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Quality of sleep and its daily relationship to pain intensity in hospitalized adult burn patients

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Abstract

Sleep disturbances are frequently reported in victims following burn injuries. This prospective study was designed to assess sleep quality and to examine its daily relationship to pain intensity within the first week of hospitalization. Twenty-eight non-ventilated patients were interviewed during 5 consecutive mornings (number of observations = 140) to collect information about perceived quality of sleep (visual analogue scale, number of hours, number of awakenings, presence of nightmares). Pain intensity was assessed at rest (nighttime, morning, during the day) and following therapeutic procedures using a 0–10 numeric scale. Seventy-five percent of patients reported sleep disturbances at some point during the study although, in most patients, sleep quality was not consistently poor. Pooled cross-section regression analyses showed significant temporal relationships between quality of sleep and pain intensity such that a night of poor sleep was followed by a significantly more painful day. Pain during the day was not found to be a significant predictor of poor sleep on the following night. These results support previous findings that perceived quality of sleep following burn injury is poor. Moreover, they show a daily relationship between quality of sleep and acute burn pain in which poor sleep is linked to higher pain intensity during the day.

Keywords: Sleep quality; Acute pain; Burns; Hospitalization

1. Introduction

Hospitalized patients often complain of sleep disturbances (Jones et al., 1979; Lauri et al., 1997). These may be caused by a variety of exogenous factors such as environmental noise, bright lighting and repetitive staff interventions, or endogenous factors such as delirium, post-traumatic stress symptoms, depression, general anxiety, stress, inability to lie comfortably and pain (Miller et al., 1976; Rosenberg et al., 1994; see Phillips and Cousins, 1986; Wooten, 1994 for reviews).

Although the functions of sleep still remain unknown, it is widely thought to be a necessary restorative and energy conservation process (Zepelin and Rechtschaffen, 1974; Adam and Oswald, 1983; Berger and Phillips, 1995). Therefore, poor sleep can have serious detrimental effects on health and recovery from illness. Sleep deprivation can lead to sleepiness, increased fatigue, negative mood and periods of misperception and disorientation (Johnson, 1969; Bonnet, 1994; Totterdell et al., 1994). It can also produce a decrease in the secretion of growth hormone (Takahashi et al., 1968; Sassin et al., 1969) which promotes anabolic activity, regulates body growth and stimulates tissue restoration (Adam and Oswald, 1977). Poor sleep may also be a contributing factor in lowering pain threshold, thereby altering pain perception (Cooperman et al., 1934; Ross, 1965; Johnson, 1969; Bonnet, 1994; Lentz et al., 1999).

Most research investigating relationships between sleep and pain has focused on either chronic pain patients (Pilowsky et al., 1985; Atkinson et al., 1988; Mahowald et al., 1989; Moldofsky 1993; Affleck et al., 1996; Drewes et al., 1998; Morin et al., 1998; Smith et al., 2000) or healthy volunteers exposed to experimental pain stimulation (Drewes et al., 1997; Lavigne et al., 2000). Whether pain and poor sleep are causally linked remains unknown (Lavigne et al., 2001). Nevertheless, taken together, such studies support the notion of an intimate relationship between chronic pain and poor sleep where pain may lead to sleep difficulties which, in turn, may exacerbate pain. To

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our knowledge, no study has investigated these relationships in acute pain situations in hospitalized patients.

Patients hospitalized for burn injuries experience severe pain on a daily basis. Pain is not only associated with the burn injury itself, but also with daily therapeutic procedures such as dressing changes, debridement and physiotherapy. Although sleep disturbances have often been observed in hospitalized burn patients (Miller et al., 1976; Blumenfield and Reddish, 1987; Courtemanche and Robinow, 1989; Silva et al., 1991; Helm, 1992; Ehde et al., 1999), very few studies have investigated the frequency and duration of these problems. There have been reports that burn patients are sleep deprived (Dotson et al., 1986) and have persistent sleep disturbances such as insomnia, frightening dreams and nightmares (Kravitz et al., 1993; Lawrence et al., 1998). Only one study (Gottschlich et al., 1994) measured sleep objectively with polysomnography in burn patients noting a decrease of rapid eye movement sleep and an almost total absence of slow-wave sleep. However, this study was limited to a small sample ($n = 11$) of children who were mechanically ventilated.

In light of the above findings, we suspect that a large proportion of patients who are hospitalized for burn injuries suffer from sleep difficulties, that these disturbances can lead to increased pain intensity, and that increased pain intensity, in turn, can affect sleep quality. The goal of the present study was thus to measure interrelationships between sleep quality and pain intensity among a sample of adult burn patients during the first week following admission to hospital.

2. Methods

2.1. Subjects

Adult patients were recruited among successive admissions to the burn centers of the Hotel-Dieu du Centre hospitalier de l'Université de Montréal (CHUM) and the Centre hospitalier affilié universitaire de Québec (CHA)- Pavillon St-Sacrement, between 1998 and 1999. Patients were selected for participation in this study if they were admitted within 72 h of their burn injuries, expected to be hospitalized for at least 7 days, conscious, alert, and capable of answering questionnaires in French. Because of age-related changes in sleep architecture, participants over 50 years old were excluded. Patients requiring assisted mechanical ventilation or suffering from a diagnosed active neurological, substance abuse and/or psychiatric disorders (i.e. depression, chronic insomnia) were excluded.

Forty patients fitting the selection criteria were approached. Of these, 33 consented to participate. Five patients were subsequently dropped because of intubation ($n = 1$), amputation ($n = 1$), delirium related to alcohol withdrawal ($n = 2$), and voluntary withdrawal from the study ($n = 1$), leaving a total sample size of 28 patients who completed the study.

2.2. Procedure

The protocol was approved by the Ethics Committee of both institutions. Patients were approached within 96 h of their injuries by the first investigator (I.R.) or one of the trained research assistants to explain the procedures of the study. Patients signed the consent form and were then administered the *Sleep Questionnaire for Adults* to identify pre-existing sleep disturbances.

During the 5 following days, structured interviews were conducted every morning between 05:00 and 07:30 just after the patient woke up. These were to collect information on sleep during the preceding night, dream content (data not reported here) and pain levels. Interviews lasted 10–15 min and were taped on microcassette. Whether the patient stayed in a private or semi-private room each night was also noted. During the day, the treating nurse assessed pain levels at rest every 4 h with a 0–10 intensity scale routinely employed on the burn unit. In addition, the first investigator or the research assistant met with the patient within 30 min following a dressing change or other painful therapeutic procedures to assess average pain level with the same numerical pain scale.

2.3. Measures

2.3.1. Pre-existing sleep problems

Pre-existing sleep disturbances were assessed with the Sleep Questionnaire for Adults (SQA; VitalAire Healthcare/Santé and Canadian Sleep Society, document FR BR 29, 1998). This 22-item self-report questionnaire, constructed by an expert committee of the Canadian Sleep Society, identifies symptoms of insomnia and several parasomnias.

2.3.2. Quality of sleep

Subjective ratings of sleep quality were obtained with four measures. (1) The patients were asked to rate how they had slept the previous night using a visual analogue scale (VAS) (Scott and Huskisson, 1976; McDowell and Newell, 1996) consisting of a 10-cm horizontal line where the leftmost extreme corresponded to 'slept very poorly' and the rightmost to 'slept very well'. Also, patients estimated (2) the total number of hours slept, (3) the number of awakenings during the night and (4) the presence or absence of nightmares. Although subjective ratings of sleep quality are not as reliable as objective polysomnographic (PSG) measures, it has been reported that patients' estimates of sleep parameters shift in the same direction as objective measures (Lewis, 1969; Baekeland and Hoy, 1971) and that sleep quality VAS scores parallel changes detected with PSG recordings (Terzano and Parrino, 1992; Terzano et al., 1997). Moreover, facial burns prevent the use of electrodes for PSG recordings in most burn patients.

2.3.3. Pain

Pain intensity was measured with a 0–10 numeric scale where 0 represented 'no pain at all' and 10 'unbearable pain'. Numerical rating scales are sensitive, reliable and simple to apply (Jensen et al., 1986; Guyatt et al., 1987; Jensen and Karoly 1992; Cleeland and Ryan 1994).

2.4. Medication and other medical information

Analgesic medication was administered to the patients according to a standardized protocol in both burn centers. Every patient was provided with two prescriptions, one for pain at rest (background pain) and one for pain during therapeutic procedures such as dressing changes (procedural pain). The first prescription consisted of a continuous intravenous (i.v.) infusion or oral sustained-release formulation of morphine along with rescue doses given as needed. Procedural pain was controlled with i.v. bolus of morphine or oral short-acting morphine. Pain intensity, measured every 4 h at rest and after each painful therapeutic procedure was charted in patients' medical files and used to adjust their analgesic medication. Anxiolytic medication consisted of lorazepam or oxazepam and was administered as needed during the day, at bedtime and/or during the night.

A research nurse reviewed all patients' medical charts to record information about analgesic and anxiolytic medications administered on data collection days (type, dose, route). The analgesic medication data were transformed into morphine equivalent doses using an oral/parenteral ratio of 2:1 (American Pain Society, 1999). Information about burn type and severity (expressed in percent of total body surface area) was also extracted from medical charts.

2.5. Data analysis

Patients' characteristics, medication data and sleep and pain measures were analyzed with descriptive statistics. Data obtained as continuous variables are presented as mean ± SD. Pooled cross-section regression analyses (Dielman, 1989) were conducted with the Econometrics computer program to examine interrelationships between sleep quality and pain intensity in a temporal sequence across the 5 days. This statistical method is based on ordinary least-squares regression while removing autocorrelations unique to each cross section (i.e. subjects). This adjustment allows each within-subjects observation to be treated as an independent assessment, thereby generating a much larger sample and increasing statistical power.

3. Results

3.1. Patient characteristics

Twenty-four men and four women aged between 17 and 50 years (34.8 ± 10.0) completed the study. Burn size varied from 3.5 to 64%, with an average TBSA of 15.5% ± 13.5 and average length of hospitalization of 16.6 ± 9.7 days (range 5–40). Burns were all due to thermal injuries except for three that were caused by electrical ($n = 2$) and chemical ($n = 1$) agents.

3.2. Sleep quality

On average, data collection began 39.9 h (±21.2) following the burn injury. Thirty-six percent of the patients reported pre-existing insomnia symptoms on the SQA. None reported any prior medical condition that was likely to influence sleep reports. Eight patients slept in a private room for the duration of the study, 11 in a semi-private room whereas the others slept in both types of room. Patients reported sleeping an average of 6 h per night (±2.1), with frequent awakenings (4.3 ± 4.7). Thirty-nine percent of patients reported at least one nightmare during the study period. Scores on the sleep quality VAS ranged from 0 to 10 (5.4 ± 2.6). For descriptive purposes, the VAS ratings were recorded in the following three categories: poor sleep (0–4), average sleep (4.1–6) and good sleep (6.1–10). Frequencies for these categories are shown in Fig. 1. It can be seen that most patients (93%) experienced at least one night of good sleep and 75% experienced at least one night of poor sleep during the 5 days of the study.

Sleep quality VAS scores, hours of sleep and number of awakenings were all significantly ($P < 0.01$), though moderately, correlated in the expected directions, that is, patients reporting better sleep on the VAS also tended to report more hours of sleep and fewer awakenings (see Table 1). The average number of nightmares was significantly correlated only with the number of awakenings.

3.3. Pain intensity

Fig. 2 shows the mean pain levels reported by patients at different times of the day during the 5 study days. When these scores were averaged across the study period and submitted to analysis of variance (one-way with repeated measures) and post hoc mean comparisons (Tukey HSD test), the pain levels during therapeutic procedures (4.2 ± 2.0) as well as during the night (3.9 ± 2.6) were found to be significantly higher than those reported at rest (2.5 ± 1.3; $P = 0.0002$ and $P = 0.0006$, respectively) and upon awakening (2.8 ± 1.7; $P = 0.0007$ and $P = 0.0091$, respectively); ($F = 12.077$; $P < 0.0001$).

3.4. Medication

Morphine was administered to all patients except one who was also given fentanyl due to uncontrollable side effects of morphine. The fentanyl doses were transformed into morphine equivalents (100 μg of fentanyl = 10 mg of morphine) (American Pain Society, 1999). In all cases, analgesic medication was administered by i.v. route. Seven patients were switched from i.v. to the oral route during the study period. Across the 5 days of the study,

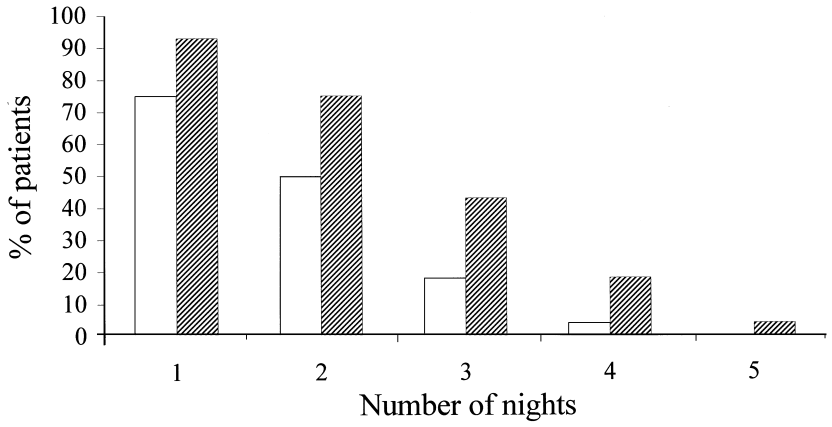

Fig. 1. Percentage of patients reporting good and poor sleep across the 5 days of the study. Open bar, poor sleep; hatched bar, good sleep.

patients received an average of 58.3 ± 36.4 mg of morphine per day for controlling pain at rest and during therapeutic procedures. When the amount of morphine given for procedural pain was excluded from the calculation, a slight but statistically significant difference was found between the quantities of morphine given at rest during the day and at night ($t = 3.4$; $P < 0.001$). The mean dose given per hour was slightly higher during the day (2.5 ± 1.8 mg/h) than at night (2.0 ± 1.5 mg/h). Approximately two-thirds of the patients were given anxiolytic medication (0.5–2.0 mg of lorazepam or 15–30 mg of oxazepam) at some point during the study period (5% only during the day, 32% only at bedtime or during the night, 63% at both times).

3.5. Interrelationships between pain and sleep

Two sets of pooled cross-section analysis were carried out to examine the interrelationships between sleep and pain. The first set was aimed at predicting sleep quality. Separate analyses were conducted for three sleep variables, i.e. sleep quality VAS, estimated hours of sleep and number of awakenings. Independent variables were: pain intensity during the night and during the previous day (at rest and during therapeutic procedures), morphine dosage (in mg) and benzodiazepine administration (yes/no) at night and during the previous day, and whether or not the patient slept in a private room. Age, burn severity (TBSA) and prior sleep disturbances were also included in the model. Results of the regression analysis and partial correlations are summarized in Table 2. Not all patients completed all rating scales at each time of observation therefore sample size varied between 100 and 140 observations depending on the variable. Bonferroni adjustments were applied to the primary outcome of the regression analyses to control alpha inflation. Throughout the first set of analyses, $P < 0.017$ was used as the threshold for statistical significance. High pain levels at night showed the strongest association with poor sleep quality. Furthermore, patients who received higher doses of morphine during the night tended to report better sleep. None of the variables assessing pain intensity earlier in the day predicted sleep quality.

Table 3 shows the results of the second set of analyses aimed at predicting pain intensity. Independent variables entered into the model were the sleep variables measured during the night: sleep quality VAS, hours of sleep, number of awakenings, and report of nightmares (yes/no), along with the other predictors included in the previous analysis. Separate regression analyses were conducted for pain during the night, and pain upon awakening, at rest and during therapeutic procedures measured on the following day. Again, Bonferroni adjustments were applied to the primary outcome and throughout this second set, $P < 0.0125$ was used as the threshold for statistical significance.

Reports of prior sleep disturbances showed the strongest association with higher levels of pain during the night, upon awakening, and during therapeutic procedures. High pain scores at night and during the day were predicted by either low scores on the sleep VAS, more awakenings or by the absence of nightmares. Patients who reported more pain during the night received more morphine and those who

Table 1
Pearson correlations between sleep variables[a]

	Sleep quality VAS	No. of hours	No. of awakenings	Nightmares
Sleep quality VAS	–	–	–	–
No. of hours	0.607*	–	–	–
No. of awakenings	−0.266*	−0.324*	–	–
Nightmares	−0.109	−0.058	0.301*	–

[a] VAS, visual analogue scale. *$P < 0.01$.

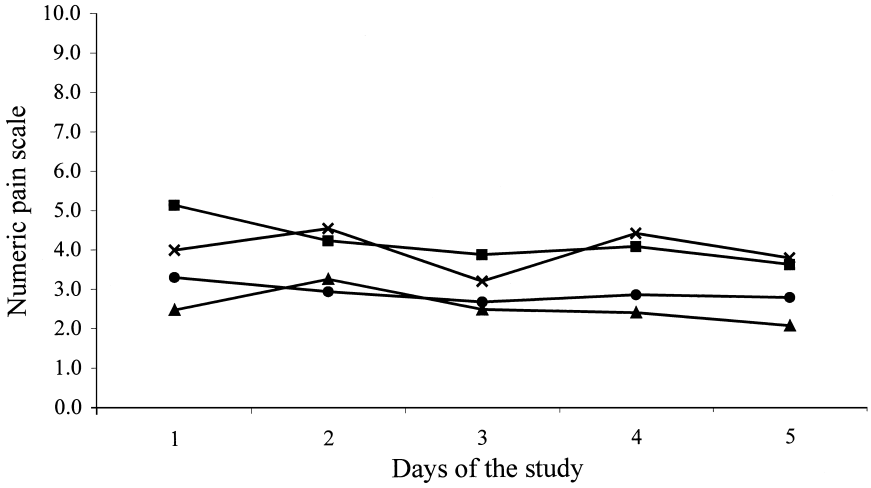

Fig. 2. Mean pain levels across the 5 days of the study. ▲, pain at rest; ●, pain upon awakening; ■, pain during procedures; ×, pain during the night.

reported more pain at rest and during therapeutic procedures received benzodiazepines more often during the day.

4. Discussion

This study is the first to demonstrate interrelationships between pain and sleep in patients hospitalized for burn injuries. Sleep disturbances were observed in about 75% of the patient sample, confirming earlier observations with hospitalized patients (Dlin et al., 1971; Johns et al., 1974; Orr and Stahl, 1977; Broughton and Baron, 1976; Jones et al., 1979; Kavey and Altshuler, 1979; Aurell and Elmqvist, 1985; Lauri et al., 1997). However, in most patients, sleep quality was not consistently poor. Almost all patients reported at least one good night of sleep and close to 50% reported as many as three good nights across the 5-day duration of the study. The diminished sleep quality that was observed did not appear to be related to pre-existing sleep disturbances (e.g. insomnia symptoms). All patients, with or without a history of sleep problems, reported poor sleep at one time or another during the first week of hospitalization.

Results from the regression analyses revealed that nighttime pain was a selective determinant of poor sleep. The intensity of pain reported during the night predicted poor sleep on all measures whereas daytime pain intensities at rest and during therapeutic procedures did not. Daytime pain in chronic pain patients (e.g. fibromyalgia) has been reported to affect sleep on the following night (Affleck et al., 1996). Therefore, it is possible that for hospitalized burn patients the effects of daytime pain on sleep may not become evident until several days or weeks have elapsed. Nevertheless, our finding of a selective effect of nighttime pain on sleep indicates that reduction of pain during the night may promote better sleep, as patients who received more analgesic medication did also report better sleep. Even if opioids have been shown to have a negative effect on sleep (Kay et al., 1969; Moote et al., 1989), this effect was a baseline condition for all patients as they all received

Table 2
Results of pooled cross-section regression analyses, with sleep quality measures as dependent variables[a]

	Sleep quality VAS ($r^2 = 0.34$)*		No. of hours ($r^2 = 0.24$)*		No. of awakenings ($r^2 = 0.27$)*	
	Partial r	$P\leq$	Partial r	$P\leq$	Partial r	$P\leq$
Pain during the night	−0.43	0.000001	−0.18	0.051	0.47	0.00000
Pain at rest[b]		NS		NS		NS
Pain during procedures[b]		NS		NS		NS
Morphine dosage: nighttime	0.24	0.009	0.36	0.00004		NS
Morphine dosage: daytime[b]		NS	−0.18	0.045		NS
Benzodiazepines: nighttime	0.19	0.034		NS		NS
Benzodiazepines: daytime[b]		NS		NS		NS
Type of room		NS		NS		NS
Age		NS		NS		NS
Burn severity		NS		NS		NS
Prior sleep disturbances		NS		NS		NS

[a] r^2 = percent of explained variance, *$P \leq 0.017$. NS, not significant.
[b] Variables measured on the preceding day.

Table 3
Results of pooled cross-section regression analyses, with pain intensity measures as dependent variables[a]

	Pain during the night ($r^2 = 0.48$)*		Pain upon awakening ($r^2 = 0.46$)*		Pain at rest ($r^2 = 0.38$)*		Pain during procedures ($r^2 = 0.58$)*	
	Partial r	$P\leq$	Partial r	$P\leq$	Partial r	$P\leq$	Partial r	$P\leq$
Sleep quality VAS[b]	−0.22	0.0176	−0.25	0.00569		NS	−0.26	0.011
No. of hours[b]		NS		NS		NS		NS
No. of awakenings[b]	0.31	0.000562	0.22	0.0159	0.28	0.003		NS
Nightmares[b]	−0.21	0.023		NS		NS	−0.33	0.001
Morphine dosage: nighttime[b]	0.30	0.00078	0.19	0.0349		NS		NS
Morphine dosage: daytime		NS		NS	0.21	0.032	0.22	0.035
Benzodiazepines: nighttime[b]		NS		NS		NS		NS
Benzodiazepines: daytime		NS		NS	0.24	0.012	0.34	0.0007
Type of room[b]		NS	−0.26	0.000034		NS		NS
Age		NS		NS	0.19	0.052		NS
Burn severity		NS		NS		NS		NS
Prior sleep disturbances	0.52	0.00000	0.37	0.0040		NS	0.56	0.00000

[a] r^2 = percent of explained variance, $*P \leq 0.0125$.
[b] Variables measured on the preceding night.

analgesics on a continuous basis, and so poor sleep was probably induced by factors other than analgesic medication.

As expected in burn patients (Perry et al., 1981; Choinière, 1994), pain intensity ratings were significantly higher for procedural pain than they were for background pain during the day. However, patients reported higher levels of background pain during the night than during the day. It is unlikely that this difference was due to the fact that patients received slightly less morphine during the night. Although significant, the difference was small (0.5 mg/h). Rather, the difference may be due to circadian variations in pain perception since pain intensity is known to fluctuate with circadian phase in other pain syndromes (see Labrecque et al., 1997 for review). Another possible explanation is that pain perception may be enhanced by certain exogenous factors only present during the night, e.g. being alone in the dark, less nursing activity in the room. Regardless of the explanation, the present results suggest that extra care be taken to reduce patients' pain during the night, if sleep quality is to be improved.

The present findings also reveal that poor sleep quality was a potent predictor of all aspects of pain in hospitalized burn patients. Poor sleep during the night (low score on the sleep quality VAS, frequent awakenings) was associated with higher pain intensities on the following day. This may indicate that treating sleep disturbances as an adjunct symptom may help to lessen patient suffering during hospitalization. Such treatment may involve simple strategies to improve sleep hygiene (e.g. less noise, fewer awakenings by staff) and better control of pain at night as described in the previous section.

Patients' self-reports of pre-existing sleep disturbances were found to be strong predictors of higher pain intensities on some measures (during the night and during therapeutic procedures). This relationship may reflect a chronic effect of non-restorative sleep on pain sensitivity and may indicate that non-restorative sleep has an immediate, as well as a chronic, influence on pain perception. It is also possible that the observed relationship between pre-existing sleep problems and pain may simply reflect a general tendency of some patients to complain more or to exaggerate symptoms, whether they be pain- or sleep-related in origin. Measures of hypochondriacal personality attributes may be needed to exclude this possibility in future studies.

An unexpected finding was that nightmare occurrence was predictive of lower pain intensities. There is no ready explanation for this finding, although some possibilities may be proposed. Since nightmares usually arise from long uninterrupted episodes of rapid eye movement (REM) sleep (Hartmann, 1984), it may be that their occurrence is an indirect result of REM sleep rebound reflecting better sleep, REM sleep consolidation, or perhaps sleep consolidation more generally. If subjects are sleeping well enough to experience lengthy nightmares, then perhaps they are drawing benefits from more restorative sleep. Another possibility is that nightmares serve some short-term adaptational function that affects pain perception. Dreaming in general has been proposed to serve a desensitization function (Perlis and Nielsen, 1993); nightmares may be viewed as an intensified form of such desensitization that may alter pain perception.

In summary, our results indicate that: (1) patients hospitalized for burn injuries suffer from sleep disturbances; (2) poorly controlled pain can worsen sleep quality; and (3) sleep disturbances can exacerbate pain during the first week of hospitalization. Although no causal relationships can be drawn from this study, the findings clearly indicate the need for judicious balance in the control of both pain and sleep to alleviate the pain-sleep disruption cycle. Certainly, all measures possible should be taken to improve sleep hygiene and promote sleep which, in turn, may reduce pain intensity the following day. In a complementary fash-

ion, greater attention to the alleviation of pain at night may directly improve sleep quality.

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References

Adam K, Oswald I. Sleep is for tissue restoration. J R Coll Phys Lond 1977;11:376–388.

Adam K, Oswald I. Protein synthesis, bodily renewal and the sleep-wake cycle. Clin Sci 1983;65:561–567.

Affleck G, Urrows S, Tennen H, Higgins P, Abeles M. Sequential daily relations of sleep, pain intensity, and attention to pain among women with fibromyalgia. Pain 1996;68:363–368.

American Pain Society. Principles of analgesic use in the treatment of acute and cancer pain. Glenview, IL: American Pain Society, 1999.

Atkinson JH, Ancoli-Israel S, Slater MA, Garfin SR, Gillin JC. Subjective sleep disturbance in chronic back pain. Clin J Pain 1988;4:225–232.

Aurell J, Elmqvist D. Sleep in the surgical intensive care unit: continuous polygraphic recording of sleep in nine patients receiving postoperative care. Br Med J 1985;290:1029–1032.

Baekeland F, Hoy P. Reported vs recorded sleep characteristics. Arch Gen Psychiatry 1971;24:548–551.

Berger RJ, Phillips NH. Energy conservation and sleep. Behav Brain Res 1995;69:65–73.

Blumenfield M, Reddish PM. Identification of psychologic impairment in patients with mild-moderate thermal injury: small burn, big problem. Gen Hosp Psychiatry 1987;9:142–146.

Bonnet MH. Sleep deprivation. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine, vol. 2. Philadelphia, PA: Saunders, 1994, pp. 50–67.

Broughton R, Baron R. Sleep patterns in the intensive care unit and on the ward after acute myocardial infarction. Electroencephalogr Clin Neurophysiol 1976;45:348–360.

Choinière M. Pain of burns. In: Wall PD, Melzack R, editors. Textbook of pain, vol. 3. London: Churchill Livingstone, 1994, pp. 523–537.

Cleeland CS, Ryan KM. Pain assessment: global use of the Brief Pain Inventory. Ann Acad Med 1994;23:129–137.

Cooperman NR, Mullin FG, Kleitman N. Studies on the physiology of sleep: XI. Further observations on the effects of prolonged sleeplessness. Am J Physiol 1934;107:589–593.

Courtemanche DJ, Robinow O. Recognition and treatment of the post-traumatic stress disorder in the burn victim. J Burn Care Rehabil 1989;10:247–250.

Dielman TE. Pooled cross-sectional and time series data analysis. New York: Marcel Dekker, 1989.

Dlin BM, Rosen H, Dickstein K, Lyons JW, Fischer HK. The problem of sleep and rest in the intensive care unit. Psychosomatics 1971;12:155–163.

Dotson CH, Kibbee E, Eland JM. Perception of sleep following burn injury. J Burn Care Rehabil 1986;7:105–108.

Drewes AM, Nielsen KD, Arendt-Nielsen L, Birket-Smith L, Hansen LM. Pain and sleep. The effects of cutaneous and deep pain on the electroencephalogram during sleep – an experimental study. Sleep 1997;20:632–640.

Drewes AM, Svendsen L, Taagholt SJ, Bjerregard K, Nielsen KD, Hansen B. Sleep in rheumatoid arthritis: a comparison with healthy subjects and studies of sleep/wake interactions. Br J Rheumatol 1998;37:71–81.

Ehde DM, Patterson DR, Wiechman SA, Wilson LG. Post-traumatic stress symptoms and distress following acute burn injury. Burns 1999;25:587–592.

Gottschlich MM, Jenkins ME, Mayes T, Khoury J, Kramer M, Warden GD, Kagan R. A prospective clinical study of the polysomnographic stages of sleep after burn injury. J Burn Care Rehabil 1994;15:486–492.

Guyatt GH, Townsend M, Berman LB, Keller JL. A comparison of Likert and visual analogue scales for measuring change in function. J Chronic Dis 1987;40:1129–1133.

Hartmann E. The nightmare: the psychology and the biology of terrifying dreams. New York: Basic Books, 1984.

Helm PA. Burn rehabilitation: dimensions of the problem. Clin Plast Surg 1992;19:551–559.

Jensen MP, Karoly P. Self-report scales and procedures for assessing pain in adults. In: Turk DC, Melzack R, editors. Handbook of pain assessment, New York: Guilford, 1992, pp. 135–151.

Jensen MP, Karoly P, Braver S. The measurement of clinical pain intensity: a comparison of six methods. Pain 1986;27:117–126.

Johns J, Larger AA, Masterton JP, Dudley HAF. Sleep and delirium after open heart surgery. Br J Surg 1974;61:377–381.

Johnson LC. Psychological and physiological changes following total sleep deprivation. In: Kales A, editor. Sleep physiology and pathology, Philadelphia, PA: Lippincott, 1969, pp. 206–220.

Jones J, Hoggart B, Withey J, Donaghue K, Ellis BW. What the patients say: a study of reactions to an intensive care unit. Intens Care Med 1979;5:89–92.

Kavey NB, Altshuler KZ. Sleep in herniorrhaphy patients. Am J Surg 1979;138:682–687.

Kay DC, Eisenstein RB, Jasinski DR. Morphine effects on human REM state, waking state and NREM sleep. Psychopharmacology (Berl) 1969;14:404–416.

Kravitz M, McCoy BJ, Tomkins DM, Daly W, Mulligan J, McCauley RL, Robson MC, Herndon DN. Sleep disorders in children after burn injury. J Burn Care Rehabil 1993;14:83–90.

Labrecque G, Karzazi M, Vanier MC. Biological rhythms in pain and analgesia. In: Redfern PH, Lemmer B, editors. Physiology and pharmacology of biological rhythms. New York: Springer-Verlag, 1997, pp. 619–649.

Lauri S, Lepisto M, Kappeli S. Patients' needs in hospital: nurses' and patients' views. J Adv Nurs 1997;25:339–346.

Lavigne GJ, Zucconi M, Castronovo C, Manzini C. Sleep arousal response to experimental thermal stimulation during sleep in human subjects free of pain and sleep problems. Pain 2000;84:283–290.

Lavigne GJ, Brousseau M, Montplaisir J, Mayer P. Pain and sleep disturbances. In: Lund JP, Lavigne GJ, Dubner R, Sessle B, editors. Orofacial pain: from basis science to clinical management, Chicago, IL: Quintessence, 2001, pp. 139–150.

Lawrence JW, Fauerbach J, Eudell E, Ware L, Munster AM. Sleep disturbances after burn injury: a frequent yet understudied complication. J Burn Care Rehabil 1998;19:480–486.

Lentz MJ, Landis CA, Rothermel J, Shaver JLF. Effect of selective slow wave sleep disruption on musculoskeletal pain and fatigue in middle aged women. J Rheumatol 1999;26:1586–1592.

Lewis SA. Subjective estimates of sleep: an EEG evaluation. Br J Psychol 1969;60:203–208.

Mahowald MW, Mahowald SR, Bundlie SR, Ytterberg SR. Sleep fragmentation in rheumatoid arthritis. Arthritis Rheum 1989;32:974–983.

McDowell I, Newell C. Pain measurements. In: McDowell I, Newell C,

editors. Measuring health. A guide to rating scales and questionnaires, vol. 2. New York: Oxford University Press, 1996, pp. 335–346.

Miller WC, Gardner N, Mlott SR. Psychological support in the treatment of severely burned patients. J Trauma 1976;16:722–725.

Moldofsky H. Sleep and musculoskeletal pain. In: Voeroy H, Merskey H, editors. Progress in fibromyalgia and myofascial pain, Amsterdam: Elsevier, 1993, pp. 137–148.

Moote CA, Knill RL, Skinner MI, Rose EA. Morphine disrupts nocturnal sleep in a does-dependent fashion. Anesth Anal 1989;68:S200.

Morin CM, Gibson D, Wade J. Self-reported sleep and mood disturbances in chronic pain patients. Clin J Pain 1998;14:311–314.

Orr WC, Stahl ML. Sleep disturbances after open heart surgery. Am J Cardiol 1977;39:196–201.

Perlis ML, Nielsen TA. Mood regulation, dreaming and nightmares: Evaluation of a desensitization function for REM sleep. Dreaming 1993;3:243–257.

Perry S, Heidrich G, Ramos E. Assessment of pain by burned patients. J Burn Care Rehabil 1981;2:322–326.

Phillips GD, Cousins MJ. Neurological mechanisms of pain and the relationship of pain, anxiety, and sleep. In: Cousins MJ, Phillips GD, editors. Acute pain management, New York: Churchill Livingstone, 1986, pp. 21–48.

Pilowsky I, Crettenden I, Townley M. Sleep disturbances in pain clinic patients. Pain 1985;23:27–33.

Rosenberg J, Wilschiodtz G, Pedersen MH, Von Jessen F, Kehlet H. Late postoperative nocturnal episodic hypoxaemia and associated sleep pattern. Br J Anaesth 1994;72:145–150.

Ross JJ. Neurological findings after prolonged sleep deprivation. Arch Neurol 1965;12:399–403.

Sassin JF, Parker DC, Mace JW, Gotlin RW, Johnson LC, Rossman LG. Human growth hormone release: relation to slow-wave sleep and sleep-waking cycles. Science 1969;165:513–515.

Scott J, Huskisson EC. Graphic representation of pain. Pain 1976;2:175–184.

Silva JA, Leong GB, Ferrari MM. Posttraumatic stress disorder in burn patients. South Med J 1991;84:530–531.

Smith MT, Perlis ML, Smith MS, Giles DE, Carmody TP. Sleep quality and presleep arousal in chronic pain. J Behav Med 2000;23:1–13.

Takahashi Y, Kipnis DM, Daughaday WH. Growth hormone secretion during sleep. J Clin Invest 1968;47:2079–2090.

Terzano MG, Parrino L. Evaluation of EEG cyclic alternating pattern during sleep in insomniacs and controls under placebo and acute treatment with zolpidem. Sleep 1992;15:64–70.

Terzano MG, Parrino L, Boselli M, Spaggiari MC, DiGiovanni G, Smerieri A. Sensitivity of cyclic alternating pattern to prolonged pharmacotherapy: a 5-week study evaluating zolpidem in insomniac patients. Clin Neuropharmacol 1997;20:447–454.

Totterdell P, Reynolds S, Parkinson B, Briner RB. Associations of sleep with everyday mood, minor symptoms, and social interaction experience. Sleep 1994;17:446–475.

Wooten V. Medical causes of insomnia. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine, vol. 2. Philadelphia, PA: Saunders, 1994, pp. 509–522.

Zepelin H, Rechtschaffen A. Mammalian sleep, longevity, and energy metabolism. Brain Behav Evol 1974;10:425–470.