Sleep*, *32*(9), 1201.
- Chou, T. C., Bjorkum, A. A., Gaus, S. E., Lu, J., Scammell, T. E., & Saper, C. B. (2002). Afferents to the ventrolateral preoptic nucleus. *The Journal of Neuroscience*, *22*(3), 977-990.

- Cicogna, P., Natale, V., Occhionero, M., & Bosinelli, M. (1998). A comparison of mental activity during sleep onset and morning awakening. *Sleep: Journal of Sleep Research & Sleep Medicine*,
- Cohen, J., Cohen, P., & Stephen, G. West, and leona S. aiken (2003). *Applied Multiple regression/correlation Analysis for the Behavioral Sciences*, 3
- Conduit, R., Bruck, D., & Coleman, G. (1997). Induction of visual imagery during NREM sleep. *Sleep: Journal of Sleep Research & Sleep Medicine*,
- Cory, T. L., Ormiston, D. W., Simmel, E., & Dainoff, M. (1975). Predicting the frequency of dream recall. *Journal of Abnormal Psychology*, 84(3), 261.
- Coull, J., Büchel, C., Friston, K., & Frith, C. (1999). Noradrenergically mediated plasticity in a human attentional neuronal network. *Neuroimage*, 10(6), 705-715.
- Cudeck, R. (2000). Exploratory factor analysis. *Handbook of Applied Multivariate Statistics and Mathematical Modeling*, , 265-296.
- Czisch, M., Wehrle, R., Kaufmann, C., Wetter, T. C., Holsboer, F., Pollmächer, T., & Auer, D. P. (2004). Functional MRI during sleep: BOLD signal decreases and their electrophysiological correlates. *European Journal of Neuroscience*, 20(2), 566-574.
- Czisch, M., Wetter, T. C., Kaufmann, C., Pollmächer, T., Holsboer, F., & Auer, D. P. (2002). Altered processing of acoustic stimuli during sleep: Reduced auditory activation and visual deactivation detected by a combined fMRI/EEG study. *Neuroimage*, 16(1), 251-258.
- Dahan, L., Astier, B., Vautrelle, N., Urbain, N., Kocsis, B., & Chouvet, G. (2006). Prominent burst firing of dopaminergic neurons in the ventral tegmental area during paradoxical sleep. *Neuropsychopharmacology*, 32(6), 1232-1241.
- Dang-Vu, T. T., Desseilles, M., Laureys, S., Degueldre, C., Perrin, F., Phillips, C., . . . Peigneux, P. (2005). Cerebral correlates of delta waves during non-REM sleep revisited. *Neuroimage*, 28(1), 14-21.
- Dang-Vu, T. T., Schabus, M., Desseilles, M., Albouy, G., Boly, M., Darsaud, A., . . . Vandewalle, G. (2008). Spontaneous neural activity during human slow wave sleep. *Proceedings of the National Academy of Sciences*, 105(39), 15160-15165.
- Davis, C., Strachan, S., & Berkson, M. (2004). Sensitivity to reward: Implications for overeating and overweight. *Appetite*, 42(2), 131-138.
- De Gennaro, L., Marzano, C., Moroni, F., Curcio, G., Ferrara, M., & Cipolli, C. (2010). Recovery sleep after sleep deprivation almost completely abolishes dream recall. *Behavioural Brain Research*, 206(2), 293-298.

- Della Marca, G., Farina, B., Mennuni, G., Mazza, S., Di Giannantonio, M., Spadini, V., . . .
 Mazza, M. (2004). Microstructure of sleep in eating disorders: Preliminary results. *Eat Weight Disord*, 9(1), 77-80.
- Dement, W. (1965). An essay on dreams: The role of physiology in understanding their nature. *New Directions in Psychology II*, , 135-257.
- Dement, W., & Kleitman, N. (1957). Cyclic variations in EEG during sleep and their relation to eye movements, body motility, and dreaming. *Electroencephalography and Clinical Neurophysiology*, 9(4), 673-690.
- Dement, W., & Wolpert, E. A. (1958). The relation of eye movements, body motility, and external stimuli to dream content. *Journal of Experimental Psychology*, 55(6), 543.
- Destexhe, A., Hughes, S. W., Rudolph, M., & Crunelli, V. (2007). Are corticothalamic 'up' states fragments of wakefulness? *Trends in Neurosciences*, 30(7), 334-342.
- Dijk, D., Duffy, J. F., & Czeisler, C. A. (2001). Age-related increase in awakenings: Impaired consolidation of nonREM sleep at all circadian phases. *Sleep*, 24(5), 565.
- Domínguez-Borràs, J., Garcia-Garcia, M., & Escera, C. (2008). Emotional context enhances auditory novelty processing: Behavioural and electrophysiological evidence. *European Journal of Neuroscience*, 28(6), 1199-1206.
- Domino, E. F., & Yamamoto, K. (1965). Nicotine: Effect on the sleep cycle of the cat. *Science*, 150(3696), 637-638.
- Dreher, J., Kohn, P., Kolachana, B., Weinberger, D. R., & Berman, K. F. (2009). Variation in dopamine genes influences responsivity of the human reward system. *Proceedings of the National Academy of Sciences*, 106(2), 617-622.
- Economo, C. (1930). Sleep as a problem of localization. *The Journal of Nervous and Mental Disease*, 71(3), 249-259.
- Ehrhart, J., Ehrhart, M., Muzet, A., Schieber, J., & Naitoh, P. (1980). K-complexes and sleep spindles before transient activation during sleep. *Sleep*, 4(4), 400-407.
- Eichenlaub, J., Bertrand, O., Morlet, D., & Ruby, P. (2013). Brain reactivity differentiates subjects with high and low dream recall frequencies during both sleep and wakefulness. *Cerebral Cortex*,
- Eichenlaub, J., Ruby, P., & Morlet, D. (2012). What is the specificity of the response to the own first-name when presented as a novel in a passive oddball paradigm? an ERP study. *Brain Research*, 1447, 65-78.
- Ermis, U., Krakow, K., & Voss, U. (2010). Arousal thresholds during human tonic and phasic REM sleep. *Journal of Sleep Research*, 19(3), 400-406.

- Esposito, M. J., Nielsen, T. A., & Paquette, T. (2004). Reduced alpha power associated with the recall of mentation from stage 2 and stage REM sleep. *Psychophysiology*, *41*(2), 288-297.
- Esser, S. K., Hill, S., & Tononi, G. (2009). Breakdown of effective connectivity during slow wave sleep: Investigating the mechanism underlying a cortical gate using large-scale modeling. *Journal of Neurophysiology*, *102*(4), 2096-2111.
- Farina, B., Della Marca, G., Grochocinski, V. J., Mazza, M., Buysse, D. J., Di Giannantonio, M.,...Frank, E. (2003). Microstructure of sleep in depressed patients according to the cyclic alternating pattern. *Journal of Affective Disorders*, *77*(3), 227-235.
- Fein, G., Feinberg, I., Insel, T. R., Antrobus, J. S., Price, L. J., Floyd, T. C., & Nelson, M. A. (1985). Sleep mentation in the elderly. *Psychophysiology*, *22*(2), 218-225.
- Ferri, R., Bruni, O., Miano, S., & Terzano, M. G. (2005). Topographic mapping of the spectral components of the cyclic alternating pattern (CAP). *Sleep Medicine*, *6*(1), 29-36.
- Ferri, R., Cosentino, F. I., Elia, M., Musumeci, S. A., Marinig, R., & Bergonzi, P. (2001). Relationship between delta, sigma, beta, and gamma EEG bands at REM sleep onset and REM sleep end. *Clinical Neurophysiology*, *112*(11), 2046-2052.
- Ferri, R., Huber, R., Aricò, D., Drago, V., Rundo, F., Ghilardi, M. F.,...Tononi, G. (2008). The slow-wave components of the cyclic alternating pattern (CAP) have a role in sleep-related learning processes. *Neuroscience Letters*, *432*(3), 228-231.
- Field, A. (2009). *Discovering statistics using SPSS* Sage publications.
- Figuerero, A. J., Ross, D. M., & Petrinovich, L. (1992). The quantitative ethology of the zebra finch: A study in comparative psychometrics. *Multivariate Behavioral Research*, *27*(3), 435-458.
- Fisher, C., Byrne, J., Edwards, A., & Kahn, E. (1970). A psychophysiological study of nightmares. *Journal of the American Psychoanalytic Association*,
- Fornal, C., Auerbach, S., & Jacobs, B. L. (1985). Activity of serotonin-containing neurons in nucleus raphe magnus in freely moving cats. *Experimental Neurology*, *88*(3), 590-608.
- Fosse, R., Stickgold, R., & Hobson, J. A. (2004). Thinking and hallucinating: Reciprocal changes in sleep. *Psychophysiology*, *41*(2), 298-305.
- Foulkes, D. (1967). Nonrapid eye movement mentation. *Experimental Neurology*, *19*, 28-38.
- Foulkes, D., Larson, J. D., Swanson, E. M., & Rardin, M. (1969). Two studies of childhood dreaming. *American Journal of Orthopsychiatry*, *39*(4), 627-643.

- Foulkes, D., & Pope, R. (1973). Primary visual experience and secondary cognitive elaboration in stage REM: A modest confirmation and an extension. *Perceptual and Motor Skills*, 37(1), 107-118.
- Foulkes, D., Spear, P. S., & Symonds, J. D. (1966). Individual differences in mental activity at sleep onset. *Journal of Abnormal Psychology*, 71(4), 280.
- Foulkes, D., & Vogel, G. (1965). Mental activity at sleep onset. *Journal of Abnormal Psychology*, 70(4), 231.
- Foulkes, W. D. (1962). Dream reports from different stages of sleep. *The Journal of Abnormal and Social Psychology*, 65(1), 14.
- Freud, S. (1953/2006). *Interpreting Dreams* (J. A. Underwood, Trans.). United Kingdom, UK: Penguin.
- Gadea-Ciria, M., Stabler, H., Lloyd, K., & Bartholini, G. (1973). Acetylcholine release within the cat striatum during the Sleep–Wakefulness cycle. *Nature*, 243(5409), 518-519.
- Goodenough, D. R., Lewis, H. B., Shapiro, A., & Sleser, I. (1965). Some correlates of dream reporting following laboratory awakenings. *The Journal of Nervous and Mental Disease*, 140(5), 365-373.
- Gottesmann, C. (2004). Brain inhibitory mechanisms involved in basic and higher integrated sleep processes. *Brain Research Reviews*, 45(3), 230-249.
- Grosser, G. S., & Siegal, A. W. (1971). Emergence of a tonic-phasic model for sleep and dreaming: Behavioral and physiological observations. *Psychological Bulletin*, 75(1), 60.
- Guazzelli, M., Feinberg, I., Aminoff, M., Fein, G., Floyd, T., & Maggini, C. (1986). Sleep spindles in normal elderly: Comparison with young adult patterns and relation to nocturnal awakening, cognitive function and brain atrophy. *Electroencephalography and Clinical Neurophysiology*, 63(6), 526-539.
- Guilleminault, C., Kirisoglu, C., da Rosa, A. C., Lopes, C., & Chan, A. (2006). Sleepwalking, a disorder of NREM sleep instability. *Sleep Medicine*, 7(2), 163-170.
- Guilleminault, C., Lee, J. H., Chan, A., Lopes, M., Huang, Y., & da Rosa, A. (2005). Non-REM-sleep instability in recurrent sleepwalking in pre-pubertal children. *Sleep Medicine*, 6(6), 515-521.
- Guilleminault, C. (2006). Hypersynchronous slow delta, cyclic alternating pattern and sleepwalking. *Sleep*, 29(1), 14-15.
- Haimov, I., & Lavie, P. (1997). Circadian characteristics of sleep propensity function in healthy elderly: A comparison with young adults. *Sleep*, 20(4), 294-300.

- Hair, J., Black, W., & Babin, B. Anderson, re, & tatham, r. L.(2006). *Multivariate Data Analysis*,
- Halász, P. (1982). The role of the nonspecific phasic activation in the sleep regulation and in the mechanism of generalised epilepsy with spike-wave pattern. *Academic Doctoral Thesis, Semmelweis University, Budapest*,
- Halász, P. (1993). Arousals without awakening—dynamic aspect of sleep. *Physiology & Behavior*, 54(4), 795-802.
- Halász, P. (1998). Hierarchy of micro-arousals and the microstructure of sleep. *Neurophysiologie Clinique/Clinical Neurophysiology*, 28(6), 461-475.
- Halász, P. (2005). K-complex, a reactive EEG graphoelement of NREM sleep: An old chap in a new garment. *Sleep Medicine Reviews*, 9(5), 391-412.
- Halász, P., & Bódizs, R. (2013). *Dynamic structure of NREM sleep* Springer.
- Halász, P., Terzano, M., Parrino, L., & Bódizs, R. (2004). The nature of arousal in sleep. *Journal of Sleep Research*, 13(1), 1-23.
- Halász, P., Kundra, O., Rajna, P., Pal, I., & Vargha, M. (1979). Micro-arousals during nocturnal sleep. *Acta Physiologica Academiae Scientiarum Hungaricae*, 54(1), 1-12.
- Hall, C. S., & Van de Castle, Robert L. (1966). The content analysis of dreams.
- Hartmann, E., Russ, D., Oldfield, M., Falke, R., & Skoff, B. (1980). Dream content: Effects of L-DOPA. *Sleep Research*, 9(1.53)
- Hauri, P., Sawyer, J., & Rechtschaffen, A. (1967). Dimensions of dreaming: A factored scale for rating dream reports. *Journal of Abnormal Psychology*, 72(1), 16.
- Heiser, C., Baja, J., Lenz, F., Sommer, J., Hörmann, K., Herr, R., & Stuck, B. (2012). Effects of an artificial smoke on arousals during human sleep. *Chemosensory Perception*, 5(3-4), 274-279.
- Hernández-Peón, R. (1967). Neurophysiology, phylogeny, and functional significance of dreaming. *Experimental Neurology*, 19, 106-141.
- Hirshkowitz, M. (2002). Arousals and anti-arousals. *Sleep Medicine*, 3(3), 203-204.
- Hobson, J. A., Pace-Schott, E. F., & Stickgold, R. (2000). Dreaming and the brain: Toward a cognitive neuroscience of conscious states. *Behavioral and Brain Sciences*, 23(6), 793-842.
- Hobson, J.A., & McCarley, R.W. (1977). The brain as a dream state generator: An activation-synthesis hypothesis of the dream process. *Am J Psychiatry*, 134(12)

- Hodes, R., & Dement, W. C. (1964). Depression of electrically induced reflexes (“H-reflexes”) in man during low voltage EEG “sleep”. *Electroencephalography and Clinical Neurophysiology*, 17(6), 617-629.
- Hoelscher, T. J., Klinger, E., & Barta, S. G. (1981). Incorporation of concern-and nonconcern-related verbal stimuli into dream content. *Journal of Abnormal Psychology*, 90(1), 88.
- Holeckova, I., Fischer, C., Giard, M., Delpuech, C., & Morlet, D. (2006). Brain responses to a subject's own name uttered by a familiar voice. *Brain Research*, 1082(1), 142-152.
- Horne, J. (2009). REM sleep, energy balance and ‘optimal foraging’. *Neuroscience & Biobehavioral Reviews*, 33(3), 466-474.
- Horne, J. (2012). Why REM sleep? clues beyond the laboratory in a more challenging world. *Biological Psychology*,
- Huber, R., Ghilardi, M. F., Massimini, M., & Tononi, G. (2004). Local sleep and learning. *Nature*, 430(6995), 78-81.
- Hutchison, K. E., Wood, M. D., & Swift, R. (1999). Personality factors moderate subjective and psychophysiological responses to d-amphetamine in humans. *Experimental and Clinical Psychopharmacology*, 7(4), 493.
- Jahnke, K., von Wegner, F., Morzelewski, A., Borisov, S., Maischein, M., Steinmetz, H., & Laufs, H. (2012). To wake or not to wake? the two-sided nature of the human K-complex. *Neuroimage*, 59(2), 1631-1638.
- Jakobson, A. J., Conduit, R., & Fitzgerald, P. B. (2012). Investigation of visual dream reports after transcranial direct current stimulation (tDCS) during REM sleep. *International Journal of Dream Research*, 5(1), 87-93.
- Jakobson, A. J., Fitzgerald, P. B., & Conduit, R. (2012). Induction of visual dream reports after transcranial direct current stimulation (tDCs) during stage 2 sleep. *Journal of Sleep Research*, 21(4), 369-379.
- Johnson, L. C., & Lubin, A. (1967). The orienting reflex during waking and sleeping. *Electroencephalography and Clinical Neurophysiology*, 22(1), 11-21.
- Johnson, P. M., & Kenny, P. J. (2010). Dopamine D2 receptors in addiction-like reward dysfunction and compulsive eating in obese rats. *Nature Neuroscience*, 13(5), 635-641.
- Jones, B. E. (2003). Arousal systems. *Frontiers in Bioscience*, 8(5), 438-451.
- Jones, B. E., Harper, S. T., & Halaris, A. E. (1977). Effects of locus coeruleus lesions upon cerebral monoamine content, sleep-wakefulness states and the response to amphetamine in the cat. *Brain Research*, 124(3), 473-496.

- Jouvet, M., Michel, F., & Courjon, J. (1959). On a stage of rapid cerebral electrical activity in the course of physiological sleep. *Comptes Rendus Des Seances De La Societe De Biologie Et De Ses Filiales*, *153*, 1024-1028.
- Jus, A., Jus, K., Villeneuve, A., Pires, A., Lachance, R., Fortier, J., & Villeneuve, R. (1973). Studies on dream recall in chronic schizophrenic patients after prefrontal lobotomy. *Biological Psychiatry*,
- Kaasinen, V., Vilkmann, H., Hietala, J., Någren, K., Helenius, H., Olsson, H., . . . Rinne, J. O. (2000). Age-related dopamine D2/D3 receptor loss in extrastriatal regions of the human brain. *Neurobiology of Aging*, *21*(5), 683-688.
- Kamiya, J. (1961). Behavioral, subjective, and physiological aspects of drowsiness and sleep. *Functions of Varied Experience*, , 145-174.
- Karacan, I., Goodenough, D., Shapiro, A., & Starker, S. (1966). Ereption cycle during sleep in relation to dream anxiety. *Archives of General Psychiatry*, *15*(2), 183.
- Kato, T., Rompre, P., Montplaisir, J., Sessle, B., & Lavigne, G. (2001). Sleep bruxism: An oromotor activity secondary to micro-arousal. *Journal of Dental Research*, *80*(10), 1940-1944.
- Kissler, J., Herbert, C., Winkler, I., & Junghofer, M. (2009). Emotion and attention in visual word processing—An ERP study. *Biological Psychology*, *80*(1), 75-83.
- Koulack, D. (1969). Effects of somatosensory stimulation on dream content. *Archives of General Psychiatry*, *20*(6), 718.
- Koulack, D., & Goodenough, D. R. (1976). Dream recall and dream recall failure: An arousal-retrieval model. *Psychological Bulletin*, *83*(5), 975.
- Krebs, R. M., Schott, B. H., & Düzel, E. (2009). Personality traits are differentially associated with patterns of reward and novelty processing in the human substantia nigra/ventral tegmental area. *Biological Psychiatry*, *65*(2), 103-110.
- Lansink, C. S., Goltstein, P. M., Lankelma, J. V., McNaughton, B. L., & Pennartz, C. M. (2009). Hippocampus leads ventral striatum in replay of place-reward information. *PLoS Biology*, *7*(8), e1000173.
- Lena, I., Parrot, S., Deschaux, O., Muffat-Joly, S., Sauvinet, V., Renaud, B., . . . Gottesmann, C. (2005). Variations in extracellular levels of dopamine, noradrenaline, glutamate, and aspartate across the sleep–wake cycle in the medial prefrontal cortex and nucleus accumbens of freely moving rats. *Journal of Neuroscience Research*, *81*(6), 891-899.
- Llinás, R. R., & Paré, D. (1991). Of dreaming and wakefulness. *Neuroscience*, *44*(3), 521-535.

- Llinás, R. R., & Steriade, M. (2006). Bursting of thalamic neurons and states of vigilance. *Journal of Neurophysiology*, *95*(6), 3297-3308.
- Lovallo, W. R., Farag, N. H., Vincent, A. S., Thomas, T. L., & Wilson, M. F. (2006). Cortisol responses to mental stress, exercise, and meals following caffeine intake in men and women. *Pharmacology Biochemistry and Behavior*, *83*(3), 441-447.
- Lu, J., Jhou, T. C., & Saper, C. B. (2006). Identification of wake-active dopaminergic neurons in the ventral periaqueductal gray matter. *The Journal of Neuroscience*, *26*(1), 193-202.
- Lu, J., Sherman, D., Devor, M., & Saper, C. B. (2006). A putative flip–flop switch for control of REM sleep. *Nature*, *441*(7093), 589-594.
- Macaluso, G., Guerra, P., Di Giovanni, G., Boselli, M., Parrino, L., & Terzano, M. (1998). Sleep bruxism is a disorder related to periodic arousals during sleep. *Journal of Dental Research*, *77*(4), 565-573.
- Maquet, P., Péters, J., Aerts, J., Delfiore, G., Degueldre, C., Luxen, A., & Franck, G. (1996). Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. *Nature*, *383*(6596), 163-166.
- Massaquoi, S., & McCarley, R. (1992). Extension of the limit cycle reciprocal interaction model of REM cycle control. an integrated sleep control model. *Journal of Sleep Research*, *1*(2), 138-143.
- Massimini, M., Tononi, G., & Huber, R. (2009). Slow waves, synaptic plasticity and information processing: Insights from transcranial magnetic stimulation and high-density EEG experiments. *European Journal of Neuroscience*, *29*(9), 1761-1770.
- Massimini, M., Ferrarelli, F., Huber, R., Esser, S. K., Singh, H., & Tononi, G. (2005). Breakdown of cortical effective connectivity during sleep. *Science*, *309*(5744), 2228-2232.
- Massimini, M., Huber, R., Ferrarelli, F., Hill, S., & Tononi, G. (2004). The sleep slow oscillation as a traveling wave. *The Journal of Neuroscience*, *24*(31), 6862-6870.
- McCarley, R. W., & Hobson, J. A. (1975). Neuronal excitability modulation over the sleep cycle: A structural and mathematical model. *Science*, *189*(4196), 58-60.
- Medoff, L., & Foulkes, D. (1971). Microscopic” studies of mentation in stage REM: A preliminary report. *11th Annual Meeting of the APSS, Bruges*,
- Mell, T., Wartenburger, I., Marschner, A., Villringer, A., Reischies, F. M., & Heekeren, H. R. (2009). Altered function of ventral striatum during reward-based decision making in old age. *Frontiers in Human Neuroscience*, *3*

- Mena-Segovia, J., & Bolam, J. P. (2011). Phasic modulation of cortical high-frequency oscillations by pedunculopontine neurons. *Progress in Brain Research*, 193, 85-92.
- Michel, F., Rechtschaffen, A., & Vimont-Vicary, P. (1963). *Activité électrique des muscles oculaires extrinsèques au cours du cycle veille-sommeil*
- Moerman, N., Bonke, B., & Oosting, J. (1993). Awareness and recall during general anesthesia: Facts and feelings. *Anesthesiology*, 79(3), 454-464.
- Molinari, S., & Foulkes, D. (1969). Tonic and phasic events during sleep: Psychological correlates and implications. *Perceptual and Motor Skills*,
- Monroe, L. J., Rechtschaffen, A., Foulkes, D., & Jensen, J. (1965). Discriminability of REM and NREM reports. *Journal of Personality and Social Psychology*, 2(3), 456.
- Moruzzi, G. (1963). Active processes in the brain stem during sleep. *Harvey Lectures*, 58, 233-297.
- Moruzzi, G. (1964). Reticular influences on the EEG. *Electroencephalography and Clinical Neurophysiology*, 16(1), 2-17.
- Moruzzi, G., & Magoun, H. W. (1949). Brain stem reticular formation and activation of the EEG. *Electroencephalography and Clinical Neurophysiology*, 1(1), 455-473.
- Mouret, J., & Coindet, J. (1980). Polygraphic evidence against a critical role of the raphe nuclei in sleep in the rat. *Brain Research*, 186(2), 273-287.
- Mullin, F. J., Kleitman, N., & Cooperman, N. (1937). Studies on the physiology of sleep changes in irritability to auditory stimuli during sleep. *Journal of Experimental Psychology*, 21(1), 88.
- Németh, G., Hegedüs, K., & Molnár, L. (1988). Akinetic mutism associated with bicingular lesions: Clinicopathological and functional anatomical correlates. *European Archives of Psychiatry and Neurological Sciences*, 237(4), 218-222.
- Netter, P., Hennig, J., & Roed, I. (1996). Serotonin and dopamine as mediators of sensation seeking behavior. *Neuropsychobiology*, 34(3), 155-165.
- Nielsen, T. (2010). Ultradian, circadian, and sleep-dependent features of dreaming. *Principles and Practice of Sleep Medicine*,
- Nielsen, T. A. (1993). Changes in the kinesthetic content of dreams following somatosensory stimulation of leg muscles during REM sleep. *Dreaming*, 3(2), 99.
- Nielsen, T. A. (2000). A review of mentation in REM and NREM sleep: "covert" REM sleep as a possible reconciliation of two opposing models. *Behavioral and Brain Sciences*, 23(6), 851-866.

- Nielsen, T. A. (2004). Chronobiological features of dream production. *Sleep Medicine Reviews*, 8(5), 403-424.
- Nieoullon, A. (2002). Dopamine and the regulation of cognition and attention. *Progress in Neurobiology*, 67(1), 53-83.
- Nir, Y., & Tononi, G. (2010). Dreaming and the brain: From phenomenology to neurophysiology. *Trends in Cognitive Sciences*, 14(2), 88-100.
- Nobili, L., Ferrara, M., Moroni, F., De Gennaro, L., Russo, G. L., Campus, C., . . . De Carli, F. (2011). Dissociated wake-like and sleep-like electro-cortical activity during sleep. *Neuroimage*, 58(2), 612-619.
- Nobili, L., Sartori, I., Terzaghi, M., Stefano, F., Mai, R., Tassi, L., . . . Russo, G. L. (2006). Relationship of epileptic discharges to arousal instability and periodic leg movements in a case of nocturnal frontal lobe epilepsy: A stereo-EEG study. *Sleep-New York then Westchester-*, 29(5), 701.
- Nofzinger, E. A. (2005). Neuroimaging and sleep medicine. *Sleep Medicine Reviews*, 9(3), 157-172.
- Nofzinger, E. A., Mintun, M. A., Wiseman, M., Kupfer, D. J., & Moore, R. Y. (1997). Forebrain activation in REM sleep: An FDG PET study. *Brain Research*, 770(1), 192-201.
- Ogilvie, R., Hunt, H., Sawicki, C., & Samahalskyi, J. (1981). Psychological correlates of spontaneous middle ear muscle activity during sleep. *Sleep*, 5(1), 11-27.
- Oswald, I., Taylor, A. M., & Treisman, M. (1960). Discriminative responses to stimulation during human sleep. *Brain: A Journal of Neurology*,
- Oudiette, D., Dealberto, M., Ugucioni, G., Golmard, J., Merino-Andreu, M., Tafti, M., . . . Arnulf, I. (2012). Dreaming without REM sleep. *Consciousness and Cognition*, 21(3), 1129-1140.
- Pagel, J. F. (2005). Medications and their effects on sleep. *Primary Care: Clinics in Office Practice*, 32(2), 491-509.
- Pagel, J., & Shocknesse, S. (2007). Dreaming and insomnia: Polysomnographic correlates of reported dream recall frequency. *Dreaming*, 17(3), 140.
- Pan, W., & Hyland, B. I. (2005). Pedunculopontine tegmental nucleus controls conditioned responses of midbrain dopamine neurons in behaving rats. *The Journal of Neuroscience*, 25(19), 4725-4732.
- Panksepp, J. (1998). *Affective neuroscience: The foundations of human and animal emotions* Oxford University Press.

- Park, J. h., An, H., Jang, E. s., & Chung, S. (2012). The influence of personality and dysfunctional sleep-related cognitions on the severity of insomnia. *Psychiatry Research, 197*(3), 275-279.
- Parrino, L., Boselli, M., Buccino, G. P., Spaggiari, M. C., Di Giovanni, G., & Terzano, M. G. (1996). The cyclic alternating pattern plays a gate-control on periodic limb movements during non-rapid eye movement sleep. *Journal of Clinical Neurophysiology, 13*(4), 314-323.
- Parrino, L., Boselli, M., Spaggiari, M. C., Smerieri, A., & Terzano, M. G. (1998). Cyclic alternating pattern (CAP) in normal sleep: Polysomnographic parameters in different age groups. *Electroencephalography and Clinical Neurophysiology, 107*(6), 439-450.
- Parrino, L., Ferri, R., Bruni, O., & Terzano, M. G. (2012). Cyclic alternating pattern (CAP): The marker of sleep instability. *Sleep Medicine Reviews, 16*(1), 27-45.
- Parrino, L., Ferri, R., Zucconi, M., & Fanfulla, F. (2009). Commentary from the Italian association of sleep medicine on the AASM manual for the scoring of sleep and associated events: For debate and discussion. *Sleep Medicine, 10*(7), 799-808.
- Parrino, L., Ferrillo, F., Smerieri, A., Spaggiari, M. C., Palomba, V., Rossi, M., & Terzano, M. G. (2004). Is insomnia a neurophysiological disorder? the role of sleep EEG microstructure. *Brain Research Bulletin, 63*(5), 377-383.
- Parrino, L., Milioli, G., De Paolis, F., Grassi, A., & Terzano, M. G. (2009). Paradoxical insomnia: The role of CAP and arousals in sleep misperception. *Sleep Medicine, 10*(10), 1139-1145.
- Parrino, L., Smerieri, A., Rossi, M., & Terzano, M. G. (2001). Relationship of slow and rapid EEG components of CAP to ASDA arousals in normal sleep. *Sleep, 24*(8), 881-885.
- Paus, T. (2001). Primate anterior cingulate cortex: Where motor control, drive and cognition interface. *Nature Reviews Neuroscience, 2*(6), 417-424.
- Perogamvros, L., Baud, P., Hasler, R., Cloninger, C. R., Schwartz, S., & Perrig, S. (2012). Active reward processing during human sleep: Insights from sleep-related eating disorder. *Frontiers in Neurology, 3*
- Perogamvros, L., & Schwartz, S. (2012). The roles of the reward system in sleep and dreaming. *Neuroscience & Biobehavioral Reviews, 36*(8), 1934-1951.
- Perrin, F., Bastuji, H., Mauguière, F., & García-Larrea, L. (2000). Functional dissociation of the early and late portions of human K-complexes. *Neuroreport, 11*(8), 1637-1640.

- Perrin, F., Garcia-Larrea, L., Mauguière, F., & Bastuji, H. (1999). A differential brain response to the subject's own name persists during sleep. *Clinical Neurophysiology*, *110*(12), 2153-2164.
- Pivik, R. T. (1970). *Mental Activity and Phasic Events during Sleep*. (Unpublished Doctoral thesis, Department of Psychology, Stanford University).
- Pivik, R. T. (1991). Tonic states and phasic events in relation to sleep mentation. In A. M., Arkin, J. S. Antrobus, & S. J. Ellman (Eds.). *The mind in sleep: Psychology and psychophysiology*. England, UK: Lawrence Erlbaum.
- Pivik, R. T., & Dement, W. (1970). Phasic changes in muscular and reflex activity during non-REM sleep. *Experimental Neurology*, *27*(1), 115-124.
- Pivik, R. T., & Foulkes, D. (1968). NREM mentation: Relation to personality, orientation time, and time of night. *Journal of Consulting and Clinical Psychology*, *32*(2), 144-151.
- Pivik, R. T., Halper, C., & Dement, W. (1969). Phasic events and mentation during sleep. *Meeting of the Association for the Psychophysiological Study of Sleep, Boston, Mass*.
- Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology*, *118*(10), 2128-2148.
- Portas, C. M., Krakow, K., Allen, P., Josephs, O., Armony, J. L., & Frith, C. D. (2000). Auditory processing across the sleep-wake cycle: Simultaneous EEG and fMRI monitoring in humans. *Neuron*, *28*(3), 991-999.
- Poza, J. J., & Martí Massó, J. F. (2006). Total dream loss secondary to left temporo-occipital brain injury. [Perdida completa de ensonaciones tras una lesión cerebral temporooccipital izquierda] *Neurologia (Barcelona, Spain)*, *21*(3), 152-154.
- Rama, A. N., Charles Cho, S., & Kushida, C. A. (2005). NREM-REM sleep. *Handbook of Clinical Neurophysiology*, *6*, 21-29.
- Ranson, S. (1939). Somnolence caused by hypothalamic lesions in the monkey. *Archives of Neurology and Psychiatry*, *41*(1), 1.
- Rattenborg, N. C., Lima, S. L., & Lesku, J. A. (2012). Sleep locally, act globally. *The Neuroscientist*, *18*(5), 533-546.
- Rechtschaffen, A. (1973). The psychophysiology of mental activity during sleep. *The Psychophysiology of Thinking: Studies of Covert Processes*, , 153-205.
- Rechtschaffen, A., Michel, F., & Metz, J. (1972). Relationship between extraocular and PGO activity in the cat. *Psychophysiology*, *9*, 128.
- Rechtschaffen, A., & Siegel, J. (2000). Sleep and dreaming. *Principles of Neuroscience*, *4*, 936-947.

- Rechtschaffen, A., Hauri, P., & Zeitlin, M. (1966). Auditory awakening thresholds in REM and NREM sleep stages. *Perceptual and Motor Skills*, 22(3), 927-942.
- Rechtschaffen, A., & Kales, A. (1968). A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects.
- Rechtschaffen, A., Verdone, P., & Wheaton, J. (1963). Reports of mental activity during sleep. *Canadian Psychiatric Association Journal*, 257, 409-414.
- Rosenblatt, S., Antrobus, J., Zimler, J., Antrobus, J., & Bertini, M. (1992). The effect of postawakening differences in activation on the REM-NREM report effect and recall information from films. *The Neuropsychology of Sleep and Dreaming*, , 215-224.
- Rosenlicht, N., Maloney, T., & Feinberg, I. (1994). Dream report length is more dependent on arousal level than prior REM duration. *Brain Research Bulletin*, 34(2), 99-101.
- Rotenberg, V. S. (1993). REM sleep and dreams as mechanisms of the recovery of search activity. *The Functions of Dreaming*, , 261-292.
- Sakai, K., Crochet, S., & Onoe, H. (2001). Pontine structures and mechanisms involved in the generation of paradoxical (REM) sleep. *Archives Italiennes De Biologie*, 139(1), 93-107.
- Sallinen, M., Kaartinen, J., & Lyytinen, H. (1996). Processing of auditory stimuli during tonic and phasic periods of REM sleep as revealed by event-related brain potentials. *Journal of Sleep Research*, 5(4), 220-228.
- Salzarulo, P., Fagioli, I., Lombardo, P., Gori, S., Gneri, C., Chiaramonti, R., & Murri, L. (1999). Sleep stages preceding spontaneous awakenings in the elderly. *Sleep Research Online : SRO*, 2(3), 73-77.
- Sanders, R. D., Tononi, G., Laureys, S., & Sleigh, J. (2012). Unresponsiveness≠ unconsciousness. *Anesthesiology*, 116(4), 946.
- Sandyk, R. (1997). Treatment with weak electromagnetic fields restores dream recall in a parkinsonian patient. *International Journal of Neuroscience*, 90(1-2), 75-86.
- Sanford, L., Silvestri, A., Ross, R., & Morrison, A. (2001). Influence of fear conditioning on elicited ponto-geniculo-occipital waves and rapid eye movement sleep. *Archives Italiennes De Biologie*, 139(3), 169-183.
- Saper, C. B. (1987). Diffuse cortical projection systems: Anatomical organization and role in cortical function. *Comprehensive Physiology*,
- Saper, C. B., Chou, T. C., & Scammell, T. E. (2001). The sleep switch: Hypothalamic control of sleep and wakefulness. *Trends in Neurosciences*, 24(12), 726-731.
- Saper, C. B., Fuller, P. M., Pedersen, N. P., Lu, J., & Scammell, T. E. (2010). Sleep state switching. *Neuron*, 68(6), 1023-1042.

- Saper, C. B., Scammell, T. E., & Lu, J. (2005). Hypothalamic regulation of sleep and circadian rhythms. *Nature*, *437*(7063), 1257-1263.
- Sastre, J., Buda, C., Kitahama, K., & Jouvet, M. (1996). Importance of the ventrolateral region of the periaqueductal gray and adjacent tegmentum in the control of paradoxical sleep as studied by muscimol microinjections in the cat. *Neuroscience*, *74*(2), 415-426.
- Schieber, J. P., Muzet, A., & Ferriere, P. J. (1971). Phases of spontaneous transitory activation during normal sleep in humans. [Les phases d'activation transitoire spontanees au cours du sommeil normal chez l'homme] *Archives Des Sciences Physiologiques*, *25*(4), 443-465.
- Schredl, M., Atanasova, D., Hoermann, K., Maurer, J. T., Hummel, T., & Stuck, B. A. (2009). Information processing during sleep: The effect of olfactory stimuli on dream content and dream emotions. *Journal of Sleep Research*, *18*(3), 285-290.
- Schredl, M., & Doll, E. (1998). Emotions in diary dreams. *Consciousness and Cognition*, *7*(4), 634-646.
- Schredl, M., Schäfer, G., Weber, B., & Heuser, I. (1998). Dreaming and insomnia: Dream recall and dream content of patients with insomnia. *Journal of Sleep Research*, *7*(3), 191-198.
- Schredl, M., Schmitt, J., Hein, G., Schmoll, T., Eller, S., & Haaf, J. (2006). Nightmares and oxygen desaturations: Is sleep apnea related to heightened nightmare frequency? *Sleep and Breathing*, *10*(4), 203-209.
- Schredl, M., Wittmann, L., Ciric, P., & Götz, S. (2003). Factors of home dream recall: A structural equation model. *Journal of Sleep Research*, *12*(2), 133-141.
- Schrouff, J., Perlberg, V., Boly, M., Marrelec, G., Boveroux, P., Vanhaudenhuyse, A., . . . Péligrini-Issac, M. (2011). Brain functional integration decreases during propofol-induced loss of consciousness. *Neuroimage*, *57*(1), 198-205.
- Schweinhart, P., Seminowicz, D. A., Jaeger, E., Duncan, G. H., & Bushnell, M. C. (2009). The anatomy of the mesolimbic reward system: A link between personality and the placebo analgesic response. *The Journal of Neuroscience*, *29*(15), 4882-4887.
- Sforza, E., Chapotot, F., Pigeau, R., Paul, P. N., & Buguet, A. (2004). Effects of sleep deprivation on spontaneous arousals in humans. *Sleep-New York then Westchester-*, *27*, 1068-1076.
- Shapiro, A., Goodenough, D. R., & Gryler, R. B. (1963). Dream recall as a function of method of awakening. *Psychosomatic Medicine*, *25*(2), 174-180.

- Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., . . . Dunbar, G. C. (1998). The mini-international neuropsychiatric interview (MINI): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, *59*, 22-33.
- Sherin, J., Shiromani, P., McCarley, R., & Saper, C. (1996). Activation of ventrolateral preoptic neurons during sleep. *Science*, *271*(5246), 216-219.
- Shinohara, M., Mizushima, H., Hirano, M., Shioe, K., Nakazawa, M., Hiejima, Y., . . . Kanba, S. (2004). Eating disorders with binge-eating behaviour are associated with the allele of the 3'-UTR VNTR polymorphism of the dopamine transporter gene. *Journal of Psychiatry and Neuroscience*, *29*(2), 134.
- Shouse, M. N., & Siegel, J. M. (1992). Pontine regulation of REM sleep components in cats: Integrity of the pedunculopontine tegmentum (PPT) is important for phasic events but unnecessary for atonia during REM sleep. *Brain Research*, *571*(1), 50-63.
- Smerieri, A., Parrino, L., Agosti, M., Ferri, R., & Terzano, M. G. (2007). Cyclic alternating pattern sequences and non-cyclic alternating pattern periods in human sleep. *Clinical Neurophysiology*, *118*(10), 2305-2313.
- Snyder, F. (1966). Toward an evolutionary theory of dreaming. *The American Journal of Psychiatry*,
- Solms, M. (1997). *The neuropsychology of dreams: A clinico-anatomical study*. Lawrence Erlbaum Associates Publishers.
- Solms, M. (2000). Dreaming and REM sleep are controlled by different brain mechanisms. *Behavioral and Brain Sciences*, *23*(6), 843-850.
- Solms, M. (2001). The interpretation of dreams and the neurosciences. *Psychoanalysis and History*, *3*(1), 79-91.
- Solms, M. (2012). Are Freud's "erogenous zones" sources or objects of libidinal drive? *Neuropsychoanalysis: An Interdisciplinary Journal for Psychoanalysis and the Neurosciences*, *14*(1), 53-56.
- Solms, M., & Panksepp, J. (2012). The "Id" knows more than the "Ego" admits: Neuropsychoanalytic and primal consciousness perspectives on the interface between affective and cognitive neuroscience. *Brain Sciences*, *2*(2), 147-175.
- Solms, M., & Turnbull, O. (2002). *Brain and the inner world: An introduction to the neuroscience of the subjective experience* Other PressLlc.
- Spriggs, W. (2009). *Essentials of polysomnography* Jones & Bartlett Publishers.

- Steininger, T. L., Alam, M. N., Gong, H., Szymusiak, R., & McGinty, D. (1999). Sleep-waking discharge of neurons in the posterior lateral hypothalamus of the albino rat. *Brain Research*, *840*(1), 138-147.
- Steriade, M. (2000). Corticothalamic resonance, states of vigilance and mentation. *Neuroscience*, *101*(2), 243-276.
- Steriade, M. (2006). Grouping of brain rhythms in corticothalamic systems. *Neuroscience*, *137*(4), 1087-1106.
- Steriade, M., Nunez, A., & Amzica, F. (1993). A novel slow (< 1 Hz) oscillation of neocortical neurons in vivo: Depolarizing and hyperpolarizing components. *The Journal of Neuroscience*, *13*(8), 3252-3265.
- Steriade, M., & Llinás, R. R. (1988). The functional states of the thalamus and the associated neuronal interplay. *Physiological Reviews*, *68*(3), 649-742.
- Stevens, J. (2009). *Applied multivariate statistics for the social sciences* Taylor & Francis US.
- Strauss, E. H., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* Oxford University Press.
- Stuart, K., & Conduit, R. (2009). Auditory inhibition of rapid eye movements and dream recall from REM sleep. *Sleep*, *32*(3), 399.
- Takeuchi, T., Miyasita, A., Inugami, M., & Yamamoto, Y. (2001). Intrinsic dreams are not produced without REM sleep mechanisms: Evidence through elicitation of sleep onset REM periods. *Journal of Sleep Research*, *10*(1), 43-52.
- Takeuchi, T., Ogilvie, R. D., Murphy, T. I., & Ferrelli, A. V. (2003). EEG activities during elicited sleep onset REM and NREM periods reflect different mechanisms of dream generation. *Clinical Neurophysiology*, *114*(2), 210-220.
- Terzaghi, M., Sartori, I., Mai, R., Tassi, L., Francione, S., Cardinale, F., . . . Manni, R. (2008). Coupling of minor motor events and epileptiform discharges with arousal fluctuations in NFLE. *Epilepsia*, *49*(4), 670-676.
- Terzano, M. G., & Parrino, L. (1992). Evaluation of EEG cyclic alternating pattern during sleep in insomniacs and controls under placebo and acute treatment with zolpidem. *Sleep: Journal of Sleep Research & Sleep Medicine*,
- Terzano, M. G., & Parrino, L. (2000). Origin and significance of the cyclic alternating pattern (CAP): Review article. *Sleep Medicine Reviews*, *4*(1), 101-123.

- Terzano, M. G., Parrino, L., Anelli, S., Halasz, P., & Portera-Sánchez, A. (1989). Modulation of generalized Spike-and-Wave discharges during sleep by cyclic alternating pattern. *Epilepsia*, *30*(6), 772-781.
- Terzano, M. G., Parrino, L., Boselli, M., Smerieri, A., & Spaggiari, M. C. (2000). CAP components and EEG synchronization in the first 3 sleep cycles. *Clinical Neurophysiology*, *111*(2), 283-290.
- Terzano, M. G., Parrino, L., Boselli, M., Spaggiari, M. C., & Di Giovanni, G. (1996). Polysomnographic analysis of arousal responses in obstructive sleep apnea syndrome by means of the cyclic alternating pattern. *Journal of Clinical Neurophysiology*, *13*(2), 145-155.
- Terzano, M. G., Parrino, L., Fioriti, G., Orofiamma, B., & Depoortere, H. (1990). Modifications of sleep structure induced by increasing levels of acoustic perturbation in normal subjects. *Electroencephalography and Clinical Neurophysiology*, *76*(1), 29-38.
- Terzano, M. G., Parrino, L., Rosa, A., Palomba, V., & Smerieri, A. (2002). CAP and arousals in the structural development of sleep: An integrative perspective. *Sleep Medicine*, *3*(3), 221-229.
- Terzano, M. G., Parrino, L., Smerieri, A., Chervin, R., Chokroverty, S., Guilleminault, C., . . . Rosa, A. (2001). Atlas, rules, and recording techniques for the scoring of cyclic alternating pattern (CAP) in human sleep. *Sleep Medicine*, *2*(6), 537-553.
- Terzano, M. G., Parrino, L., Smerieri, A., Carli, F., Nobili, L., Donadio, S., & Ferrillo, F. (2005). CAP and arousals are involved in the homeostatic and ultradian sleep processes. *Journal of Sleep Research*, *14*(4), 359-368.
- Terzano, M. G., Parrino, L., & Spaggiari, M. C. (1988). The cyclic alternating pattern sequences in the dynamic organization of sleep. *Electroencephalography and Clinical Neurophysiology*, *69*(5), 437-447.
- Terzano, M., Mancina, D., Salati, M., Costani, G., Decembrino, A., & Parrino, L. (1985). The cyclic alternating pattern as a physiologic component of normal NREM sleep. *Sleep: Journal of Sleep Research & Sleep Medicine*,
- Terzano, M., & Parrino, L. (1991). Functional relationship between micro-and macrostructure of sleep. *Phasic Events and Dynamic Organization of Sleep*, 101-120. New York, NY: Raven Press.
- Thompson, D. F., & Pierce, D. R. (1999). Drug-induced nightmares. *The Annals of Pharmacotherapy*, *33*(1), 93-98.

- Tononi, G. (2004). An information integration theory of consciousness. *BMC Neuroscience*, 5(1), 42.
- Tononi, G., & Sporns, O. (2003). Measuring information integration. *BMC Neuroscience*, 4(1), 31.
- Tracy, R. L., & Tracy, L. N. (1974). Reports of mental activity from sleep stages 2 and 4. *Perceptual and Motor Skills*, 38(2), 647-648.
- Volkow, N. D., Fowler, J. S., Wang, G., & Goldstein, R. Z. (2002). Role of dopamine, the frontal cortex and memory circuits in drug addiction: Insight from imaging studies. *Neurobiology of Learning and Memory*, 78(3), 610-624.
- Volkow, N. D., Logan, J., Fowler, J. S., Wang, G., Gur, R. C., Wong, C., . . . Hitzemann, R. (2000). Association between age-related decline in brain dopamine activity and impairment in frontal and cingulate metabolism. *American Journal of Psychiatry*, 157(1), 75-80.
- Vyazovskiy, V. V., Olcese, U., Hanlon, E. C., Nir, Y., Cirelli, C., & Tononi, G. (2011). Local sleep in awake rats. *Nature*, 472(7344), 443-447.
- Vyazovskiy, V. V., Olcese, U., Lazimy, Y. M., Faraguna, U., Esser, S. K., Williams, J. C., . . . Tononi, G. (2009). Cortical firing and sleep homeostasis. *Neuron*, 63(6), 865-878.
- Wamsley, E. J., Hirota, Y., Tucker, M. A., Smith, M. R., & Antrobus, J. S. (2007). Circadian and ultradian influences on dreaming: A dual rhythm model. *Brain Research Bulletin*, 71(4), 347-354.
- Wamsley, E., & Antrobus, J. S. (2008). Homeostatic and circadian influences on dreaming: NREM mentation during a short daytime nap. *International Journal of Dream Research*, 1(2), 27-33.
- Watson, R. (1972). Mental correlates of periorbital PIPs during REM sleep. *Sleep Res*, 1, 116.
- Watson, R., & Rechtschaffen, A. (1969). Auditory awakening thresholds and dream recall in NREM sleep. *Perceptual and Motor Skills*, 29(2), 635-644.
- Wauquier, A., Aloe, L., & Declerck, A. (1995). K-complexes: Are they signs of arousal or sleep protective? *Journal of Sleep Research*, 4(3), 138-143.
- Webb, W., & Agnew Jr, H. (1978). Sleep onset facilitation by tones. *Sleep*, 1(3), 281-286.
- Wechsler, D. (1999). *Wechsler abbreviated scale of intelligence* Psychological Corporation.
- Wehrle, R., Kaufmann, C., Wetter, T. C., Holsboer, F., Auer, D. P., Pollmächer, T., & Czisch, M. (2007). Functional microstates within human REM sleep: First evidence from

- fMRI of a thalamocortical network specific for phasic REM periods. *European Journal of Neuroscience*, 25(3), 863-871.
- Wei, H. G., Riel, E., Czeisler, C. A., & Dijk, D. (1999). Attenuated amplitude of circadian and sleep-dependent modulation of electroencephalographic sleep spindle characteristics in elderly human subjects. *Neuroscience Letters*, 260(1), 29-32.
- Weisz, R., & Foulkes, D. (1970). Home and laboratory dreams collected under uniform sampling conditions. *Psychophysiology*, 6(5), 588-596.
- Winget, C. N., & Kramer, M. (1979). *Dimensions of dreams* University Presses of Florida Gainesville.
- Wollman, M. C., & Antrobus, J. S. (1987). Cortical arousal and mentation in sleeping and waking subjects. *Brain and Cognition*, 6(3), 334-346.
- Yu, C. K. (2001a). Neuroanatomical correleates of dreaming. II: The ventromesial frontal region controversy (dream instigation). *Neuro-Psychoanalysis*,
- Yu, C. K. (2001b). Neuroanatomical correlates of dreaming: The supramarginal gyrus controversy (dream work). *Neuro-Psychoanalysis*,
- Zimmerman, W. B. (1970). Sleep mentation and auditory awakening thresholds. *Psychophysiology*, 6(5), 540-549.
- Zucconi, M., Oldani, A., Ferini-Strambi, L., & Smirne, S. (1995). Arousal fluctuations in non-rapid eye movement parasomnias: The role of cyclic alternating pattern as a measure of sleep instability. *Journal of Clinical Neurophysiology*, 12(2), 147-154.

APPENDIX A

Scoring Criteria for CAP and Non-CAP

All criteria for the scoring of CAP and non-CAP (NCAP) have been derived from Terzano and Parrino (2000), and Terzano et al. (2001).

CAP and non-CAP during non-REM sleep

To score a CAP sequence two or more CAP *cycles* need to have occurred. Each CAP cycle consists of an *A phase* and a *B phase*, lasting between 2 and 60 seconds each (Figure A1). Furthermore, all CAP cycles begin with an A phase and end with a B phase. If any two A phases are separated by an interval of > 60 seconds during NREM sleep, this period is scored as non-CAP (NCAP) sleep. Moreover, if three consecutive A phases are followed by a NCAP condition, only the two complete CAP cycles will be scored as part of the CAP sequence (and not the last stand alone A phase). These scoring criteria are implemented because the CAP procedure is based upon the succession of *complete* CAP cycles (phase A + B) and the maintenance of a balanced duration between them.

In NREM sleep, the phase A patterns are composed of single, or clustered, arousal-related phasic events that are at least 1/3 bigger than the background EEG. The phase A activities can further be classified into three different subtypes depending on the proportion of high-voltage slow waves (EEG synchrony) to low-amplitude fast rhythms (EEG desynchrony). In particular,

- *Subtype A₁* consists of $\geq 80\%$ EEG synchrony and $\leq 20\%$ EEG desynchrony.
- *Subtype A₂* is a mixture of slow and fast EEG rhythms with between 20 – 50% of the phase occupied by EEG desynchrony.
- *Subtype A₃* is comprised of > 50% EEG desynchrony; any autonomic activity or body movements that occurs in conjunction with an A phase can be classified as part of the sequence.

When scoring CAP it is important to firstly define the preceding sleep stage according to the conventional criteria (Rechtschaffen & Kales, 1968), as what constitutes a particular subtype (*A₁*, *A₂* or *A₃*) is partly reliant on the sleep stage from which it emerges:

- intermittent alpha rhythms (EEG synchronization, *A₁*) and sequences of vertex sharp waves (EEG synchronization, *A₁*), in S1;
- sequences of two or more K-complexes alone (EEG synchronization, *A₁*) or followed by alpha-like components (EEG desynchronization, *A₂* or *A₃*) and beta rhythms (EEG desynchronization, *A₂* or *A₃*), in S2;

- delta bursts (EEG synchronization, A₁) which exceed by at least a third the amplitude of the background activity, in S3 and S4;
- transient activation phases (EEG desynchronization, A₃) and EEG arousals (EEG desynchronization, A₃), in all the stages.

(Terzano & Parrino, 2000, p. 120)

As CAP commonly occurs during the transition between sleep stages, a single CAP sequence may contain different sleep-related patterns, as well as be comprised of different arousal subtypes.

Figure A1. A CAP Sequence

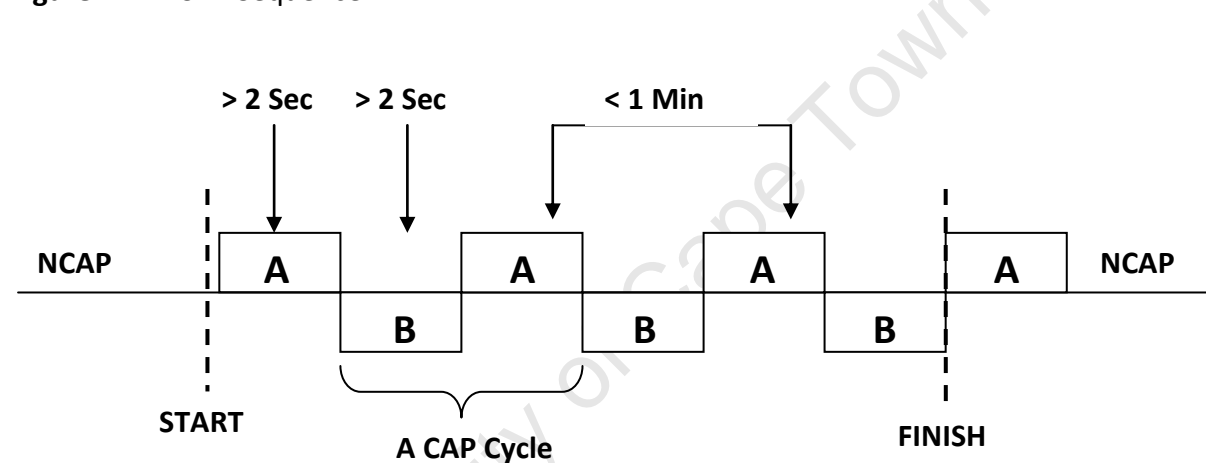


Figure A1. An illustration of a CAP sequence during sleep comprised of three CAP cycles. A single CAP cycle consists of an A and B phase, both lasting between 2 and 60 seconds each. In addition, the CAP cycles have to occur within 60 seconds of each other, and at least two full CAP cycles need to take place for a CAP sequence can be scored (Terzano & Parrino, 2000).

CAP during REM sleep

REM sleep is characterised by a distinctive lack of EEG synchronisation and is instead comprised solely of desynchronised patterns. Therefore, while individual A₃ arousals (or ASDA defined microarousals) commonly occur within 3 to 4 minute mean intervals during REM sleep, these arousals do not meet the temporal requirements (the 60 seconds rule) to be scored as CAP sequences. Under extreme pathological sleep conditions, such as in the case of sleep apnoea, it is possible to identify CAP A₃ sequences within REM sleep.

APPENDIX B
The Multidimensional Dream Questionnaire

DREAM DIMENSION QUESTIONNAIRE

FOCUS ON WHAT WAS GOING THROUGH YOUR MIND IMMEDIATELY BEFORE YOU WERE AWOKEN.

TRY AND REMEMBER AS MUCH AS POSSIBLE, IN AS MUCH DETAIL AS POSSIBLE, BEFORE YOU COMPLETE THE QUESTIONNAIRE.

Q 1 HOW DEEP was your sleep?

1: awake 2: drowsy 3: in light sleep 4: in deep sleep

Q 2 HOW MUCH did you dream?

4: greatly 3: fairly 2: relatively little 1: white dream 0: not at all

Q 3 How accurately do you REMEMBER your dream?

3: greatly 2: fairly 1: relatively little 0: not at all

Q 4 How EMOTIONAL was your dream overall?

3: greatly 2: fairly 1: relatively little 0: not at all

Q 5 WHAT EMOTIONS were present in the dream?

Anger/ Rage/ Aggression 3: intense 2: moderate 1: relatively little

Fear/ Anxiety / Apprehension 3: intense 2: moderate 1: relatively little

Sorrow/ Grief/ Loss 3: intense 2: moderate 1: relatively little

Seeking/ Curiosity/ Anticipation 3: intense 2: moderate 1: relatively little

Care/Nurturance/ Affection 3: intense 2: moderate 1: relatively little

Sexual Love/ Erotism 3: intense 2: moderate 1: relatively little

Playfulness/ Joy/ Exuberance 3: intense 2: moderate 1: relatively little

Dominance/ Power/ Control 3: intense 2: moderate 1: relatively little

Q 6 How PLEASANT was the dream?

1: very pleasant

2: moderately pleasant

3: slightly pleasant

4: Neutral

5: slightly unpleasant

6: moderately unpleasant

7: very unpleasant

Q 7 How much VERBAL AGGRESSION was in the dream?

6: Extreme degradation of a person (abusive, malicious)

5: Moderate degradation of a person

4: Criticisms/ arguments about behaviour/ beliefs of a person

3: Degradation of associated persons

2: Degradation of associated objects

1: Mild teasing/ hostile tension present

0: No verbal aggression

Q 8 How much PHYSICAL AGGRESSION was in the dream?

A: Killing a person or animal intentionally; suicide

B: Contemplating killing a person/animal; major aggression & violence (unprovoked)

C: Provoked attack & violence; accidental killing; killed by disease/ natural forces; serious, hostile threat

D: Intentional destruction property; accidental injury; defensive aggression; contemplation accidental death/ terminal illness of self/other

E: Verbal, gestural threats; carrying weapon/s; accidental but major property destruction; possibility of aggression (tension)

F: Rough play; minor property destruction

G: No physical aggression

Q 8.2 WHO was responsible for the physical aggression?

1: you

2: others

3: both you and others

Q 9 How AUDITORY was the dream?

3: greatly auditory 2: moderately 1: relatively little 0: not at all

Q10 How **VISUAL** was the dream?

- 4 = very much detail and clarity (like waking)
- 3 = moderate detail and clarity
- 2 = dream with only a little detail and clarity
- 1 = vague or hazy dream with no detail
- 0 = no visual imagery

Q11 Did you dream in **COLOUR**?

- 3: greatly 2: fairly 1: relatively little 0: black & white B: not visual

Q12 How much **ACTIVE THOUGHT** was in your dream?

- 3: greatly 2: fairly 1: relatively little 0: not at all

Q13 How much **PASSIVE THOUGHT** was in the dream?

- 3: greatly 2: fairly 1: relatively little 0: not at all

Q14 Were you trying **TO DO** anything in your dream?

- 3: greatly 2: fairly 1: relatively little 0: not at all

Q15 How much **ACTIVITY/ MOVEMENT** was there in the dream?

- 2: vigorous activity 1: moderate activity 0: no physical activity

Q16 How **UNFAMILIAR, STRANGE** or **DISTORTED** was the dream in relation to waking life?

- 1: exactly like waking 4: new/ unfamiliar but likely
- 2: similar with minor changes 5: new/unfamiliar and unlikely
- 3: similar with major changes 6: unfamiliar and bizarre

Q17 What **TIME** in your life do you associate with the dream events?

- 1: sleep laboratory 5: past year
- 2: past day 6: past 5 years
- 3: past week 7: over 5 years ago
- 4: past month 8: no particular time

Q18 How much **SEXUAL INTERACTION** was in the dream?

- 5: Intercourse 4: Foreplay 3: kissing 2: Sexual Advances 1: Thoughts/ Fantasies 0: None

Q19 How much **CONTROL** did you feel you had in the dream?

- 3: complete control 2: moderate control 1: just a little control 0: no control

Q20 What was **YOUR ROLE** in the dream?

- 1: main character 2: active participant 3: present but inactive 4: passive observer

Q21 **HOW LONG** was the dream?

- 5-10 s 10-60 s 1 - 5 min 5 - 15 min 15 min - 1 hr 1 hr +

Q22 How **CONNECTED** or **STORYLIKE** was the dream?

- 1: clear connection 2: weak connection 3: fragmented events 4: fragmented imagery/thoughts

Name: _____

Participant No.: _____

Dream No.: _____

Time of night: _____

Comments: _____

APPENDIX C
The Basic Emotions: Instructions for Participants

Q5. WHAT EMOTIONS were present in the dream?

You are required to select those categories that contain the emotions present in your dream.

You may select more than one category if necessary. Once you have selected a category,

indicate the intensity of the emotions present:

3 = these emotions were very intense

2 = a moderate amount of these emotions were present

1 = very little of these emotions were present

Definitions		
A	Anger/Rage: Aggression:	Anger refers to feelings of strong displeasure or hostility; annoyance; irritation; fury; resentment. Rage refers to feelings of violent, explosive anger. Hostility; violence; feelings of aggression.
B	Sexual Love/ Erotism:	Sexual love refers to the <i>fulfilment</i> of sexual gratification of any kind. The <i>desire</i> for sexual gratification, or the <i>anticipation</i> of any sexual interactions, should be rated under category D-SEEKING/CURIOSITY/ANTICIPATION. Only the <i>fulfilment</i> of any sexual urges should be rated under love.
C	Playfulness: Joy: Exuberance:	Finding or making causes for amusement; pleasantly humorous or jesting; full of fun and high spirits. Any actions relating to play should be rated here as well. Happiness; pleasure; enjoyment; bliss; delight. Enthusiasm; excitement; liveliness; energy; high-spirits; cheerfulness
D	Seeking/Curiosity: Anticipation:	To try to locate or discover; the act of searching for something; to try to obtain. <i>Curiosity</i> refers to feelings of inquisitiveness or interest. To look forward to, especially with pleasure; expectance; an expectation; suspense; hopefulness. If during the dream you feel pleasant anticipation (or expectation) of any kind, you should rate that feeling here.
E	Care/Nurturance: Affection:	To watch over; be responsible for; physical and emotional care and nourishment; to take care of or to nurture someone or something. A feeling of warm personal attachment or deep affection, as for a parent, child, spouse or friend. (<i>Note</i> : feelings of love should be

		included here only if of a non-sexual type- if feelings of love are both sexual and affectionate, then both categories (B AND E) should be chosen and rated according to the strength of the feelings involved).
F	Fear: Anxiety: Apprehension:	A distressing emotion aroused by impending danger, evil, pain, etc., whether the threat is real or imagined; the feeling or condition of being afraid; dismay, dread, terror, fright, panic. Distress or uneasiness of mind caused by fear of danger or misfortune. Uneasiness; worry; nervousness; hesitation.
G	Sorrow/Grief/ Loss:	Mental suffering or pain caused by injury, loss, or despair; a source of deep mental anguish, torment, distress.
H	Dominance/ Power/ Control:	Feelings of authority or control over others; asserting authority over others; control over events and people; feelings of power, high-status. DO NOT rate feelings of <i>wanting</i> power here, those should be considered anticipatory and rated under category D (see above). Only feelings of <i>having</i> power, authority or control should be rated.

*All descriptions were based on definitions from Panksepp (1998).

APPENDIX D

Relation to Everyday Experience

The relation to everyday experience scale has been copied from Winget and Kramer (1979, p. 127), and was originally developed by Foulkes et al. (1969). Raters were required to give each verbal dream report a rating of between 1 and 5, as per the criteria below.

1. Referring without distortion to some specific event in the subject's life—e.g., recreation or anticipation of a particular sporting event.
2. Basically realistic, everydayish, but not indicated as referring to some specific event that has or will happen—e.g., the subject and his friends are walking on a street downtown looking in stores for a baseball.
3. Some relation to the subject's everyday life: characters, settings, or actions are partially familiar, but with unrepresentative elements—e.g., the subject and his friends are playing ball on their usual field but stumble upon a sunken treasure chest.
4. Minimal contact with the subject's everyday life: characters, settings, or actions are familiar, but dream is basically unrepresentative—e.g., the subject and his school/ neighbourhood friends are digging for treasure on the beach of a South Sea island.
5. No apparent relation to the subject's everyday life: characters, settings, or actions are not representative—e.g., pirates are digging for sunken treasure.

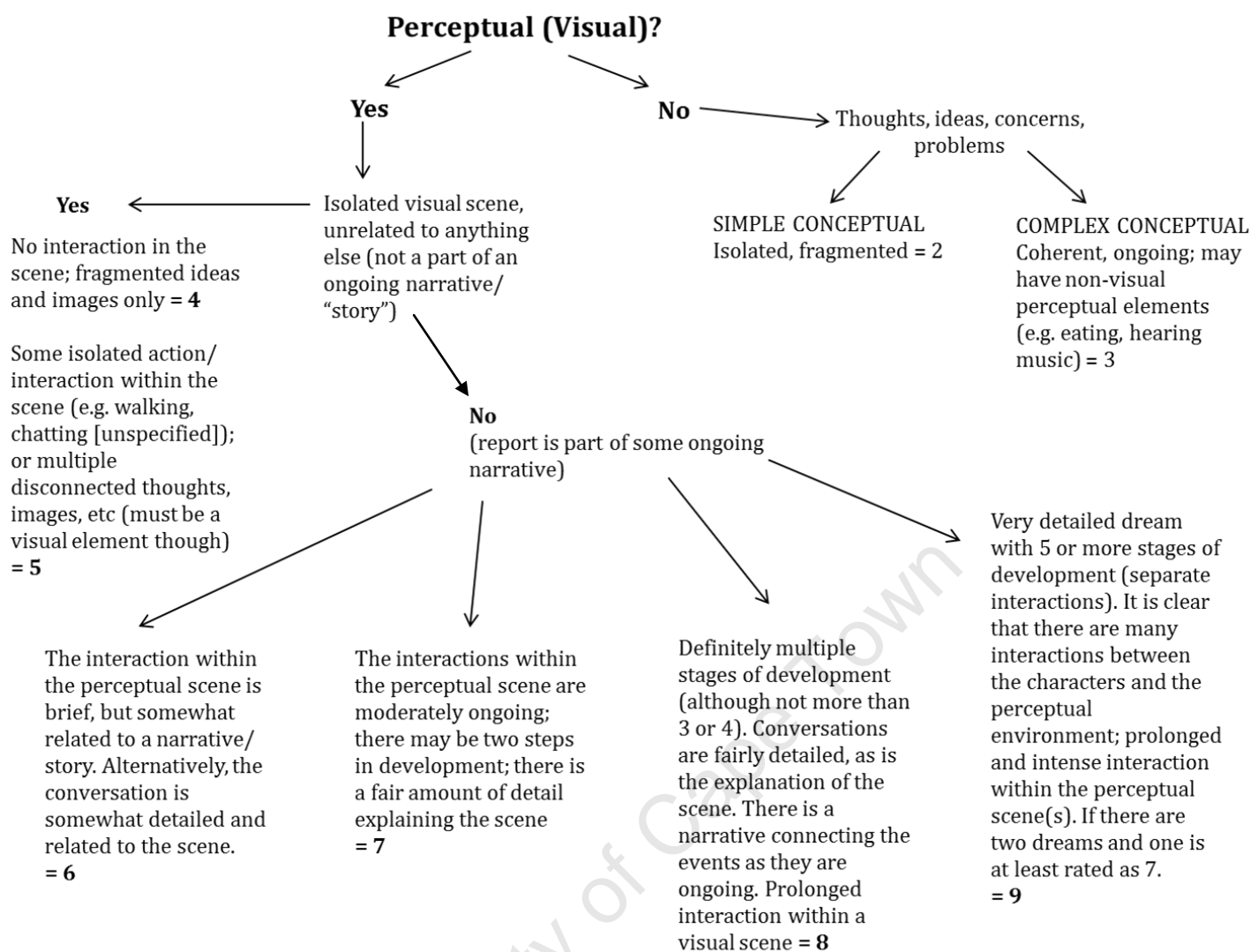
APPENDIX E

Perceptual-Interaction Rating Scale (PIRS): Instructions for External Raters

The aim of this scale is to rate the amount of interaction between the dream characters and the dream environment—be the characters people, animals or even inanimate objects. The more interaction described, the more perceptuality is inferred, as it is taken for granted that the great majority of interactions can only take place within a suitable dream (pseudosensory) environment. If the interaction is described as being solely between the characters in the dream a coherent visual-perceptual element will still be assumed, as it is presumed that a visual element is still required for these interactions to take place (unless the participant states otherwise).

It is important to note that interactions within a dream are not necessarily counted one-by-one because they are often not described in this way; it is up to you to judge the amount of interaction in the scene by logical inference. For instance, if the dreamer describes talking to somebody, regardless of whether they give any detail of the conversation, we can assume that this was an interaction between at least two dream characters that took place. See the examples provided below for further explanation.

Finally, the primary mode of perception for this scale is vision—this must be established first. Please follow the flow chart (below) for directions on how to rate the dream report according to its visual perceptual quality in combination with the interactions within the dream.



Negative Recall

0 – No Recall

No dream or mentation is recalled.

1 - White Dream

The participant is certain that they were dreaming, but cannot remember the content of the dream. If a single image can be recalled, give a rating of 4; if the participant describes ‘random images’ but cannot name them, this is considered a white dream. Likewise, if they describe ‘thoughts’ going through their mind, but cannot recall the content of the thoughts, then the report is rated as a white dream.

Example

P: ... I can't remember right now, but it had something to do with football....yeah, something to do with football.

(Even though a general theme is remembered, there is *no specific content*. Also, we are given the impression that there was a dream about football that cannot be remembered now. Be

careful to differentiate this kind of report from a rating of 2, 3 or 4. If the dreamer can only remember one thing, like sitting in a car, this describes the content of the dream and not just a general theme, and should be rated as 4).

Nonvisual Recall (Conceptual)

2 - Simple Conceptual Experience

Isolated and fragmented thoughts/ ideas/ motivations; an overall lack of coherence is characteristic.

Example

P: I'm thinking. I don't really, um, have anything specifically...

[Okay, so you felt awake?]

Yeah I was, yeah. I was actually thinking about when you come in, 'cos I was wondering, 'cos it felt like longer than the other breaks or gaps.

(This is considered conceptual activity; it is brief and is therefore considered simple conceptual rather than complex).

3 – Complex Conceptual Experience

A complex conceptual experience is one which is coherent and ongoing (but nonvisual), that may be accompanied by nonvisual perceptual elements (e.g., eating, hearing music).

Example

P: I was thinking of musicians...

No it wasn't images, it was just like...um, I don't know, it was just the idea of the fact that people can make music...

Okay, so I don't think I dreamt of visions of musicians but instead like a song that I heard from a soundtrack I have been listening to. It was just, I don't know, like hearing the song, like hearing different songs from different soundtracks, and I don't think I was seeing anything... yeah, it was mostly an auditory dream.

(The participant reports thinking about how musicians make music, as well as hearing multiple songs. This indicates that the conceptual experience consisted of a coherent, logical type of thinking and that it also involved a complex auditory experience—hearing music.)

Visual-perceptual, non-narrative/ incoherent

4 – Fragmented Visual Imagery

This is the most basic type of visual imagery and involves NO interactions. It may involve seeing a visual scene or briefly being in a visual scene, but does not involve interactions within the dream environment, or between participants in the dream. Along with

the visual scene or fragmented image there may be an incoherent and fragmented idea or thought as well.

Examples

1. *P: Um...nothing, um...um, I was in the middle of a fair, like a daze if you know what I mean, there wasn't anything significant.*

2. *P: I was kneeling outside a park or something, and it was cold.*

(Even though kneeling is a verb here, it is a static action and rather like a pose—there is no interaction within the scene).

5 – Isolated Interaction within a Visual-Perceptual Scene

There is some isolated interaction or action within a visual scene. Even if the participant does not purposely say that the scene is visual, they will describe where they were and what they could see, and in this way we can infer the visual element. If the participant describes walking, running, talking, etc. this is considered an interaction within the dream environment. Important: If the participant describes talking or chatting in a generic sense the report is rated as 5; however, if the details of the conversation are recalled, or if they describe running/ walking for some intended purpose, then this interaction becomes part of a more *comprehensive* dream narrative and is rated as 6 instead. Also, if there are multiple ideas, scenes, isolated thoughts and interactions it is rated as 5 (i.e. if there are two or more reports that independently would receive a rating of 4, a 5 is given).

Example

*P: It was kind of like a tuck-shop, vending machine type scenario. It was **definitely images more than a story**, and thoughts, and wasn't very connected. Fragmented a little bit. Also just very chilled and **chatting, people chatting a bit**.⁵³*

(The participant mentions here that the visual elements are incoherent and do not form part of a dream narrative. They then mention people chatting; for this reason the report is rated as 5 and not 4).

*P: I was **watching TV** and that, and it was weird, I don't think I was dreaming properly...and it was weird because I was like trying to justify like I could watch it, but obviously I'm not allowed to watch it now.*

P: Yeah, just the odd image and thoughts and stuff.

⁵³ Bolded words and phrases indicate either important information about the visual-perceptual elements of the dream or about interactions within the dream.

(Here the interaction is watching TV. The person is having thoughts or thinking of ways to justify watching TV, because they are supposed to be participating in this study. While this could be argued to be a part of some narrative, the justification is more of a thought that occurred during the dream rather than a dream plot or narrative that explains the interactions/actions of the dream.)

Visual Perceptual Dreaming, Part of an ongoing narrative

6 – Limited Interactions within a Visual-Perceptual Scene

The interaction within the visual-perceptual scene is brief, but somewhat related to a narrative or story (or for some purpose). If the scene consists solely of a conversation or speaking between characters, there is some description of the content of the conversation or speech (unlike the ‘generic’ talking of 5 above).

Examples

*P: I was dreaming about inter-racial relationships and something about a coloured child. I was with a coloured child who was **telling me about their parents**, and I was just thinking about it and **talking to the child** and, um, **saying something about how I want an inter-racial relationship**. And then, yeah, it was kind of story-like in a sense. It was less like just images and more like a proper dream, not like the other ones.*

(The overarching ‘theme’ of the dream appears to be inter-racial relationships; this gives the interactions some coherence, as the conversation in the dream is somewhat detailed and also about inter-racial relationships.)

*P: Um, I was in a group of people and yeah, there was **a lot of conversation about sleep and quality of sleep**, and just everything that this, well what I’m doing here I think... Um, it was **very theoretical**, just theories and talking of **ideas and stuff**.*

(Here the interaction is a conversation, but there is detail about the content of conversation. From what we are told the conversation [interaction] is somewhat ongoing and part of a theme—the conversation is about sleep and sleep quality, and ideas surrounding this.)

*P: It was more like an interaction than a full dream, but I dreamt that **I ran out of all my money before the end of the month and I had to go ask my dad for more**. And I felt, like, so **irritated** with myself because wanting to like prove to him that I could stick to a proper budget. And he was like, **“Oh, don’t worry, I’ll just put more into your account”**. And it wasn’t a big deal, but it felt to me as if it was a big deal...*

(We are given some of the content of the conversation [the main interaction] in the dream. The feelings and thoughts are also somewhat detailed and not generic; they form ideas that are cogent with the interactions in the dream).

7 – Moderate Interaction Within a Visual-Perceptual Scene

The interactions within the visual scene are moderately ongoing and there may even be two “steps” of development. There is a fair amount of detail explaining the scene as well.

Examples

*P: I was at a holiday destination, like spring break at Sun City, and my boyfriend’s friend was there who studies in Canada, and **he was just talking to my boyfriend** about all this stuff. And then he was like, “oh yeah” and he mentioned something, and then **I found out that my boyfriend is not going to be in Joburg**, where I’ going, for the duration of the time that like he said he was. So, um, he isn’t going to be there at all, and then I got really really upset cos I just didn’t know, found out, and was like, “what?” And then **he just kind of fobbed me off like he didn’t care** about it and I was just really sad. And I’m happy it was a dream.*

(There are multiple interactions in the dream that follow a general narrative. There is also detail about where the dreamer is and who is in the scene. NOTE: A narrative does not necessarily have to be ‘rational’ or realistic—it can be entirely fantastical as well.)

*P: I was in my garden and I was busy comparing things in my mind and I was **busy telling someone about how afraid I am of these little animals**, they were like shrews, squirrel type things in the garden, and big cats and I was saying **I can’t go through the one place of the garden because there’s shrews and big cats there**, which were frightening me. I dunno why, but that’s what I was dreaming about. And **I tried to rake up some leaves and clean up a portion of the garden, but I didn’t wanna go where these little creatures were.***

(Note the two stages of development: i) chatting to her friend about the animals in the garden; and ii) then cleaning the garden and being afraid. The scene is also somewhat detailed and coherent.)

8 – Prolonged Interactions within a Visual-Perceptual Scene

Definitely several stages of development (between three and four); the conversations are fairly detailed, as is the explanation of the scene. There is a narrative connecting the events of the scene as they are ongoing.

Example

*P: Okay, well, I have a friend and her name is Candice, and she has just gone back to Joburg to visit her family. In my dream she didn’t go back and she’s living on her own, and so she didn’t go back, but all of her family did, and so she was alone. So then she was all alone and she was very upset, and so **I took the car and went to go and visit her**. And my family was fortunate enough at the time to be staying at the “One and Only” ...that never*

*happened before, but lovely thought. And so then **she came and stayed with us**, but I remember that **my mom was very upset** in the dream and **we had a big fight** because she was like, “**You shouldn’t just invite people without asking my permission**”. And then I remember eventually she was like, “**no, it’s fine, don’t worry**”. It was a Thursday night so then **we went out and her boyfriend came along**. And that’s when I woke up.*

(The stages of development are as follows: i) picked up her friend; ii) brought her back to the apartment; iii) had a fight with her mom; iv) they all went out to dinner with her boyfriend. The dream has multiple stages of development and is fairly detailed).

9 – Intensive Interaction within a Visual-Perceptual Scene

Very detailed dream with five or more stages of development. It is clear that there are many interactions between the characters and the perceptual environment. If there are two dreams and one is at least rated as a 7, and the other at least as a 5, then 9 is rated overall.

Examples

*P: We’re at a house, almost like we’re on holiday. It was me and my sister, but it changed from my sister to my friend, if you know what I mean, and she didn’t believe that there were moles in the garden. There’s a lot more to it, but the place that we were at was a holiday house, but it was like um, like an exoskeleton type of thing. It was like you can live in it, but there wasn’t a roof and there wasn’t walls and stuff like that, it was just a bare structure of the walls and stuff like that. And, um, kids used it for um for hiding, like having little treasure hunts, and hiding in there. And the **I was looking for moles**...so I came across this little hole okay, and **I like kicked out a piece of the ground** and I was like, “hey this could have a mole in it”. So **I looked and there was some like rodents in the hole**. So **I got my iphone out to check with the torch** or whatever, and they were little hedgehogs inside like a little home type of thing. So **I got a cup and tried to catch the hedgehogs**, like they were so small. And then, um yeah like, **I went to show her and she was quite happy**.*

Yeah, then it changed to something more recent, but that’s more like new that came in if you know what I mean? Like similar to the other night when you came in and the car rushed by.

(The stages of development are as follows: i) The dreamer is with his sister/ friend and they are walking in the scene and she doesn’t believe there are hedgehogs; ii) he is looking for a mole in the exoskeleton of a building—it is unclear whether he can actually see children playing or just ‘knows’ that this happens here; iii) he finds a hole, looks into it with his iphone and finds baby hedgehogs. He catches one with a cup. [Although this is one step of development in the dream, these are many detailed interactions between the dreamer and

other objects in the dream, as well as between the dreamer and the hedgehogs]; iv) the dreamer takes the hedgehogs back to his friend to show her).

*P: Um, there was this new revolutionary product on the market that, my god, and it was helping me **clean my car**, and my best friend Robyn was also there, and **we were cleaning ourselves with it**. It was a really really good product, and **we were talking about turning it into a business venture** and stuff 'cos it was such a good product and we were like **spraying down our cars** and he was helping us, and I felt sorry for him because it was half seven at night and we **told him that he could stay until eight**. And we were just washing our cars and stuff – quite a pleasant dream.*

There was a bit of a weird dream before that about being in a house and with all these different levels, an also with my best friend Robyn, and the house turned into a Woolworths type place and we didn't know if we were going to get out. It was a separate dream.

(The first dream is rated as 7 and the last dream as 5 or 6).

APPENDIX F

Participant Consent and Information Form

Informed Consent to Participate in Research and Authorization for Collection, Use, and Disclosure of Sleep Patterns, Dream Recall Reports and Other Personal Data

You are being asked to take part in a research study. This form provides you with information about the study and seeks your authorization for the collection, use and disclosure of your sleep patterns, dream recall reports, as well as other information necessary for the study. The Principal Investigator (the person in charge of this research) or a representative of the Principal Investigator will also describe this study to you and answer all of your questions. Your participation is entirely voluntary. Before you decide whether or not to take part, read the information below and ask questions about anything you do not understand.

1. Title of Research Study

“Non-REM Dreaming in Relation to the Cyclic Alternating Pattern (CAP): An Exploratory Study”

2. Principal Investigator and Telephone Number

Danyal Wainstein

University of Cape Town

Contact number:

3. What is the purpose of this research study?

This research aims to investigate how dreaming is related to patterns of brain arousal occurring naturally during sleep every night.

4. What will be done if you take part in this research study?

In this experiment you will be asked to sleep at the Vincent Pallotti Private Hospital sleep laboratory for 3 inconsecutive nights. On the first night you will be left to sleep undisturbed (this will be your adaptation night); on the 2nd and 3rd nights you will be awakened five to six times each night to collect dream reports.

The date of each of the three sleep study nights will be arranged at least one week in advance, at a time convenient to you. You will retain your routine bedtime and waking time but will be asked to avoid caffeine and sugar in your diet for a few hours before bedtime. You will be required to come to the sleep laboratory based at Vincent Pallotti Private Hospital between 18 30 and 19 00 and will be briefed once more, in detail, on the procedure. You will be attached to a polysomnograph (PSG) machine which is designed to monitor your sleep patterns. Electrodes will be placed on your head, chest, near your chin and temples; these are completely safe and present no danger whatsoever to your health. They are designed to record the small electrical impulses naturally generated by your brain and will help us to determine which stage of sleep you are experiencing at a given point in time, by displaying

your brain's electrical patterns on a computer monitor. One or two researchers will survey the monitor in an adjoining room. They will be available to you for assistance at any time. In the morning the electrodes will be removed.

After the sleep sessions are over, you will be informed in detail about the design of the study and the research questions we hope to address with this study. You will also have the opportunity to ask questions and thus learn more about psychological/ sleep research. If you have any questions now or at any time during the study, you may contact the Principal Investigator listed in #3 of this form.

5. If you choose to participate in this study, how long will you be expected to participate in the research?

The sleep study: 3 nights only over a maximum period of two weeks.

6. How many people are expected to participate in the research?

30

7. What are the possible discomforts and risks?

Sleeping in an environment other than your own bedroom might feel strange and uncomfortable at first. Great precautions will be taken to ensure your safety and comfort. The sleep laboratory at Vincent Pallotti is fully equipped with a proper bed, clean bedding, restrooms and a kitchenette. It is situated in a secure building with adequate surveillance and alarm system. Attempts will be made to familiarise you with the PSG and the electrodes used will be padded and lubricated so as to be as non-intrusive as possible.

The awakenings during the night will include being gently awakened and asked a few questions about whether the interviewer interrupted any thoughts, images or more complex perceptual experiences. You will only be required to honestly describe what was happening. The *absence* of any mental activity will be equally as important to the study as the presence of mental experiences, and therefore there is no pressure to report any mental activity. The five to six awakenings per night may leave you feeling a little tired the following day.

However, you should still receive enough sleep to function adequately the following day, even though you may be a bit tired. Nonetheless, it is not advised that you participate on a night preceding a test or exam, as your performance on such demanding activities may be hindered in some way by not have slept normally the previous night.

8. What are the possible benefits to you?

You will receive monetary compensation for participating in this study. If you *complete all three nights* in the sleep lab you will receive a total of R400. You will not be paid for the adaptation night; however, you will be paid R200 after the second night and R200 again after the third night. Other possible benefits include learning about sleep and dream research protocols.

9. If you choose to take part in this research study, will it cost you anything?

Your only cost will be transportation to and from Vincent Pallotti hospital. If you do not have transportation to and from the hospital, inform the Principle Investigator, and we will ensure that you have safe transportation.

10. Can you withdraw from this research study?

You are free to withdraw your consent and to stop participating in this research study at any time. If you do withdraw your consent, this will not impact the research negatively. However, withdraw from the study will result in the forfeit of your monetary compensation.

If you have any questions regarding your rights as a research subject, you may phone the Psychology Department offices at 021-650-3430.

11. If you withdraw, can information about you still be used and/or collected?

Information already collected may be used.

12. Once personal and performance information is collected, how will it be kept secret (confidential) in order to protect your privacy?

Information collected will be stored in locked filing cabinets or in computers with security passwords. Only certain people have the right to review these research records. These people include the researchers for this study and certain University of Cape Town officials. Your research records will not be released without your permission unless required by law or a court order.

13. What information about you may be collected, used and shared with others?

The information gathered from you will be: (1) demographic information (2) information on any your quality of sleep (3) your verbal comprehension ability (4) your medical history (5) records of your sleep architecture (6) psychiatric inventory, (7) your dream recall reports, (8) personality characteristics (via a questionnaire). If any of this data is published in a scientific journal, the Principal Investigator will ensure that you cannot be identified in any way, and your complete anonymity will be maintained.

14. How will the researcher(s) benefit from your being in the study?

In general, presenting research results helps the career of a scientist. Therefore, the Principal Investigator and others attached to this research project may benefit if the results of this study are presented at scientific meetings or in scientific journals. This study is being undertaken for the Principal Investigator's masters degree.

15. Signatures

As a representative of this study, I have explained to the participant the purpose, the procedures, the possible benefits, and the risks of this research study; as well as how the participant's performance and other data will be collected, used, and shared with others:

Signature of Person Obtaining Consent and Authorization Date

You have been informed about this study's purpose, procedures, possible benefits, and risks; and how your performance and other data will be collected, used and shared with others. You have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time. You voluntarily agree to participate in this study. You hereby authorize the collection, use and sharing of your performance and other data. By signing this form, you are not waiving any of your legal rights.

Name of Participant ("Study Subject")

Signature of Person Consenting and Authorizing Date

APPENDIX G

Principal Components Analysis of the Multidimensional Dream Questionnaire Analysis One

The *Multidimensional Dream Questionnaire* (MDQ) is a self-report measure based on the eight dimensions of dreaming identified by Hauri et al. (1967), in conjunction with Panksepp's (1998) basic emotions, and a few other measures of interest (see Dream Measures and Appendix B). Due to the fact that the eight factors were used in conjunction with these additional variables, an exploratory, rather than a confirmatory, analysis was carried out to identify the simple structure.

A principal component analysis (PCA) was conducted on 25 items from the Multidimensional Dream Questionnaire (MDQ). Questions 1, 2, and 3 were excluded from the analysis, as they were intended as global measurements, unrelated to any *one* factor. Additionally, Question 17 was excluded from the analysis because it was measured at a nominal level. Oblique rotation (direct oblimin, with delta set to 0) was used, as it is the preferred method for the achievement of simple structure (Cudeck, 2000). Listwise exclusion of missing data was applied, resulting in a sample of 159 dream reports from 20 participants. In order to deal with the non-independence of the data, the unstandardized residuals for each item were used rather than the raw scores (Figueredo et al., 1992). The Kaiser-Meyer-Olkin (KMO) measure verified the sampling adequacy for the analysis, $KMO = .706$ ('good' according to Field, 2009), and all KMO values for individual items were above the acceptable limit of .5, except for LUST (.481), SEEKING (.489), Bizarreness (.448) and Passive Thought (.459). Bartlett's test of sphericity $\chi^2(300) = 1257.79, p < .0001$, indicated that correlations between items were sufficiently large for PCA. Communalities after extraction were all above .5, with 15 items (60%) above the recommended .7 (Field, 2009); exceptions were Storylikeness (.472) and Control (.379), indicating that these variables may potentially be problematic due to a lack of shared variance. The determinant for the *R*-matrix (correlation matrix) indicated potential multicollinearity, as it was lower than the suggested limit of .00001 (Fields, 2009); however, because of the method of analysis (PCA), multicollinearity in the data did not present an issue.

An initial analysis was run to obtain eigenvalues for each component in the data. Nine components had eigenvalues over Kaiser's criterion of 1 and in combination explained 68.17% of the variance. The scree plot was slightly ambiguous and showed inflexions that would justify retaining three, six or nine components. In order to make an informed decision regarding the number of factors to be retained, the factor loadings after rotation were

considered. The pattern matrix (Table G1) showed the nine extracted components to be mostly independent; however, certain components had too few factor loadings (for instance, Component 6), and two variables failed to load significantly onto any of the components (Control and Length). Furthermore, certain of the variables with KMO values below .5 either did not cluster with any other variables (Passive Thought), or did not cluster in a theoretically meaningful way (SEEKING)⁵⁴. Therefore, before deciding on the final number of components to be retained, these variables were removed from the model in an attempt to produce a more meaningful simple structure.

Analysis Two

The KMO measure indicated slightly better sampling adequacy after the removal of the four variables, $KMO = .724$, and Bartlett's test of sphericity $\chi^2(210) = 1045.13, p < .0001$, continued to show that correlations between the items were sufficiently large for PCA. With the exceptions of LUST (.481), Sexuality (.487) and Bizarreness (.469), all KMO values for individual items were above the acceptable limit of .5, with most falling above the recommended limit of .7 (Field, 2009). Furthermore, all communalities after extraction were above .6, except for Storylikeness (.438), GRIEF (.484), and CARE (.332). Additionally, CARE failed to load significantly onto any of the components, and was thus removed before further interpretation of the component structure.

⁵⁴ It is important to note that only a percentage of the 159 dream reports used for this analysis contain a positive rating for each of the basic emotions. For example, SEEKING was found to be the most common basic emotion, with 54% of all reports positively rated for this emotion. This means that, of the total 159 dream reports used, approximately only 85 have a positive value for SEEKING—the remainder have SEEKING rated as 0. This creates a definite skew in the data, for all of the basic emotions. Therefore, any PCA analysis hoping to accurately identify the component structure of the basic emotions would require a sample larger than at present. Nonetheless, the basic emotions have been included in this analysis, as some still have sufficient sampling adequacy (KMO values greater than .5). Accordingly, those basic emotions with low KMO values (>.5), with component loadings that do not make theoretical sense, will be excluded from further analysis.

Table G1.
Pattern Matrix^a for Analysis 1 of the PCA

Items	Rotated Component Loadings								
	1	2	3	4	5	6	7	8	9
Auditory	.866								
Storylikeness	.574								
Emotionality	.446								
Pleasantness		-.815							
FEAR		-.799							
GRIEF		-.565							
PLAY		.561							
DOMINANCE			-.842						
P. Aggression			-.830						
RAGE			-.508						
V. Aggression			-.499						
Sexual Interaction				.829					
LUST				.828					
Visual Clarity					.938				
Colour					.914				
Control									
Passive Thought						-.837			
Length									
Bizarreness							.821		
Activity							.559		
SEEKING								-.775	
CARE								.537	
Role									-.932
To Do									-.552
Active Thought	.411								-.475

Note. Only loadings above .4 have been included. Extraction Method: Principal Component Analysis. Rotation Method: Oblimin with Kaiser Normalization. To Do = Were you trying to do anything in your dream?; P. Aggression = physical aggression; V. Aggression = verbal aggression.

^a Rotation converged in 15 iterations.

Analysis Three

After removing CARE, the sample continued to be suitable for PCA analysis, KMO = .729, Bartlett's Sphericity $\chi^2(190) = 1018.01, p < .0001$. The individual KMO values for

LUST (.481), Sexuality (.487) and Bizarreness (.469) remained below .5. Communalities were mostly above .6, with only Storylikeness (.430) below the recommended limit.⁵⁵ While not imperative to the analysis, the determinant of the R-matrix now indicated that there was unlikely to be any multicollinearity in the data, Determinant = .001.

The analysis was re-run to obtain eigenvalues for each component in the data. Seven components had eigenvalues over Kaiser's criterion of 1 and in combination explained 69.01% of the variance. The scree plot was slightly ambiguous and showed inflexions that would justify retaining four or seven components. In order to make an informed decision regarding the number of components to be retained, the variable loadings after rotation were considered.

An independent component structure was shown by the pattern matrix (Table G2), with only one item (To do) loading significantly on two components. The items that cluster on the same components suggest that Component 1 represents *Dream Intensity*, Component 2 *Pleasantness*, Component 3 *Hostility*, Component 4 *Visual Perceptuality*, Component 5 *Sexuality*, Component 6 *Bizarreness*, and Component 7 *Active Participation*.

Examination of the structure matrix (Table G3)⁵⁶ revealed, however, that Components 1 and 7 (dream intensity and active participation) were substantially interrelated, with *emotionality*, *active thought* and *to do* loading significantly on both components. Theoretically, it might be argued that active participation is an element of dream intensity (Hauri et al., 1967), and that in this analysis at least, the overlap between the two justifies merging them into a single dream intensity component. The analysis was rerun and six components were extracted as a test of whether removing one component would result in the automatic merging of Components 1 and 7.

⁵⁵ At this point, these variables were retained because they continued to load meaningfully onto certain components, despite their limitations.

⁵⁶ The structure matrix represents the correlation coefficients between each variable and the factor/ component, and therefore takes into account the relationship between components more than the pattern matrix (Fields, 2009). On theoretical grounds, it is expected that certain of the components will be interrelated – interpretation of the structure matrix will help to clarify the extent of this interrelatedness, as well as aid in the identification of those components that are most interrelated.

Table G2.

Pattern Matrix^a for Analysis 3 of the PCA

	Rotated Component Loadings						
	1 Dream Intensity	2 Pleasantness	3 Hostility	4 Visual Perception	5 Sexuality	6 Bizarreness	7 Active Participation
Auditory	.886						
Storylikeness	.600						
Active Thought	.590						
Emotionality	.539						
To Do	.518						-.433
Pleasant		-.813					
FEAR		-.800					
GRIEF		-.617					
PLAY		.513					
P. Aggression			-.857				
DOMINANCE			-.817				
RAGE			-.537				
V. Aggression			-.527				
Visual Clarity				.941			
Colour				.912			
LUST					.852		
Sexual					.836		
Bizarreness						.842	
Activity						.577	
Role							-.843

Note. Only loadings above .4 have been included. Extraction Method: Principal Component Analysis. Rotation Method: Oblimin with Kaiser Normalization. To Do = Were you trying to do anything in your dream?; P. Aggression = physical aggression; V. Aggression = verbal aggression.

^a Rotation converged in 18 iterations.

Table G3.
Structure Matrix for Analysis 3 of the PCA

	Rotated Component Loadings						
	1 Dream Intensity	2 Pleasantness	3 Hostility	4 Visual	5 Sexuality	6 Bizarreness	7 Active Participation
Auditory	.809						
Emotionality	.700			.468			-.442
Active Thought	.665						-.454
To Do	.653						-.570
Storylikeness	.636						
Pleasant		-.823					
FEAR		-.788					
GRIEF		-.599					
PLAY		.550					
P. Aggression			-.816				
DOMINANCE			-.813				
V. Aggression	.446		-.677				
RAGE	.425		-.650				
Visual Clarity				.895			
Colour				.892			
LUST					.842		
Sexual					.839		
Bizarreness						.801	
Activity						.615	-.417
Role							-.845

Note. Only loadings above .4 have been included. Extraction Method: Principal Component Analysis. Rotation Method: Oblimin with Kaiser Normalization. To Do = Were you trying to do anything in your dream?; P. Aggression = physical aggression; V. Aggression = verbal aggression.

Analysis Four

The values for the KMO test of sampling adequacy, as well as Bartlett's test of Sphericity, were unchanged from the third analysis (above). However, the individual KMO values for LUST (.481), Sexuality (.487) and Bizarreness (.469) remained below .5. In addition, Storylikeness (.398), Role (.479) and GRIEF (.465) now had communalities lower than the recommended limit of .5. No multicollinearity was observed in the data, Determinant = .001.

As expected, when six components were deliberately extracted, Components 1 and 7 merged to form a single component (Table G4). All Six components had eigenvalues over Kaiser's criterion of 1 and in combination explained 63.82% of the variance. Moreover, both

the pattern and structure (Table G5) matrices showed the six components to be mostly independent, with only a few interrelated items apparent in the structure matrix. ***Reliability***

Analysis of the Six Component Model

Reliability analysis of the Dream Intensity, Aggression and Visual Perception components showed high internal consistency for these subscales, with Cronbach's $\alpha > .75$ for each (Table G4). Additionally, all *item-to-total* correlations were above the recommended limit of .3 (Fields, 2009), and all *inter-item* correlations were sufficiently high as well, with only two inter-item correlations (for the Dream Intensity subscale) found to be lower than the suggested .3 (Hair, Black, Babin, Anderson, & Tatham, 2006). Tests of Cronbach's α if an item is deleted indicated that all items within each subscale were positively contributing to the overall reliability for that scale. Based on both the reliability analysis, as well as the distinctive item loadings for Components 1, 3 and 4, no items were removed from these subscales.

Analysis of the remaining three subscales (Pleasantness, Sexuality and Bizarreness) revealed a less than optimum level of reliability. In particular, Sexuality and Bizarreness both had very low reliability, Cronbach's $\alpha < .35$, while the Pleasantness subscale was found to border the lower limit of acceptable reliability, Cronbach's $\alpha = .65$ (Table G4). Further exploration of the Pleasantness subscale showed that GRIEF had a low item-to-total correlation (.311), and that the value of alpha would increase to .68 were this item removed. Based on these reliability statistics, the principle components analysis was rerun excluding GRIEF, as well as the four items comprising the Sexuality and Bizarreness subscales (LUST, Sexuality, Bizarreness and Activity).⁵⁷

⁵⁷ It is also worth noting that the four items that make up these two subscales had individual KMO values lower than the recommended .5, indicating potential problems with sampling adequacy.

Table G4.

Pattern Matrix^a for Analysis 4 of the PCA

	Rotated Component Loadings					
	1 Dream Intensity	2 Pleasantness	3 Hostility	4 Visual	5 Sexuality	6 Bizarreness
To Do	.75	-.08	-.08	.03	-.15	.10
A. Thought	.74	-.08	.02	.01	-.07	-.09
Auditory	.71	.23	-.07	-.22	.06	.14
Emotionality	.65	-.20	-.21	.22	.09	-.00
Role	.62	-.05	.10	.10	.05	-.30
Storylikeness	.57	-.04	-.12	-.01	.06	.14
Pleasant	-.16	-.79	-.18	-.19	-.02	-.01
FEAR	.25	-.78	.07	.08	-.00	-.07
GRIEF	.08	-.65	.14	.07	-.02	.21
PLAY	.27	.48	.30	.26	-.04	.19
DOMINANCE	.02	.11	-.80	-.03	-.06	.01
P. Aggression	-.06	.01	-.77	.09	.09	.17
RAGE	.16	.03	-.69	.02	-.07	-.19
V. Aggression	.18	-.24	-.67	.12	-.09	-.11
Visual Clarity	-.09	-.05	-.04	.91	.03	-.02
Colour	-.04	.09	-.08	.89	-.04	.00
LUST	-.08	-.07	.02	-.05	.85	-.11
Sexual	.07	.05	.01	.04	.84	.08
Bizarreness	-.07	-.12	.09	-.08	-.05	.86
Activity	.23	.06	-.12	.30	.08	.54
Eigenvalues	3.60	2.23	2.81	2.43	1.54	1.45
% of variance	21.68	12.94	8.67	7.31	7.18	6.04
α	.79	.65	.76	.81	.31	.32

Note. Bolded values indicate substantial component loadings above .4. Extraction Method: Principal Component Analysis. Rotation Method: Oblimin with Kaiser Normalization. To Do = Were you trying to do anything in your dream?; P. Aggression = physical aggression; V. Aggression = verbal aggression; α = Cronbach's alpha.

^a Rotation converged in 10 iterations.

Table G5.
Structure Matrix for Analysis 4 of the PCA

	Rotated Component Loadings					
	1 Dream Intensity	2 Pleasantne ss	3 Hostility	4 Visual	5 Sexuality	6 Bizarreness
To Do	.779					
Emotionality	.761			.426		
A. Thought	.738					
Auditory	.666					
Role	.612					
Storylikeness	.603					
Pleasant		-.811				
FEAR		-.784				
GRIEF		-.623				
PLAY		.527				
DOMINANCE			-.792			
P. Aggression			-.754			
V. Aggression			-.750			
RAGE			-.729			
Visual Clarity				.888		
Colour				.881		
Sexual					.845	
LUST					.837	
Bizarreness						.835
Activity				.431		.587

Note. Only loadings above .4 have been included. Extraction Method: Principal Component Analysis. Rotation Method: Oblimin with Kaiser Normalization. To Do = Were you trying to do anything in your dream? P. Aggression = physical aggression; V. Aggression = verbal aggression.

Analysis Five

The KMO measure indicated slightly better sampling adequacy after the removal of the aforementioned five variables, KMO = .761; Bartlett's test of sphericity $\chi^2(105) = 825.48, p < .0001$, continued to show that correlations between the items were sufficiently large for PCA. All KMO values for individual items were above the acceptable limit of .5, with most falling above the recommended limit of .7. All communalities after extraction were above .5, except for Storylikeness (.395), and Role (.364). These variables were retained

despite these low communalities because of their theoretically meaningful loading on the dream intensity subscale (Hauri et al., 1967). No multicollinearity was indicated in the data, Determinant = .003.

The analysis was rerun to obtain eigenvalues for each component in the data. Four components had eigenvalues over Kaiser's criterion of 1 and in combination explained 62.97% of the variance. Similarly, the scree plot showed an inflexion that would justify retaining four components. Given the convergence of the scree plot and Kaiser's criterion on four components, this is the number of components that were retained in the final analysis.

The pattern matrix of component loadings after rotation showed that the items loaded significantly on only one component (Table G6). Also, unlike the previous six component solution, the structure matrix (Table G7) shows very little interrelatedness between the components, with only one item (Emotionality) loading significantly on two components (albeit, far more significantly on the first one). Furthermore, loadings on both matrices are relatively high, with most exceeding .7, indicating a strong relationship between each of the items and the corresponding component. Lastly, only 43 nonredundant residuals (40%) with absolute values greater than 0.05 were present; this amount falls below the recommended limit of 50% (Fields, 2009), further demonstrating that the model is a suitable fit to the data.

The items that cluster on the same components suggest that Component 1 represents *dream intensity*, Component 2 *pleasantness*, Component 3 *hostility*, and Component 4 *visual perceptuality*. The four components retained are identical to components 1 - 4 of the previous model, and therefore, the integrity of the original model has been preserved despite the removal of GRIEF from the pleasantness subscale, and the four items comprising the Sexuality and Bizarreness subscales. In fact, the amount of variance accounted for by the new four component solution is roughly equal to that of the previous six component solution (62.97% vs. 63.82%, respectively), and the sampling adequacy for the new model has even been slightly improved ($KMO = .76$). Therefore, the stability of the component structure, as well as the good internal consistency and reliability of the four components (Table G6), indicates that this model likely provides the most parsimonious and stable latent structure for the present MDQ.

Reliability Analysis of the Four Component Model

Reliability analysis of the dream intensity, hostility and visual perception components showed high internal consistency for these subscales, with Cronbach's $\alpha > .75$ for each (Table G6). Additionally, all *item-to-total* correlations were above the recommended limit of .3 (Fields, 2009), and all *inter-item* correlations were sufficiently high as well, with only two

correlations (for the dream intensity subscale) found to be lower than the suggested $r = .3$ (Hair et al., 2006). Tests of Cronbach's α if an item is deleted indicated that all items within each subscale were positively contributing to the overall reliability for that scale. Based on both the reliability analysis, as well as the distinctive item loadings for Components 1, 3 and 4, no items were removed from these subscales.

Analysis of the remaining pleasantness subscale revealed a less than optimum level of reliability, Cronbach's $\alpha = .68$. However, all item-to-total correlations were above the recommended limit of .3 (Field, 2009), and most inter-item correlations were satisfactory as well, except for PLAY, $r = .287$, and FEAR, $r = .268$. Tests of Cronbach's α if an item is deleted indicated that all items contributed to the overall reliability.

Table G6.

Multidimensional Dream Questionnaire – Rotated Components Matrix^a

Items ^b	Rotated Component Loadings			
	1 Dream Intensity	2 Pleasantness	3 Hostility	4 Visual
To Do	.768	.084	.053	.023
Active Thought	.744	.069	-.013	.003
Auditory	.698	-.287	.072	-.187
Emotionality	.676	.139	.177	.220
Role	.606	.051	-.071	.024
Storylikeness	.597	-.050	.096	.013
FEAR	.316	.835	-.188	.131
Pleasantness	-.106	.824	.092	-.141
PLAY	.259	-.595	-.246	.230
DOMINANCE	.004	-.104	.833	-.047
P. Aggression	-.044	-.061	.767	.070
RAGE	.136	.085	.680	.027
V. Aggression	.192	.287	.646	.128
Visual Clarity	-.056	-.005	.033	.914
Colour	-.010	-.113	.076	.891
Eigenvalues	3.49	2.08	2.73	2.19
% of variance	27.52	15.58	10.62	9.25
α	.79	.68	.76	.81

Note. Bolded numbers indicate component loadings above .4. Extraction Method: Principal Component Analysis. Rotation Method: Oblimin with Kaiser Normalization. α = Cronbach's alpha. P. Aggression = physical aggression; V. Aggression = verbal aggression.

^a Rotation converged in 10 iterations.

^b $N = 159$ dream reports from 20 participants.

Table G7.
Structure Matrix for Analysis 5 of the PCA

	Rotated Component Loadings			
	1 Dream Intensity	2 Pleasantness	3 Hostility	4 Visual Perception
To Do	.79	.10	.23	.23
Emotionality	.77	.17	.37	.41
Active Thought	.74	.08	.16	.20
Auditory	.66	-.26	.16	.01
Storylikeness	.62	-.03	.22	.18
Role	.60	.05	.07	.18
Pleasant	-.12	.84	.18	-.18
FEAR	.32	.81	.02	.18
PLAY	.26	-.64	-.26	.29
DOMINANCE	.17	.03	.81	.04
P. Aggression	.14	.06	.76	.13
V. Aggression	.37	.39	.75	.23
RAGE	.29	.19	.73	.13
Visual Clarity	.19	-.02	.11	.90
Colour	.24	-.13	.14	.90

Note. Bolded numbers indicate component loadings above .4. Extraction Method: Principal Component Analysis. Rotation Method: Oblimin with Kaiser Normalization.

APPENDIX H

Reliability Analysis for Types of Dream Recall

Although the recollection of *any* mentation has previously been used as a criterion to dichotomise positive and negative dream recall (Foulkes, 1962), the value of differentiating between cognitive activity and dreaming has more recently been advocated (Nielsen, 2000). Consequently, each dream report was allocated to one of four categories according to its PIRS rating (Appendix E). The four groups were defined as: i) *no recall* (PIRS = 0); ii) *no content* (an entirely or almost entirely forgotten dream; PIRS = 1),⁵⁸ iii) *cognitive activity* (static visual images, thinking, reflecting, bodily feelings, or vague or fragmentary impressions, PIRS = 2-4); and iv) *dreaming* (any visual-perceptual experience involving at least a single interaction between any dream character and another dream character, or any dream character and the dream environment, PIRS > 4).

A Chi-Square contingency table was used to establish the interrater agreement for the types of recall categories; results are shown in Table H1. Overall, both raters allocated approximately equal number of dream reports to each of the categories, $\chi^2(3) = 2.32, p < .52$, 2 tailed. Despite tendencies for R1 to rate more reports as dreaming than as cognitive activity (compared with R2), these trends were found to be nonsignificant (standardised residual for cognitive activity = $\pm 0.8, p > .05$). Therefore, there was general agreement as to the approximate percentage for each of the recall categories. Further analyses of the types of dream recall were subsequently based on R1's (author) allocations (see Methods).⁵⁹

⁵⁸ Literature shows that there are differences in peripheral autonomic activation (e.g., heart rate, respiration) preceding no content dreams compared with no recall reports (Fisher, Byrne, Edwards, & Kahn, 1970). Additionally, an independent samples *t* test comparing depth of sleep for no recall and no content dreams showed that there participants reported significantly deeper sleep, $t(81.77) = -3.40, p < .0005$, and less waking perception, $t(79.87) = 3.04, p < .002$ (both 1 tailed), following reports of no content compared to reports of no recall. These categories were therefore differentiated on the basis that no content reports appear to be genuinely forgotten dreams.

⁵⁹ As mentioned in the Methods chapter, R1 was blind to all preawakening conditions (sleep stage, sleep stability, time of night), as well as participant identity, when the dream reports were rated. Furthermore, the reports were randomised to further ensure that no bias was involved in the rating.

Table H1.
*Chi-Square Contingency Table for Absolute Inter-Rater Agreement –
 Types of Dreaming*

Type of Dreaming	Rater		Total Count
	R1	R2	
No Recall			
Count	36	38	74
Expected Count	37	37	.
% Within Type	48.60	51.40	.
Std. Residual	-0.20	0.20	.
No Content			
Count	22	20	42
Expected Count	21	21	.
% Within Type	52.40	47.60	.
Std. Residual	0.20	-0.20	.
Cognitive Activity			
Count	37	48	85
Expected Count	42.50	42.50	.
% Within Type	43.50	56.50	.
Std. Residual	-0.80	0.80	.
Dreaming			
Count	87	76	163
Expected Count	81.50	81.50	.
% Within Type	53.40	46.60	.
Std. Residual	0.60	-0.60	.
Total Count	182	182	

Note. R1 = Rater 1; R2 = Rater 2; Count = the actual number of dream reports rated under each category; Expected Count = the expected number of dream reports for each category; % Within Type = the percentage within each category for each rater; Std. Residual = standardised residuals.

APPENDIX I

Descriptive Statistics for the Qualitative Dream Measures

Table I1 is a display of the Pearson correlation coefficients for the qualitative dream content variables in this study. Subjective depth of sleep was positively related to all measurements of global dreaming. This implies that, regardless of stage of sleep, positive dream recall was associated with an increased subjective depth of sleep. Everyday experience (EE), or bizarreness, was also significantly related to all three of the global dream measures; this association is congruent with previous studies that have found bizarreness to be associated with more intense dreaming (Hobson et al., 2000). The lack of significant correlation between EE and the other qualitative dream measures confirmed the divergent validity of this measure.

Table I1.

Dream Measures: General Intercorrelations

	H	VP	P	DI	EE	DS	PIRS	TRC	AD
Hostility ^a	1	.19	-.14	.39***	.15	.21*	.29***	.32***	.16
Visual Perception ^a		1	.10	.34***	.15	.24*	.49***	.27***	.41***
Pleasantness ^a			1	-.001	-.004	.21*	.09	.05	.21*
Dream Intensity ^a				1	.14	.31***	.51***	.38***	.50***
Everyday Experience ^a					1	.16	.47***	.28***	.22*
Depth of Sleep ^b						1	.30***	.16	.35***
PIRS ^b							1	.69***	.87***
TRC ^b								1	.54***
Amount of Dreaming ^b									1

Note. Bolded values indicate statistical significance. PIRS = perceptual-interaction rating scale; TRC = total recall count.

^a Positive recall only, $n = 168$

^b All recall, including both positive and negative, $n = 267$

* $p < .01$, ** $p < .001$, *** $p < .0001$, 2-tailed.

Three of the four MDQ subscales—dream intensity, hostility and visual perception—were significantly positively related to all three of the global dream measures. Furthermore, each of these MDQ subscales was also significantly related to depth of sleep. Pleasantness was unrelated to any other measures, except depth of sleep and AD, implying that more pleasant dreams were self-rated as more dreamlike. Hostility was relatively unrelated to pleasantness as well, indicating that aggression in dreams, and the basic emotions accompanying aggression—RAGE and DOMINANCE—are not necessarily unpleasant; this conclusion was reached in Hauri et al.'s (1967) original study as well.

The Basic Emotions

Table I2 is a display of the Pearson correlation coefficients for the self-rated basic emotions in relation to one another, as well as in relation to global dream composite (DC) measure and TRC. With the exception of a few significant relationships, the basic emotions are largely independent from one another. The significant relationships that did emerge, though, were consistent with what might be expected—for instance, GRIEF and FEAR were positively related.

In relation to global dreaming, four of the basic emotions warrant attention—RAGE, SEEKING, CARE, and PLAY—these basic emotions were positively significantly related to DC and TRC. Interestingly, three of the four latter emotions were positive and in combination (*positive emotion*), very significantly related to both DC and TRC. In contrast, the aversive emotions (FEAR and GRIEF) were very weakly, and nonsignificantly, related to overall dreaming.⁶⁰ Notably, mostly due to RAGE, *hostile emotion* was also significantly positively related to DC and TRC. Question 4 of the MDQ (Appendix B) asked participants to rate how emotional their dream was overall, regardless of the specific emotions involved. When the raw scores of the basic emotions were added up they shared a highly significant positive relationship with this question, $r = .70, p < .0001$, indicating that the *more* basic emotions felt, and the more *intensely* these emotions were felt, the more emotional the dream was overall. Positive, aversive and hostile emotions were all equally related to overall emotionality, indicating that no one group of emotions were experienced more intensely.

⁶⁰ The emotions of RAGE and DOMINANCE have not been included as aversive emotions because, as shown in Table I1, hostility was unrelated to feelings of pleasantness or unpleasantness. It cannot be assumed, then, that these emotions are aversive.

Table I2.

The Basic Emotions and Overall Dream Recall: Intercorrelations

The Basic Emotions ^a	R	F	G	S	C	P	D	TRC	DC
RAGE	1	.11	-.07	-.10	.09	-.01	.45***	.34***	.26**
FEAR		1	.30**	-.15	.02	-.26**	0	.03	.17
GRIEF			1	-.03	.15	-.09	.06	-.02	.13
SEEKING				1	.07	.16	-.09	.29***	.22*
CARE					1	.12	.10	.20*	.25**
PLAY						1	-.03	.16	.34***
DOMINANCE							1	.11	.13
				OE	PE	AE	HE		
Overall Emotion				1	.40***	.44***	.40***	.33***	.53***
Positive Emotion					1	-.17	-.03	.33***	.42***
Aversive Emotion						1	.05	.01	.18
Hostile Emotion							1	.27**	.23*

Note. Bolded values indicate statistical significance. Overall emotion was rated on Question 4 of the MDQ (see Appendix B).

TRC = Total Recall Count; DC = Dream Composite (standardised average of self-rated amount of dreaming and PIRS).

^a Positive Recall only, $n = 168$.

$p < .01$, ** $p < .001$, *** $p < .0001$, 2-tailed.

APPENDIX J

The Sleep Cycle and Dreaming

Table J1 presents data for all 267 dream reports as a function of the sleep cycle (i.e., the descending slope, trough, ascending slope, and REM), in relation to dream quantity and quality. A series of one-way ANOVA's revealed that overall emotionality, $F(3, 164) = 5.10$, $p = .002$, visual perception, $F(3, 164) = 6.69$, $p < .0001$, dream intensity, $F(3, 164) = 7.56$, $p < .0001$, TRC, $F(3, 114.42) = 12.26$, $p < .0001$, and DC, $F(3, 130.05) = 27.60$, $p < .0001$, all differed significantly as a function of the portion of the sleep cycle in which they occurred. Overall, hostility, $F(3, 155) = 1.09$, $p = .36$, pleasantness, $F(3, 155) = 1.24$, $p = .30$, and bizarreness, $F(3, 167) = 1.70$, $p = .17$, did not differ according to the different portions of the sleep cycle. Post hoc testing for the significant ANOVAs revealed that, in most cases, REM sleep mentation was significantly different from mentation from other parts of the sleep cycle:

- i. *Overall emotionality.* REM was significantly different from the trough ($p = .012$) and the ascending slope ($p = .004$), but not the descending slope ($p = .07$), of the sleep cycle.
- ii. *Visual Perception.* REM sleep differed significantly from the ascending slope ($p = .001$) and trough ($p = .001$). There was a trend towards a significant difference on the descending slope ($p = .05$).
- iii. *Dream Intensity.* REM sleep differed significantly from the descending ($p = .008$), ascending ($p = .001$), and trough ($p < .0001$) portions of the sleep cycle.
- iv. *TRC.* REM sleep differed significantly from the descending ($p = .002$), ascending ($p = .003$), and trough ($p < .0001$) portions of the cycle. Additionally, the ascending portion of the cycle had significantly increased TRC compared with the trough ($p < .01$).
- v. *DC.* REM sleep was significantly more dreamlike, according to the DC measure, than the ascending, descending and trough portions of the sleep cycle ($p < .0001$). Additionally, the trough of the cycle had significantly lower DC scores than the ascending slope ($p = .005$), and there was a trend towards a significant difference between the descending slope and the trough ($p = .04$).

Accordingly, REM sleep mentation differed in both quality (i.e., visual perception, emotionality, dream intensity) and quantity (TRC and DC) compared with the rest of the sleep cycle. In certain cases, the content differences between REM and the descending slope

reports only bordered on significance. Congruent with the literature, REM sleep yielded the highest dreaming rates, with 87% of the mentation from this stage, on average, classified as dreaming (Nielsen, 2000). Moreover, REM mentation was more emotional, vivid and involved more active participation than NREM mentation. In most cases, the ascending, trough and descending portions of the sleep cycle did not differ with regards to the quality of mentation recalled; however, there were significant differences in dream quantity between the ascending and trough portions of the sleep cycle, while there were only trends towards significant differences in dream quantity between the descending and trough portions of the cycle. In no cases of dream quality or quantity were the ascending and descending portions of the sleep cycle different.

Furthermore, there was a significant association between the part of the sleep cycle that the awakening was made in and the type of dream recall, $\chi^2(9) = 50.47, p < .0001$. The type of recall percentages for each portion of the sleep cycle is given in Table J1. Specifically, the standardised residuals indicated that the trough of the cycle had significantly more *no recall* reports ($Z = 2.7, p < .01$), and *no content* reports ($Z = 2.5, p < .01$), and significantly less *dreaming* ($Z = -2.8, p < .01$). In contrast, REM had significantly less *no recall* ($Z = -2.4, p < .05$) and *no content* ($Z = -2.5, p < .01$) reports, and substantially more *dreaming* ($Z = 3.4, p < .001$). There were no significant differences in the amount of cognitive activity; however, there was a nonsignificant trend ($Z = 1.5, p > .05$) towards more cognitive activity on the ascending slope. The ascending and descending slopes did not differ significantly for any of the types of recall, either from each other, or from the trough of the cycle or REM sleep. Moreover, the descending slope was comprised of 90% Stage 2 sleep, and 10% Stage 3; the trough of the cycle was comprised of 20% Stage 2, 50% Stage 3, and 30% Stage 4; and the ascending slope was comprised of 100% Stage 2. Because the descending and ascending slopes were largely comprised by Stage 2 sleep (light NREM sleep) and the trough was largely comprised of slow wave sleep (deep NREM), the differences in the types of dream recall could just as easily be attributed to the stage of sleep, rather than the portion of the cycle.

Additionally, type of recall was not associated with the time of night (early vs. late) for light NREM sleep, $\chi^2(3) = 4.75, p = .20$, and REM sleep, $\chi^2(2) = .09, p = .96$. For deep NREM sleep (Stages 3 and 4), 77% of the awakenings fell within the early half of the night, and consequently a Chi square analysis could not be carried out for time of night; however, there were *no* reports of cognitive activity in deep NREM sleep in the late half of the night. Similarly, 83.3% of the cognitive activity in the ascending slope occurred in the early half

of the night. No differences were found for the descending slope. Alternatively, there was approximately 20% more dreaming in the late portion of the night for both the trough (25.8% vs. 44.1%) and the ascending slope (47.2% vs. 66.7%) of the cycle. In contrast, the descending slope showed the opposite trend, with more dreaming in the early portion of the night (63.6% vs. 48.3%). With regard to time of night effects, then, dreaming appeared to be enhanced in the late portion of the night, and cognitive activity drastically reduced; these findings are congruent with previous literature showing less thoughtlike activity in the later part of the night (Fosse et al., 2004).

Table J1.

Dream Quantity and Quality According to the Sleep Cycle

	Descending ^a (<i>n</i> = 51)	Trough ^b (<i>n</i> = 96)	Ascending ^c (<i>n</i> = 66)	REM (<i>n</i> = 54)
Global Measures ^d				
Dream Composite	-0.02	-0.45	0.03	0.79
TRC	25.76	14.00	27.82	65.85
Content Measures ^e				
Hostility	-0.01	-0.14	-0.06	0.15
Visual Perception	-0.07	-0.25	-0.22	0.46
Pleasantness	0.08	-0.21	0.05	0.06
Dream Intensity	-0.11	-0.21	-0.14	0.38
Bizarreness	2.59	2.67	2.52	2.96
Types of Recall (%) ^d				
No Recall	21.6	36.5††	18.2	7.4**
No Content	11.8	19.8††	7.6	0.0**
Cognitive Activity	11.8	11.5	18.2	5.6
Dreaming	54.9	32.3††	56.1	87.0***

Notes. Pooled averages (based on individual dream reports) were used for all calculations.

Excluding TRC, all Global and content measures are standardised. Types of recall are reported as percentages. TRC = total word count; Bizarreness ranged from 1 to 5 (least to most bizarre); pooled averages.

^a 90% S2; 10% S3.

^b 20% S2; 51% S3; 30% S4.

^c 100% S2

^d All dream reports, *n* = 267

^e Positive dream reports only, *n* = 168

* *p* < .05, ** *p* < .01, *** *p* < .001, significantly more

† *p* < .05, †† *p* < .01, ††† *p* < .001, significantly less

APPENDIX K

Cyclic Alternating Pattern Scoring: Reliability Analysis

When the arousal subtypes are calculated for the different slopes of the cycle, it is evident that there were many more A₁ arousals during the descending and trough portions of the cycle, compared with the ascending portion (Table G1). In particular, of the arousals in the descending and trough portions of the sleep cycle, around 54% and 95% were of the A₁ type, respectively. In contrast, only around 48% of the arousals in the ascending slope were A₁, with the majority being comprised of the A₂ and A₃ subtypes. This pattern of distribution is largely consistent with the normative literature on CAP (Terzano & Parrino, 2000); however, there are certain discrepancies. First, there appear to be less A₁ type arousals in the descending slopes of the cycle (53% in our study vs. 90% in the literature),⁶¹ and also less A₂ and A₃ arousals in the ascending slopes (51.5% in our study vs. 64% in the literature). However, because the protocol involved waking participants up throughout the night, it was expected that their sleep microstructure and the natural progression of their sleep would be disrupted and atypical. The overall reduction in SWS may have caused additional homeostatic sleep pressure that may have disrupted the normative distribution of the arousal subtypes. Nevertheless, the maintained overall consistency in distribution trends between this study and the literature indicates that the natural ultradian distribution of the subtypes remained grossly intact despite the disruptive nature of the protocol.

On the other hand, *sequence length* (SL) and *the number of CAP cycles per sequence* in this study were close to normative literature; there were 5.68 cycles per sequence on average (vs. 5.6 reported in the literature) and the average SL was 207 seconds (vs. 153 in the literature; Smerieri et al., 2007). Therefore, despite the changes in the distribution of the subtypes, the number of cycles per second and the average sequence length appeared to remain intact and consistent with previous studies. Lastly, the length of the A phases appeared to be shorter than reported in the literature (Terzano & Parrino, 2000), while the B phases were comparable.

⁶¹ It should be kept in mind, however, that many of the descending slopes included in the analysis here were for the late portion of the night where there was no longer a build-up to deep NREM sleep; the percentage of A₁ calculated for the early portion of the night is 75% which is closer to the amount reported in the literature.

Table K1.
Descriptives for CAP variables

CAP Parameters	N	M	SD	95% Confidence Intervals		Norms
				LB	UB	
Cycles per sequence	84	5.68	5.32	4.52	6.83	5.6
Phase A length (sec)	184	6.10	2.21	5.76	6.44	7.3 ± 1.0
Phase B length (sec)	184	23.70	7.87	22.51	24.89	20 – 40
Descending	50					
% A ₁		53.36	39.64	42.09	64.62	90%
% A ₂		25.53	32.47	16.31	34.76	10%
% A ₃		11.00	35.55	11.00	31.21	
Trough	93					
% A ₁		95.12	11.82	92.68	97.56	94%
% A ₂		4.86	11.82	2.42	7.29	6%
% A ₃		0.02	0.18	0	0.05	
Ascending	65					
% A ₁		48.57	31.71	40.71	56.42	34%
% A ₂		40.33	31.25	32.59	48.07	64%
% A ₃		11.19	22.96	5.50	16.88	
SL Descending (sec)	13	119	62	112	126	-
SL Trough (sec)	20	275	232	168	382	-
SL Ascending (sec)	23	196	142	135	257	-
SL Total (sec)	56	207	176	159	254	156 ± 36

Note. These figures are based on only a sample of CAP sequences from each sleep record (in particular, CAP sequence immediately preceding each awakening), rather than all CAP sequences from that record. For this reason, 95% confidence intervals have been calculated for the total percentage of each of the subtypes, for each portion of the sleep cycle.

M = Mean; SD = Standard Deviation; LB = Lower Bound; UB = Upper Bound; SL = Sequence Length; sec = seconds. Norms come from Smerieri et al. (2007) and Terzano and Parrino (2000).

APPENDIX L
Individual Dream Reports and Percentage Dreaming

Table L1.
Number of Reports and Percentage Dreaming per Participant for each Preawakening Condition

Participant	Light NREM Sleep				Deep NREM Sleep			
	CAP		NCAP		CAP		NCAP	
	No.	%	No.	%	No.	%	No.	%
P1	3/4	75	0/1	0	2/3	67	0/2	0
P2	2/4	50	0/2	0	1/2	50	0/1	0
P3	2/3	67	2/4	50	1/4	25	0/2	0
P4	3/3	100	4/5	80	1/1	100	1/1	100
P5	1/3	33	1/3	33	0/2	0	-	-
P6	2/4	50	2/4	50	0/2	0	-	-
P7	3/3	100	1/2	50	1/2	50	1/1	100
P8	1/4	25	2/4	50	0/4	0	0/3	0
P9	2/2	100	1/3	33	2/2	100	0/1	0
P11	0/1	0	2/5	40	2/4	50	0/1	0
P12	1/1	100	2/5	40	2/5	40	0/2	0
P13	1/2	50	2/3	67	3/5	60	0/1	0
P14	2/2	100	1/3	33	1/6	17	0/1	0
P15	4/4	100	3/3	100	1/3	33	0/1	0
P16	0/2	0	1/2	50	0/4	0	0/1	0
P17	3/4	75	2/3	67	0/3	0	-	-
P18	2/5	40	2/3	67	1/2	50	0/1	0
P19	2/3	67	1/5	20	-	-	-	-
P20	3/5	60	1/3	33	1/3	33	-	-
P22	4/5	80	0/2	0	1/3	33	-	-
Total	41/64	-	30/65	-	21/60	-	2/19	-
<i>M</i>	3.20	63.60	3.25	43.15	3.32	39.89	1.36	14.29
<i>SD</i>	1.24	32.56	1.16	26.24	1.57	29.93	0.63	36.31
	Correlations between no. of reports and % dreaming							
<i>r</i>	-.02		.34		-.32		-.24	
<i>p</i> <	.46		.08		.09		.21	

Note. Positive Recall was defined here as any PIRS rating greater than 4. Average percentage was calculated from participant percentages (unweighted means). When averages are calculated using the number of positive reports/ total reports (weighted means), different averages are obtained. There were no associations between the number of reports per participant and the percentage of dream recall (see Pearson correlations and significance values).

Missing Data

Table L1 shows that there were missing data for certain participants, especially for the Deep NREM sleep NCAP condition. In these cases, participants either failed to have adequate deep NREM sleep, or had continuously unstable deep NREM sleep. Independent samples *t* tests were run to determine whether there were any differences in dream recall, for the deep NREM sleep CAP awakenings, between participants with NCAP reports and participants without NCAP reports. All *t* tests were run as two tailed as there was no a priori expectation for an effect in either direction. Participants without NCAP awakenings had substantially lower DC scores ($M = -0.87$, $SD = 0.44$, $n = 5$) than those with NCAP awakenings ($M = -0.22$, $SD = 0.49$, $n = 14$), although this difference did not reach significance, $t(17) = 2.65$, $p < .02$. Differences in percentage dreaming for those participants without NCAP awakenings ($M = 13.20$, $SD = 18.08$, $SE = 8.08$) compared to those with NCAP awakenings ($M = 45.86$, $SD = 30.70$, $SE = 8.21$) trended towards significance as well, $t(17) = 2.22$, $p < .04$, $r = .34$. However, there was a highly significant difference in remembrance for those participants without NCAP awakenings ($M = 0.40$, $SD = 0.55$, $SE = 0.25$) compared to those with ($M = 1.61$, $SD = 0.50$, $SE = 0.13$), $t(17) = 4.50$, $p < .0001$. Consequently, these missing data appear to be nonrandom; participants who had continuously unstable deep NREM also appeared to recall less dreams, to have less dreamlike recall, as well as an inability to remember their dreams clearly (in deep NREM).

APPENDIX M

Arousal Subtypes Regression Model

Assumptions and Casewise Diagnostics: OLS Model

The standardised residuals were negatively skewed (Figure M1). Therefore, despite the significant results, the violation of this assumption might make generalizability outside of this sample difficult. It should be noted, though, that when the analysis was run with *amount of dreaming* (AD) as the outcome variable the standardised residuals were found to be less skewed (Figure M2), and the overall model was unchanged, $R = .44$, $R^2 = .19$, $\text{Adj. } R^2 = .18$, $F(1, 50) = 12.02$, $p < .001$. Therefore, despite the negative skew in the original model, the results were considered to be relatively reliable. Furthermore, cross validation of the model using Stein's formula⁶² indicated that if the model were generalised to the population upon which the sample was based, the A_1 - A_2 difference would still have a significant (albeit, reduced) effect on DC, $R = .37$, $R^2 = .14$, $t(50) = 2.85$, $p < .01$. The value for the Durbin-Watson test was 1.77, close to a value of 2, and showed that the residuals were generally uncorrelated, or independent (Field, 2009). The partial plot of the standardised residuals by the standardised predicted values (Figure M3) also showed that there was no heteroscedasticity in the sample. Therefore, there were no gross violations of the assumptions of the OLS regression model for this analysis, and although generalizability will ultimately have to be established via replication, the model (based only on the prediction of global dreaming as a function of the A_1 - A_2 difference) appears to be relatively stable.

It should be noted, however, that measurement error can drastically reduce the regression coefficients in any model; an examination of the measurement error in this model and the effects on the coefficients is beyond the scope of this text. However, this issue would need to be addressed in any study that intended to replicate the results of this study. Additionally, any study aiming to replicate these results should also aim to have more dream reports per participant, so as to increase statistical power to detect any clustering in the data and avoid alpha inflation.

Finally, for normally distributed data we would expect 5% of the standardised residuals to exceed $Z = \pm 1.96$, and 2% to exceed $Z = \pm 2.58$; in the present analysis, 3.8% of the standardised residuals exceeded $Z = \pm 1.96$, and none exceeded $Z = \pm 2.58$. These values were consistent with normal distribution of extreme cases. Casewise diagnostics were further undertaken to determine whether any cases were exerting any undue influence on the model.

⁶² $R^2 = 1 - \left[\left(\frac{n-1}{n-k-1} \right) \left(\frac{n-2}{n-k-2} \right) \left(\frac{n+1}{n} \right) \right] (1 - R^2)$; where $n = 52$, $k = 1$, $R^2 = .19$ (Field, 2009; Stevens, 2002).

An investigation of the centred leverage values, Cook's distances, and the Mahalanobis values showed that no cases were exerting any undue influence on the model; the highest values for each of these diagnostic variables was 0.11, 0.06, and 5.38, respectively—far below the limit for concern for a sample of this size (Field, 2009).

Figure M1. Standardised Residuals for DC Regressed on the A_1 - A_2 Difference

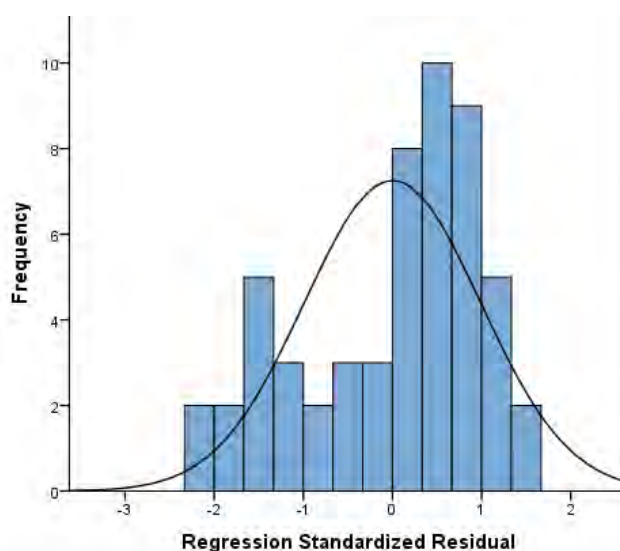


Figure M1. Histogram of the standardised residuals for global dreaming (DC) regressed on the A_1 - A_2 difference. The histogram shows a negative skew in the standardised residuals.

Figure M2. Standardised Residuals for Amount of Dreaming Regressed on A_1 - A_2 Difference

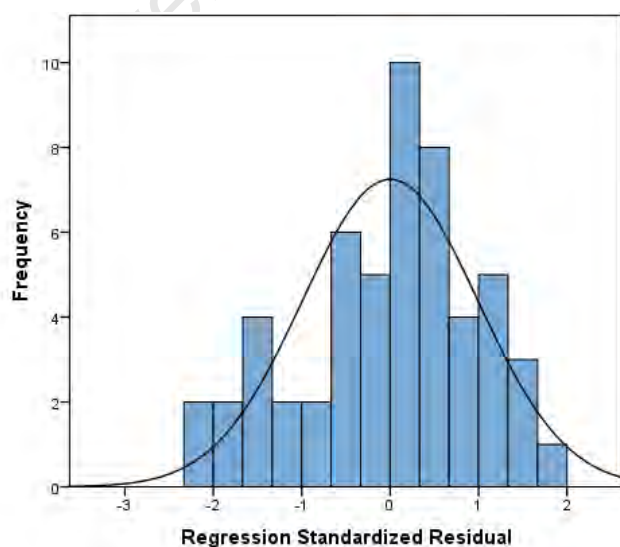


Figure M2. Histogram of the standardised residuals for amount of dreaming regressed on the A_1 - A_2 difference. The histogram shows a slightly negative skew for the standardised residuals.

Figure M3. Scatter Plot of the Standardised Residuals and Predicted Values

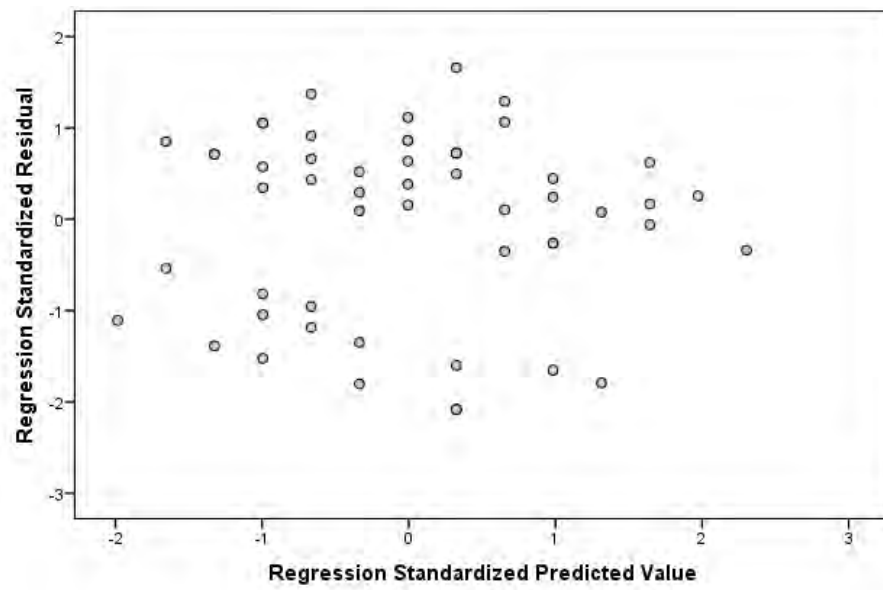


Figure M3. Scatter plot of the standardised residuals and predicted values for DC regressed on the A_1-A_2 difference. The even scatter indicates that there are no issues with heteroscedasticity.