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REVIEW ARTICLE

A Systematic Review of Treatments for Sleepwalking: 100 Years of Case Studies

Helen M Stallman[1*], Mark Kohler[1]

[1]Centre for Sleep Research, University of South Australia

ABSTRACT

While generally harmless, sleepwalking can result in injury to the sleepwalker and/or others, prompting help–seeking. This is the first study to systematically review the scientific evidence underpinning treatments for sleepwalking. A literature search of CINAHL, EMBASE, PsycINFO, PubMed, and ScienceDirect was conducted with the keywords 'sleepwalking' OR 'somnambulism', current to 29 February 2016. Studies were included if they reported on any intervention for sleepwalking. Of the original 53 sourced papers, 44 met the inclusion criteria and were subsequently included for review. None had a methodology that could demonstrate efficacy or effectiveness. Case and case series reports dominate the literature with patients treated with a range of psychological, pharmacological and other interventions. While the results of this review highlight potential treatments, well–designed randomized control trials of theoretically supported interventions that include assessment of adverse effects are urgently needed. Psychological interventions–scheduled waking and hypnosis–are potential first line interventions for evaluation, especially with children, as they are theoretically grounded, case studies suggest they may be effective, and they do not have the side–effects of pharmacological interventions.

Keywords: somnambulism, systematic review, intervention, parasomnia

INTRODUCTION

Sleepwalking, or somnambulism, is a parasomnia defined as ambulant behaviours occurring during sleep. It can involve simply walking around the bedroom or more dangerous behaviors such as leaving the house by opening doors and windows. Because these behaviours occur during sleep and within the context of impaired judgement, they can also be dangerous, such as falling from a high point (The Times, Nov 14, 2000), walking through glass doors or windows (e.g. New York Times, Jan 30, 1895), leaving the house (The Amidale Express, Jun 21, 1887), or attacking another individual within close proximity (de Bruxelles, Nov 18, 2009).

Sleepwalking is more frequently reported in children where it is generally considered benign (e.g. Bixler, Kales, Soldatos, Kales, & Healey, 1979; Klackenberg, 1982; Ohayon, Guilleminault, & Priest, 1999). The estimated prevalence of sleepwalking is 5.0% (95% CI 3.8– 6.5) in children and 1.5% (95% CI 1.0– 2.3) in adults (Stallman & Kohler, 2016). Conceptualizations of sleepwalking range from it being a single behavioural event to it being classified as a disorder which requires: a) recurrent episodes; b) contact with others while sleepwalking; and c) evidence of impaired cognitive processing by

***Correspondence:** helen.stallman@unisa.edu.au
Dr Helen Stallman, Senior Lecturer, Psychology, Centre for Sleep Research, University of South Australia, GPO Box 2471, Adelaide, South Australia 5001
Tel: +61 8 830 24360

demonstration of one or a combination of being difficult to arouse, mental confusion, at least partial amnesia of the event, inappropriate, nonsensical and/or dangerous behaviours (American Academy of Sleep Medicine, 2014). Sleepwalking is classified by the DSM–V as a mental illness if, in addition to these ICD–10–CM characteristics, the events cause clinically significant distress or impairment in social, occupational or other important areas of functioning (American Psychiatric Association, 2013). This increasing complexity to the definition necessarily results in decreasing prevalence rates, as single sleepwalking events are far more prevalent than the multiple criteria comprising the mental illness of sleepwalking (Stallman & Kohler, 2016).

While generally harmless, the perceptual and judgement deficits characteristic of sleepwalking mean there is the potential for injury with each episode. However, there is a paucity of research on when sleepwalking is considered a problem by the sleepwalker or parents of children who sleepwalk. One study found a significant relationship between frequency of sleepwalking in children and it being perceived as a problem by parents, but the effect size was very small (Stallman, Kohler, Biggs, et al., 2016).

Interventions for sleepwalking

Historically, a range of interventions have been used to prevent sleepwalkers from injuring themselves including being handcuffed or tied to the bed (e.g. Richardson, 1909) and a laser beam across the foot of the bed to trigger an alarm when it was broken by the sleepwalker (The Telegraph, Sep 11, 1935). Clinically, problem sleepwalking is treated with a range of psychological, pharmacological and other treatments. Consumer websites make treatment recommendations ranging from sleep hygiene and stress management to pharmacological treatments (e.g. National Sleep Foundation, 2015; NIH US National Library of Medicine, 2015). The National Health Service in the UK (NHS) for example (2015), recommends sleep hygiene, restricting fluid and caffeine intake, scheduled waking of children, cognitive behavioural therapy and hypnotherapy. The NHS recommends pharmacological interventions when there is a risk of injury to sleepwalker or to others. The Australian Medicines Handbook lists benzodiazepines as the only drug acceptable for use for sleepwalking (Rossi, 2015). Internationally, tranquilizers, some antidepressants and scheduled waking are also recommended (Drugs.com [Internet], 2015).

The theory of causal mechanisms differs between interventions. Behavioural therapies, such as classical conditioning (Edmonds, 1967; Meyer, 1975), scheduled waking (Frank, Spirito, Stark, & Owens–Stively, 1997; Tobin, 1993) and hypnosis to wake when one's feet touch the floor (Kennedy, 2002), aim to extinguish the behaviour by associating it with wakefulness or an unpleasant stimulus. Sleep hygiene, also a behavioural therapy, aims to prevent hypothesized triggers for sleepwalking, such as sleep deprivation or a full bladder. Hypnotherapy purports to improve control of behaviour (e.g. Kohler, 2011), release repressed emotions (Lindner, 1945; Richardson, 1909) or unconscious defences (Teplitz, 1958). Psychoanalysis works on ego development (Teplitz, 1958). Play therapy aims to help children manage emotions thought to trigger sleepwalking (Clement, 1970; Fujinawa, 1979).

Sedative pharmacological interventions are hypothesized to reduce slowwave sleep, when sleepwalking occurs. Benzodiazepines, for example, potentiate the inhibitory effects of GABA throughout the CNS, resulting in anxiolytic, sedative, hypnotic, muscle relaxant and antiepileptic effects (Rossi, 2015) and reduce slow wave sleep (Arbon, Knurowska, & Dijk, 2015; Plante et al., 2016; Shrivastava, Jung, Saadat, Sirohi, & Crewson, 2014; Stockmann et al., 2015). Antidepressants are also thought to reduce sleepwalking either through their anti–anxiolytic properties (Liliwhite, Wilson, & Nutt 1994) or by reducing partial arousal, characteristic of sleepwalking (Frölich, Wiater, & Lehmkuhl, 2001). Anticholinergic medication is used to address a proposed cholinergic mechanism in sleepwalking (e.g. Hodoba & Schmidt, 2012). Atypical antipsychotics are thought to decrease slow–wave sleep (Gill, Pillai, Koh, & Jambunathan, 2011). Melatonin therapy aims to address a faulty transition from slow wave to REM sleep states (Jan, Freeman, Wasdell, & Bomben, 2004).

Research Aim

The only systematic review of interventions for sleepwalking, conducted in 2008, found norandomized control trial evaluations (Harris & Grunstein, 2009), leaving an absence of evidence–base for treatments of this relatively common disorder. Clinical trials are essential to ensure consumers receive appropriate treatment, and also help protect them from harm or adverse consequences, including unnecessary costs when there is no evidence of benefit. In the absence of clinical trials, it is important to review other levels of evidence to inform research trials. The aim of this study is to extend previous research by conducting a systematic review of the science to date that informs sleepwalking interventions, including studies with all levels of evidence (Howick et al., 2011) in the absence of RCTs. Our aim is to evaluate the current level of evidence for the effectiveness of treatments to reduce the frequency of sleepwalking in children and adults. The review will include both psychological, pharmacological, and other interventions, in any setting.

METHOD

The following databases were included in the identification of relevant studies: CINAHL, EMBASE, PsycINFO, PubMed, and ScienceDirect. Search terms were 'sleepwalking' OR 'somnambulism'. The combined lists were screened for relevant titles; abstracts of all

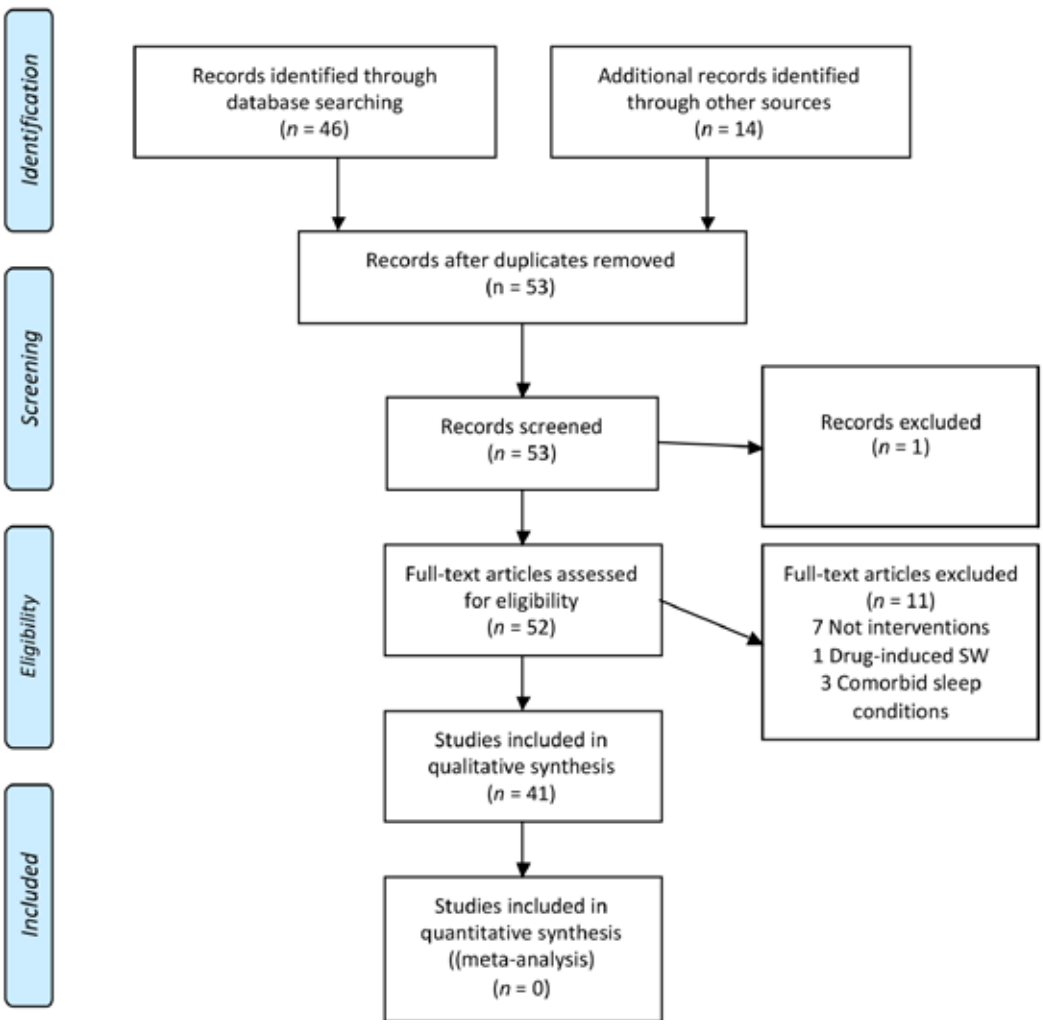

Figure 1: Flow of information through the different phases of the systematic review

marginally relevant titles were examined. Studies were included if: 1) they investigated an intervention for sleepwalking; and 2) they were published in a peer–reviewed publication. Studies were excluded if participants were forensic cases, sleepwalking was drug–induced or had comorbid sleep problems. We identified other pertinent studies through Google Scholar, citation tracking, review of reference lists of retrieved articles and our knowledge of the literature. The search dates were from the database start date through to 15 February 2016. Our findings were synthesized and described narratively.

RESULTS

Figure 1 displays the flow of information through the different phases of the systematic review. A total of 44 studies describing an intervention for sleepwalking were identified. There were no adequately powered randomized controlled efficacy or effectiveness trials, with case and case series reports dominating the literature–38 of the 41 studies identified. The only RCT conducted to date was significantly underpowered (*N* = 13) to be able to detect a significant treatment effect (Reid, Ahmed, & Levie, 1981). A multiple participants ABCBA case design

Table 1: Sleepwalking Case Reports and Case Series studies of treatment in children

Citation	Sex	Age	Intervention	Outcome measure	Outcome
Child					
Teplitz (1958)	M	10	psychoanalysis –psychic conflicts		
	M	14	including fear of death, sexual	ns	ns
	F	5	impulses, assertion, sphincter control,		
	M	10	and sibling rivalry		
	M	6			
	F	4			
Clement (1970)	M	7	play therapy, managing aggressive feelings,	Parent report	No change
			anticipatory awakening		Reduction in SW
Glick et al (1971)	M	8	diazepam	ns	No SW while taking medication
	M	9	imipramine/diazepam	ns	No change/No SW while taking diazepam
Pesikoff & Davis (1971)	na	na	imipramine	na	No SW
Fujinawa (1979)	M	8	play therapy	na	na
Gardner & Olness (1981)	M	11	hypnosis	na	na
Li (1989)	7 M 3 F	5–50	acupuncture and herbs	ns	8 cured; 1 improved; 1 no change
Tobin (1993)	M	8	scheduled awakening	Parent report	No SW after 5 days Maintained at 1 year
Schnenck & Mahowald (1995)	F	17	clonazepam	Self–report	Stopped because of side–effects
			progestins and estrogens		Stopped because of side–effects
			self–hypnosis		Unclear
Frank et al (1997)	M	6	scheduled awakening	Parent report	Reduction in SW
	M	12			No SW from intervention through 6 month f/u
	F	7			
Frölich (2001)	M	12	paroxetine	Parent report	Reduction in SW at 2 weeks No SW at 2 mths
Jan et al (2004)	M	12	melatonin	Parent report	No SW at 6 months
Gill et al (2011)	M	15	quetiapine	na	na
Hodoba & Schmidt (2012)	M	15	biperiden	ns	Stopped after 2 mths
	M	17	biperiden	self–report	Stopped after 6 weeks; returned periodically

Note. SW = sleepwalking; ns = not specified; na = paper not accessible;

study (B = no treatment; A & C = active treatment or placebo) of five adults found no significant difference between diazepam and placebo medication (Reid, Haffke, & Chu, 1984). An AB (A = placebo, B = active treatment) case design with 24 children showed a significant reduction in sleepwalking from flurazepam compared with placebo (Reimão & Lefévre, 1982).

Tables 1 and 2 provide a summary of case and case series reports of treatments with children (Table 1) and adults (Table 2). Four studies reported both child and adult cases and the cases are reported in the relevant table. Case and case series reports comprised more psychological (*n* = 22) than pharmacological (*n* = 10) interventions, with five studies including both and one study that reported a combination of acupuncture with herbs. Psychological treatments were predominant until 1980 and since then there have been relatively equal numbers of psychological and pharmacological

Table 2: Sleepwalking Case Reports and Case Series studies of Treatment in Adults

Citation	Sex (*N*)	Age	Intervention	Outcome measure	Outcome
Child					
Richardson (1909)	M	44	Hypnosis – suggestion that sleepwalking was unnecessary	ns	No SW for several weeks
Lindner (1945)	M	29	Hypnoanalysis	ns	Unclear
Pai (1946)	117 M	18–37	hypnosis, sedation, problem solving	ns	Success, not clarified
Teplitz (1958)	F	ns	psychoanalysis –ego development	ns	ns
Walton (1961)	M	35	assertion training, classical conditioning	Partner report Self–report	Reduction in SW after 2 weeks No SW after 6 weeks
Edmonds (1967)	M	21	aversion therapy – electric current, sedation	Unclear ns	No change No SW for 6 months
Dillahunt (1971)	M	ns	hypnosis – calm sleep	ns	success
Dillahunt (1973)	F	ns	hypnosis – self–regulation	ns	No SW 5 years
Eliseo (1975)	M	19	hypnosis – assertiveness, control	ns	No SW for 6 weeks Reoccurred several weeks later
Myer (1975)	M	24	classical conditioning	Partner report	Reduction in SW
Reid (1975)	6 M	ns	hypnosis – self–regulation	ns	4 had no SW after 3 weeks; 2 had no change in SW
Reid & Gutnik (1980)	M	26	hypnosis – calm, relaxed sleep Diazepam		No change Reduction in SW while taking diazepam
Nugent et al (1984)	M	20	hypnosis – calm sleep and arousal awareness	Self–report	No SW after 9 months
Cooper (1987)	M	60	imipramine	Partner report	Reduction in SW while taking imipramine
Berlin (1988)	F	33	birth control pill triazolam, Psychotherapy	Self–report	No change Reduction in SW No SW after 6 months
Li (1989)	7 M 3 F	May.50	acupuncture and herbs	ns	8 cured; 1 improved; 1 no change
Hurwitz et al (1991)	8 ns	ns	hypnosis – self–control	Self–report rating scale of level of improvement	74% reported improvement
Cuisinier & Hoogduin (1991)	M	na	hypnosis	na	na
Liliwhite et al (1994)	F	46	SSRI	EEG Partner report	Not reported
Schnenck & Mahowald (1995)	F	46	progestins and estrogens self–hypnosis	Self–report	Stopped because of side–effects Unclear

Atay & Karacan (2000)	16 M 6 F	*M* = 27.9 years	N = 11 treatedcarbamazepine, valproate, clonazepam	Self–report	mixed
Kennedy (2002)	F	30	hypnosis – calm sleep and arousal awareness	Self–report Partner– report	No SW at 1 week Reduced freq. over 5 months
Hauri et al (2007)	11 Adults*		hypnosis	self–report	50% no SW or much improved at18 mths 67% no SW or much improved at 5 yrs
Conway et al (2011)	M	33	clonazepam	Self–report	Decreased SW but unacceptable side–effects
			psychotherapy – emotional conflicts		Decreased SW
	F	39	clonazepam and psychotherapy – emotional conflicts	Self–report	Decreased SW
Kohler (2011)	5 M 3F	M = 44 SD = 12.5	hypnosis	PSG	Increase in delta, sigma and beta frequencies
Hirscher & Riemann (2011)	Adult	na	cognitive behavioral therapy	na	na
Hodoba & Schmidt (2012)	M	21	Diazepam, clonazepam, amitriptyline biperiden and diazepam	ns other report	No change Reduced frequency
	M	42	biperiden and diazepam	self–report	Stopped after two months
Attarian & Zhu (2013)	39 Adults*		clonzapam, antidepressants, other benzodiazepines, sleep hygiene, CBT, hypnosis, stress management, dental device, CPAP	ns	ns**

Note. M = male; F = female; ns = not specified; na = paper not accessible; CPAP = continuous positive airway pressure; *Only sleepwalking participants reported here; **outcomes reported were grouped for all disorders of arousal

treatments reported in the literature. The case studies highlight the changing theoretical paradigms driving treatments with psychoanalysis and early hypnosis interventions focusing underlying psychic conflicts, behavioural interventions focusing on sleepwalking as a behaviour that can be unlearned and pharmaceutical interventions aiming to change sleep architecture.

Psychological interventions used were diverse and comprised psychoanalysis, hypnosis, scheduled or anticipatory waking, assertion training, relaxation training, managing aggressive feelings, sleep hygiene, classical conditioning (including electric shock), and play therapy. The content and dose of many of these interventions was not specified. Pharmacological interventions were an anticholinergic (biperiden), antiepileptics (carbamazepine, valproate), an antipsychotic (quetiapine), benzodiazepines (clonazepam, diazepam, flurazepam, imipramine, and triazolam), melatonin, a selective serotonin reuptake inhibitor (paroxetine), a barbiturate (sodium amytal) and herbs. The only non–psychological or non–pharmacological intervention used to treat sleepwalking was acupuncture.

DISCUSSION

Sleepwalking can pose a significant risk of injury to the sleepwalker, to others and, in rare cases, it can result in death. This study is the first to systematically review all levels of evidence for the treatment of sleepwalking. Consistent with the systematic review in 2008 (Harris & Grunstein, 2009), there are still no adequately powered randomized control trials reported in the literature. Despite sleepwalking interventions being described in the literature for more than 100 years and a wide range of pharmacological and psychological interventions in use in the community and clinical practice during this time, there was no evidence for their efficacy or effectiveness. The literature is dominated by case reports and case series studies.

Single–case research designs can be useful for providing hypotheses about behaviour and opportunities for clinical innovation, permitting intensive study of rare phenomena, challenging theoretical assumptions, and providing tentative support for a psychological theory (Shaughnessy & Zechmeister,

1997). On these grounds, the repetitive use of similar interventions in case studies of sleepwalking no longer provides justification for their use in clinical research. The limitation of case studies–that is, the inability to draw cause and effect (Shaughnessy & Zechmeister, 1997)–is problematic in the treatment of sleepwalking because they are frequently accepted as valid by the general community. This is noted in both advice for professionals (e.g. Drugs.com [Internet], 2015; Rossi, 2015) and recommendations for the general community (e.g. National Sleep Foundation, 2015; 2015; NIH US National Library of Medicine, 2015). The therapy modalities identified in this study however, provide an overview of potential therapeutic approaches and pilot data to guide randomized control trials to evaluate their effectiveness alone and against other interventions.

All interventions have associated harm, which may be mild, such as the time and cost of psychological therapies or the inconvenience of parents implementing scheduled waking. The more intensive and invasive of the interventions reviewed in this study included 60 sessions of acupuncture (Li, 1989) and pain and burn marks from electric shock (Edmonds, 1967). Furthermore, there can be significant adverse effects from the medications prescribed off–label. Case studies, for example, suggest that quetiapine and paroxetine may actually trigger sleepwalking (Stallman, Kohler, & White, 2016). Both paroxetine and imipramine are associated with clinically significant increases in harm in adolescents, including suicidal ideation and behaviour, other serious adverse events for paroxetine, and cardiovascular problems for imipramine (Le Noury et al., 2015). Benzodiazepines have risks including cognitive and psychomotor effects, tolerance, dependence (Baldwin et al., 2013) and increased risk of dementia (Billioti de Gage et al., 2012). It is particularly concerning that pharmacological interventions are used, especially with children, when psychological interventions have the same level of evidence and without the same level of risk for adverse effects. Psychological therapies may also have significant side–effects. Scheduled waking, for example may disrupt NREM sleep and thereby contribute to daytime tiredness and behavioural and emotional problems. Psychological interventions that treat sleepwalking as a symptom of psychic conflict, for example psychoanalysis and play therapy, may erroneously convince sleepwalkers they are psychologically impaired.

Efficacy research trials of interventions for sleepwalking are urgently needed to protect consumers. Although case studies are used as evidence for treatment recommendations, they are unable to demonstrate efficacy as often reported (e.g. Giglio, Undevia, & Spire, 2005; Hughes, 2007). To date, research designs have been informed by logistics–that is the presence of an individual case rather than a logical problem, such as, is Intervention X effective in reducing sleepwalking? The research has not used methodologies that are able to demonstrate efficacy or effectiveness (Flay et al., 2005). Future research needs to include: a) research designs that allow for unambiguous causal statements; b) adequate sample sizes to test hypotheses; c) psychometrically sound measures; d) measures of treatment adherence; and e) measurement of adverse effects. Of particular importance in sleepwalking research, where the phenomenon is usually infrequent, irregular, and difficult to measure, measurement triangulation is essential to eliminate alternative plausible hypotheses including that the results are simply artefacts of the method of data collection or natural variation in the frequency of sleepwalking. Although problem sleepwalking is relatively uncommon, therefore limiting recruitment into randomized control trials, multi–site collaborations and research designs that account for small sample sizes (e.g. Hampson, Whitehead, Eleftheriou, & Brogan, 2014; Honkanen et al., 2001) can be used to maximize power while still applying a research methodology that can demonstrate efficacy and effectiveness. Future research also needs to include detailed descriptions of content and duration of psychological interventions and dose and duration of pharmacological interventions to enable replication and clinical application.

While the results of research to date are limited by methodological problems and mixed results, brief psychological interventions–scheduled waking or hypnosis–may be the most promising treatments for sleepwalking. Theoretically, the mechanisms of change

have the potential to continue after treatment finishes, whereas pharmaceutical interventions are more likely to require ongoing treatment to maintain treatment effects. Furthermore, the disruption to brain functioning caused by pharmaceutical interventions and potential adverse effects, particularly in the developing brains of children also make strong case for psychological interventions being the first line for empirical investigation.

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